A meta-analysis of the role of defeat and entrapment in depression, anxiety problems, post-traumatic stress disorder and suicidality

Andrew Philip Siddaway

Submitted to the University of Hertfordshire in partial fulfilment of the requirements of the degree of DClinPsy

June 2013

ACKNOWLEDGEMENTS

I am extremely grateful to my supervisors, Mr Jöerg Schulz (University of Hertfordshire) and Professor Alex M. Wood (University of Stirling), for the invaluable expertise and guidance they have provided. I would also like to thank Dr Peter J. Taylor (University of Manchester) for his help and guidance and Mr David Trickey and Dr John N. T. Martin for providing useful comments on earlier drafts.

ABSTRACT

Research investigating the role of two evolutionary constructs – perceptions of defeat and entrapment – in various psychological problems and processes has burgeoned over recent years. This meta-analysis quantitatively summarised the findings from 38 studies (11,343 participants) which examined relationships between perceptions of defeat and entrapment and four psychological problems commonly encountered in NHS clinical services: depression, suicidality, anxiety problems and Post-Traumatic Stress Disorder (PTSD). All correlations between defeat and entrapment and the four psychological problems were large by Cohen's (1988) criterion. Correlations between defeat and entrapment and depression were larger than those for the other psychological problem groups, and significantly larger than those for anxiety problems and PTSD. The magnitude of the observed correlations introduces the possibility that defeat and entrapment, and perhaps other evolutionary constructs, may be integral components or driving forces behind all psychological problems. A robust approach to sensitivity analysis provided confidence that the population effect size estimates are robust and were not severely inflated by unpublished studies not included in the meta-analysis. As there was no significant between-study heterogeneity, moderator analyses were undertaken on an exploratory basis. Findings are generally consistent with theoretical predictions from the Involuntary Defeat Strategy, the theoretical model underpinning the literature. Overall, perceptions of defeat and entrapment appear to be strong risk factors for the four psychological problems examined, perhaps representing transdiagnostic processes that are common across various psychological problems. The potential role of defeat and entrapment in mental health assessment, formulation, intervention and evaluation, is considered in detail and limitations of this meta-analysis and of the literature on which it is based are discussed, highlighting areas of research where future work is needed.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE: INTRODUCTION

1.1. Overview

This thesis presents a meta-analysis of studies describing relationships between two evolutionary mechanisms – perceptions of defeat and entrapment – and four psychological problems commonly encountered in NHS clinical services: depression, suicidality, anxiety problems and Post-Traumatic Stress Disorder (PTSD). To provide a context for the meta-analysis, this chapter begins with a brief overview of evolutionary approaches to psychological problems. Next, defeat and entrapment are defined and conceptualised and theoretical models linking defeat and entrapment to the four psychological problems are described. The conclusions of an existing narrative review of the literature are then summarised. Lastly, the advantages of the current meta-analysis over the previous review are described.

1.2. Evolutionary psychology

Over the past two decades, evolutionary psychology has emerged as a prominent new theoretical perspective within the field of psychology (see Buss, 1995, 2009; Confer, Easton, Fleischman, Goetz, et al., 2010) with direct relevance for understanding, treating and preventing psychological problems (e.g., Buss, 2009; Gilbert, 2009; Wakefield, 1992, 1999, 2007). Evolutionary psychology has also been used to recommend changes to the way in which psychological problems are conceptualised within psychiatric nomenclature. For instance, Wakefield provides a detailed critique of the concept of psychological "disorder" in which he suggests that disorder is best-understood as "harmful mental dysfunction". Wakefield suggests that conceptualising psychological problems in this way overcomes many of the criticisms raised against the DSM and ICD classification systems (see Wakefield, 1992, 1999, 2007). In this theory, Wakefield's "harmful" criterion describes the value judgements made by a particular society about psychological conditions, whereby particular psychological conditions are judged negatively by current sociocultural standards (i.e., a particular culture considers specific psychological conditions to be negative or harmful). Wakefield argues that a "mental dysfunction" exists when an evolved internal mechanism is unable to perform one of its naturally-selected functions

(Wakefield, 1992). For example, "very roughly … anxiety disorders involve failures of anxiety- and fear-generating mechanisms to work as designed; depressive disorders involve failures of sadness and loss-response regulating mechanisms" (Wakefield, 2007, p. 152). Wakefield suggests that his "dysfunction" criterion distinguishes psychological problems from a failure to function in a socially or personally preferred manner (e.g., "I'm in a dysfunctional relationship") and from various other negative mental conditions not considered disorders such as ignorance, lack of skill or lack of talent (Wakefield, 1992). It follows from this theory that understanding the evolved function of psychological mechanisms is a prerequisite to understanding when and how these mechanisms may fail to function as designed.

A very brief overview of evolutionary theory is now provided in order to contextualise later discussions of the relationships between perceptions of defeat and entrapment and psychological problems.

1.2.1. Core concepts from evolutionary theory

Evolution is a biological meta-theory which causally explains why life is able to survive and reproduce. Briefly, Darwin postulated that inherited biological traits which aid an organism's survival and reproduction would be transmitted to future generations at greater frequencies than alternative traits which do not serve these functions as well. Evolutionary psychology extends this focus to also examine psychological traits and how these may have helped humans survive and reproduce. Evolutionary psychology aims to "study human behaviour as the product of evolved psychological mechanisms that depend on internal and environmental input for their development, activation, and expression in manifest behaviour" (Confer et al., 2010, p. 110).

Central to evolutionary theory is the concept of adaptation, which is defined as "an inherited and reliably developing characteristic that came into existence as a feature of a species through natural selection because it helped to directly or indirectly facilitate reproduction during the period of its evolution" (Buss, Haselton, Shackleford, Bleske, et al., 1998, p. 535). The key features are that adaptations (1) arose in an ancestral population; (2) interact with the physical, social, or internal environment in ways that reliably solved adaptive problems (survival and reproduction) better than competing alternatives during the time period in which they

evolved (e.g., fear as a protective strategy against dangerous snakes); (3) promote the reproduction of individuals who possess the characteristics, or their genetic relatives; and (4) thus tend to become typical of most or all members of a species (Buss, 1995; Confer et al., 2010; Tooby & Cosmides, 1990). Adaptive designs therefore provide reproductive benefits *on average*, relative to their costs, and relative to alternative designs available to selection during the period of their evolution (Buss et al., 1998). Evolution is usually an incremental process, so only small changes that 'tinker' with the current design are normally possible. This means that once biological designs are established a particular way, even considerable design flaws can sometimes not be overcome by natural selection (e.g., fear of harmless snakes) (Gilbert, 1998). Adaptations are therefore not necessarily derived from good designs, but from compromises (Gilbert, 1998).

1.2.2. Social hierarchical behaviour (social rank)

The social group is thought to constitute one of the principal "selection environments" for the survival and reproduction of the human species (Baumeister & Leary, 1995; Brewer & Caporael, 1991; Buss, 1995). Brewer and Caporael (1991), for example, argue that the cooperative group may have been the primary survival strategy of humans in ancestral times, and this would have selected for adaptations suited for group-living such as cooperativeness, loyalty and fear of social exclusion. Group living fosters many advantages to humans, but it also involves competition and conflict for evolutionarily meaningful resources (e.g., social status, food, attachments, mates) (Gilbert & Allen 1998). Since continuous competition and conflict between group members would hinder survival and reproduction, a social hierarchy tends to form via some group members adopting a primitive de-escalation/submission strategy when there is competition (Rohde, 2001). Thus, competition for evolutionarily meaningful resources results in escalation/dominance for some individuals in a group (try harder, threaten, overpower) and de-escalation/submission for others (back down, submit, give up, down-grade aspirations) (Gilbert & Allen 1998; Nesse, 1998; Sloman, Gilbert & Hasey, 2003). It is argued that the de-escalation/submission strategy is inherited by all humans, and is only activated whilst particular group members compete. This strategy is thought to facilitate an adaptive social hierarchy by ensuring that individuals do not engage in conflicts or struggles they cannot win (and

be harmed in the process), or be excluded from the social network (Gilbert & Allen 1998; Nesse, 1998). It also means that the 'stronger' party will generally settle for only part of a resource if that will avoid the effort and risk of a fight (Nesse, 1998). The de-escalation/submission strategy therefore improves the ability of *all* group members to survive and reproduce: some individuals briefly taking a submissive position may not be optimal for those individuals, but it maintains the cooperative group, which is thought to be most important from an evolutionary (life and death) perspective (Gilbert & Allen 1998; Nesse, 1998).

With the context of evolutionary adaptations and human social rank in mind, two psychological mechanisms that seem central to understanding some psychological problems from an evolutionary perspective – perceptions of defeat and entrapment – will now be described.

1.3. Defeat and entrapment

Research investigating the role of perceptions of defeat and entrapment in various psychological problems and processes has burgeoned over recent years. So far, empirical evidence has implicated defeat and/or entrapment in the onset and exacerbation of various anxiety disorders (e.g., Birchwood, Trower, Brunet, Gilbert, et al., 2007; Gumley, O'Grady, Power & Schwannauer, 2004; Kendler, Hettema, Butera, Gardner, et al., 2003), Post-Traumatic Stress Disorder (PTSD) (e.g., Dunmore, Clark, & Ehlers, 1997, 1999, 2001; Ehlers Clark, Dunmore, Jaycox, et al., 1998), depression (e.g., Brown, Harris & Hepworth, 1995; Gilbert & Allan, 1998; Kendler, Hettema, Butera, Gardner, et al., 2003; Sloman et al., 2003), suicidality (e.g., Williams, 1997; Williams, Crane, Barnhofer & Duggan, 2005; Taylor, Wood, Gooding & Tarrier, 2010b), chronic pain (e.g., Tang, Goodchild, Hester & Salkovskis, 2010; Tang, Salkovskis & Hanna, 2007) and psychosis (e.g., Selten & Cantor-Graae, 2005, 2007; Taylor, Gooding, Wood, Johnson, et al., 2010a). Overall, these findings have been apparent across cross-sectional, retrospective and longitudinal designs, suggesting that perceptions of defeat and entrapment may be important transdiagnostic psychological processes which require greater clinical and research attention (Harvey, Watkins, Mansell & Shafran, 2004).

1.3.1. Conceptual clarification

It is important to clarify at this point that this meta-analysis is concerned with subjective or symbolic perceptions of defeat and entrapment (irrespective of whether the trigger is internal or external), which may of course differ from some objectively-defined marker of the constructs (Gilbert, 2000) (e.g., defeat in battle or an athletic competition, or entrapment via imprisonment) or the physical experience of these constructs, although there will of course be some phenomenological overlap between mentally and physically defeating and/or entrapping experiences.

1.3.2. Defeat

The concept of mental defeat has been developed within social rank theories of depression (e.g., Gilbert & Allen, 1998; Sloman et al., 2003). Mental defeat involves a perception of failed struggle and powerlessness resulting from the loss or significant disruption of social status, identity or a hierarchical goal (Gilbert, 2000; Gilbert & Allan, 1998; Rohde, 2001; Sloman et al., 2003; Taylor, Gooding, Wood & Tarrier, 2011a). Gilbert (2000) describes three main classes of events with the potential to induce perceptions of defeat in humans: (1) a failure to attain, or loss of, valued resources, including social and material (e.g., financial) resources; (2) social put-downs or attacks from others; and (3) internal sources of attack, such as self-criticism, unfavourable social comparisons or unachievable ambitions. Sample items from the Defeat subscale of the Defeat and Entrapment Scales (Gilbert & Allan, 1998) – the most widely used assessment of defeat and entrapment in the literature – include "I feel I have lost my standing in the world" and "I feel defeated by life". The idea that the individual feels that they have metaphorically struggled against, or been beaten back by, one or more triggering experiences, is conceptually important. Defeat is differentiated from loss or failure, as the latter do not necessarily entail this sense of failed struggle. For example, an individual's marriage may fail, with an ensuing divorce. However, if the individual was dubious about the marriage in the first place, and resigned to the failure, then the experience of mental defeat in relation to the marriage would be unlikely (Taylor et al., 2011a).

1.3.3. Entrapment

Psychological entrapment is derived from the concept of 'arrested flight' (Dixon, Fisch, Huber & Walser, 1989), whereby a powerful psychobiological

motivation to escape threat or stress is blocked (Dixon, 1998; Gilbert, 2001; Gilbert & Allan, 1998; Sloman et al., 2003; Williams, 1997). The powerful desire to escape is coupled with no or low likelihood of individual coping or agency, or rescue by others. External entrapment relates to entrapment by external events or circumstances (e.g., difficult job or relationship, unwanted role as a caregiver), whereas internal entrapment relates to entrapment by internal experiences (e.g., health problems, unwanted negative emotions or thoughts) (Gilbert & Gilbert, 2003; Gilbert, Gilbert, & Irons, 2004; Williams, 1997). Sample items from the Entrapment subscale of the Defeat and Entrapment Scales (Gilbert & Allan, 1998) include: "I am in a situation I feel trapped in" (external entrapment) and "I feel trapped inside myself" (internal entrapment). Entrapment is differentiated from hopelessness, which is thought to be a purely cognitive construct that focuses on the likelihood of future events and does not capture the motivation to escape or sense of diminished status that is important to defeat and entrapment (Gilbert & Allan, 1998).

1.3.4. Conceptualising the relationship between defeat and entrapment

The conceptual relationship between defeat and entrapment has been a matter of much debate. Historically, both the animal and human literatures on defeat and entrapment have conceptualised the two constructs as distinct (Gilbert & Allan, 1998). This view holds that defeat or entrapment are differentially activated depending on the escape potential of a particular experience: if a stressor can be escaped or resolved, an individual experiences defeat; if it cannot be escaped or resolved, the individual experiences entrapment (O'Connor, 2003; Rasmussen et al., 2010; Sloman et al., 2003; Williams, 1997). Some authors have also suggested an interaction between the two constructs. For instance, Gilbert and Allan (1998) proposed that feelings of defeat will increase if an individual focuses on their sense of entrapment.

However, the view that defeat and entrapment are separate constructs has recently been convincingly challenged by several exploratory factor analyses (Griffiths, Wood, Maltby & Taylor, 2013; Sturman, 2011 studies 1-3; Taylor, Wood, Gooding, Johnson & Tarrier, 2009) and a confirmatory factor analysis (Sturman, 2011), in independent samples, as well as evidence that defeat and entrapment are strongly inter-correlated (r = .81 - .85) (Rasmussen et al., 2010; Stowkowy & Addington, 2012; Taylor et al., 2010a), and demonstrate similar patterns of correlation

with other variables (Sturman, 2011; Taylor et al., 2011a). Evidence of this nature suggests that rather than being distinct, defeat and entrapment may in fact be different facets of a single underlying latent construct reflecting perceptions of being powerless or lacking the capacity to effect change in order to move on from an uncontrollable, unremitting, and inescapable status or role (Gilbert & Gilbert, 2003; Johnson, Gooding & Tarrier, 2008; Taylor et al., 2009; Williams, 1997). Indirect evidence in support of this position is also provided via theoretical suggestions that defeat and entrapment share a number of overlapping features (Johnson et al., 2008). For example, definitions of defeat suggest that it encompasses a lack of possible solutions or ways forward; elements also strongly associated with the concept of entrapment (Rooke & Birchwood, 1998). Similarly, both concepts share strong associations with other evolutionary constructs tapping "involuntary subordination" (e.g., measures of low social rank, loss of aspirations, submissiveness and low perceived status – explored further below) (Gilbert et al., 2002; Rooke & Birchwood, 1998; Sturman, 2011). Furthermore, qualitative investigations of entrapment have shown that depressed individuals may perceive themselves as trapped in a subordinate role (Gilbert & Gilbert, 2003), which is conceptualised as an aspect of defeat (Gilbert & Allan, 1998). Recently, two theories have been put forward which are able to synthesise the range of theories and evidence just discussed.

1.3.5. Sturman (2011) model

Sturman (2011) recently presented three exploratory factor analyses and a confirmatory factor analysis demonstrating that defeat and entrapment, along with various other evolutionary constructs, load onto a single latent involuntary subordination variable. This perspective suggests that self-report measures of defeat, entrapment, negative social comparison and submissive behaviour are all lower-order characteristics of a single higher-order involuntary subordination construct (which accounts for their shared variance). Social comparison was measured using the Social Comparison Rating Scale (Allan & Gilbert, 1995). Participants rate the degree to which they feel inferior or superior, incompetent or more competent, or unlikeable or more likable in relation to others. Submissive behaviour was measured using the Submissive Behavior Scale (Gilbert & Allan, 1994). On this scale, participants rate

the frequency of their submissive behaviours (e.g., "I agree that I am wrong, even though I know I'm not").

The fact that the different evolutionary social rank constructs loaded onto a single factor was used by Sturman (2011) as the theoretical basis for combining self-report measures of defeat, entrapment, negative social comparison and submissive behaviour in order to develop a new measure of involuntary subordination: the Involuntary Subordination Questionnaire (ISQ). Sturman (2011) presents evidence that the ISQ has strong psychometric properties, positively correlates with self-criticism, neuroticism and perfectionism, and negatively correlates with self-efficacy, high self-esteem and extroversion. Interestingly, the ISQ also showed moderate correlations with nonverbal behaviours thought to be indicative of subordination during interviews with undergraduates about their occupational experience (e.g., duration of time looking at the interviewer versus looking down when the interviewer leaned in towards the participants versus when he did not). This movement away from purely self-report data was a useful contribution to the literature. However, it must be noted that when statistical analyses examined differences between men and women, the correlations between the ISQ and nonverbal behaviours were moderate to strong in the men, and small to moderate and non-significant for women. Since the interviewer (Sturman) was male, these findings raise questions for future research about whether gender roles may affect the relationship between self-reported involuntary subordination and observable subordinate behaviours, or how involuntary subordination manifests behaviourally in each gender. Questions also remain around the full range of subordinate nonverbal behaviours and the potential for differences between undergraduates and individuals experiencing clinical-level symptoms.

1.3.6. Taylor et al (2011a) model

In probably the most comprehensive theoretical model to date, Taylor et al. (2011a) integrated the various theoretical accounts of how defeat and entrapment are thought to underlie different psychological problems into a single model (see Figure 1). At the centre of the model is the psychobiological Involuntary Defeat Strategy (IDS) response, which is seen as a direct consequence of perceived defeat. The IDS may then contribute to perceptions of entrapment, contingent on an individual's judgment about their ability to escape the initial defeating experience. The two

perceptions may be initially distinct during an aversive experience, but go on to form a self-reinforcing "depressogenic feedback loop" that is characterized by a chronically overactive IDS response. This model suggests that once the "depressogenic feedback loop" is operational, defeat and entrapment equally co-occur, as both perceptions emerge from the same cause and reinforce each other continuously in a vicious circle. The elements of the Taylor et al. (2011a) model will now be described in more detail, since this theory is the first to attempt to integrate the literature into one model and this model conceptually underpins the present meta-analysis.

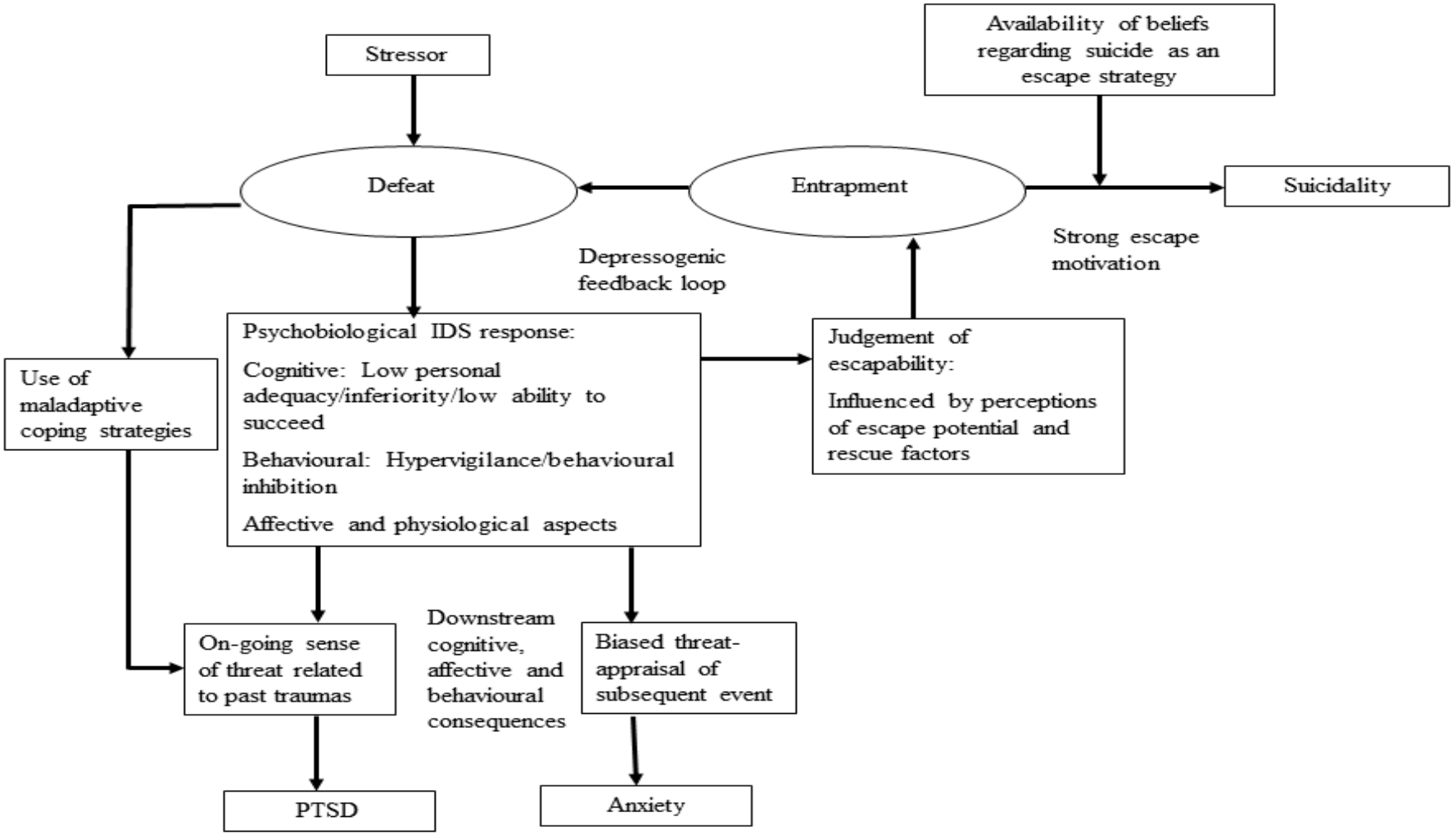
Stressor
Availability of beliefs regarding suicide as an escape strategy
Defeat
Entrapment
Suicidality
Strong escape motivation
Depressogenic feedback loop
Use of maladaptive coping strategies
Psychobiological IDS response:
Cognitive: Low personal adequacy/inferiority/low ability to succeed
Behavioural: Hypervigilance/behavioural inhibition
Affective and physiological aspects
Judgement of escapability:
Influenced by perceptions of escape potential and rescue factors
On-going sense of threat related to past traumas
Downstream cognitive, affective and behavioural consequences
Biased threat-appraisal of subsequent event
PTSD
Anxiety

Figure 1. Diagrammatic overview of the putative relationships between defeat and entrapment and the effects they exert upon depression, suicidality, anxiety, and posttraumatic stress disorder (PTSD). In this model, initial stressors trigger perceptions of defeat and the concomitant activation of the involuntary defeat strategy (IDS), which has cognitive, behavioural, and affective components. The IDS may lead to perceptions of entrapment, contingent on an individual's judgment of their ability to escape or resolve the situation. Perceived entrapment can then produce depressive symptoms. Entrapment may also further maintain initial perceptions of defeat, forming a depressogenic feedback loop. Lastly, perceived entrapment may result in suicidality, dependent on the availability of beliefs about the use of suicide as an escape strategy. IDS activation can also have downstream consequences, (a) biasing threat appraisals of future events to produce anxiety and (b) maintaining the sense of threat associated with past events to produce PTSD. Defeat may also lead to the use of maladaptive coping strategies that may further contribute to PTSD symptoms. From "The role of defeat and entrapment in depression, anxiety, and suicide," by P. J. Taylor, P. Gooding, A. M. Wood., and N. Tarrier, 2011, *Psychological Bulletin, 137*, p. 395. Copyright 2011 by the American Psychological Association. Reprinted with permission from P. J. Taylor.

1.4. The Involuntary Defeat Strategy (IDS)

Social rank theories suggest that the psychobiological underpinnings of relationships between perceptions of defeat and entrapment and psychological problems involve activation of the IDS (Sloman, 2000; Taylor et al., 2011a) – also called the "involuntary subordinate strategy" (e.g., Price et al., 1994) and "involuntary subordination" (e.g., Sturman, 2011). The human IDS is thought to be a primitive, evolutionarily adaptive, short-term stress and threat-defence response to perceptions of defeat (Sloman, 2000; Sloman et al., 2003), inherited from animals via a common evolutionary ancestry (Price et al., 1994; Sloman, 2000). It is believed to be an evolutionary mechanism or state that is common to all humans (similar to the "fight or flight" response), but which is only activated (as a damage limitation strategy) in the context of social competition or conflict for evolutionarily meaningful resources (Sloman, 2000; Sloman et al., 2003). The term 'involuntary' refers to the fact that a

primitive psychobiological de-escalation/submission response to defeat can be automatically triggered (Gilbert, 1992). The plausibility of the IDS as an evolutionary adaptation is supported by examples of other unpleasant yet functional adaptations in humans such as physical pain, vomiting and fever (Nesse, 1998). For example, humans experience pain during tissue damage; the pain deters the individual from continuing the behaviour that triggered the pain, and from repeating similar behaviours in the future (Nesse, 1998). Although pain is an aversive short-term experience, it is evolutionarily adaptive in helping humans avoid incurring injury and worsening existing injury.

Social rank theories suggest that unsuccessful social competition (defeat) activates the IDS. The function of the IDS is to trigger a defensive state characterised by a submissive no-threat status to others. Activation of the IDS involves behaviours to terminate or disengage from a struggle and facilitate withdrawal or flight from unachievable ambitions, acceptance of the new status quo, and inhibition of further futile competition so as to avoid excessive costs (Price et al., 1994; Sloman et al., 2003).These functions are reflected in the motivational, physiological, affective, cognitive and behavioural components of the IDS, including negative cognitions concerning personal adequacy and ability to succeed (e.g., Gilbert & Allen, 1998; Sloman, 2000), decreased motivation for continued competition with opponents who are perceived to be more powerful, thereby protecting individuals from injury (Sturman & Mongrain, 2000b), a toning-down of the positive (reward-orientated) affect system (e.g., Gilbert & Allen, 1998; Sloman et al., 2003), behavioural inhibition and hypervigilance (e.g., Shively, 1998; Shively et al., 1997; Sloman et al., 2003).

Central to the IDS theory is the suggestion that human competition for evolutionarily meaningful resources is often neither aggressive nor ritualised, as is the case with agonistic encounters in animals (Sloman et al., 2003). Instead, human competition and conflict are considerably more complex and diverse, not limited to direct interpersonal conflicts and more often based on attempting to elicit resources from others by competing to be socially *attractive* (e.g., attractive as a partner or friend) (Gilbert, 1989; Sloman et al., 2003). In this sense, the competition is to be chosen. Thus, the human IDS is thought to become activated in individuals via perceptions of being unable to compete in personally meaningful social arenas

because one lacks qualities that will win the positive attention of others. For example, perceiving oneself as unattractive, overweight, incompetent, unwanted, defective or unloved. In-keeping with evolutionary psychology theory (e.g., Buss, 2009), under optimal circumstances, the IDS is assumed to become active for only a brief period of time; deactivating once the individual has managed to escape, obtain help, or accept a particular defeat and move on to new goals or ambitions (Sloman, 2000). For example, an individual's IDS might deactivate when they escape an abusive relationship, elicit meaningful help from others or accept their job loss. Adaptive IDS activation therefore involves *flexibly* responding to internal and external feedback in order to avoid pursuing goals that cannot be obtained or would decrease the ability to survive and reproduce if they were pursued regardless of the danger or cost (Gilbert, 1998). Adaptive IDS activation would thus typically result in a person's ability to elicit positive resources from their environment being re-affirmed, bolstering their self-confidence and sense of control in life, and reducing anxiety (Sloman et al., 2003).

1.4.1. Psychological problems and the IDS

How, then, could the IDS be involved in the onset and maintenance of psychological problems? From the outset, it would appear to be a paradox that a supposedly adaptive evolutionarily mechanism could underpin psychological problems, particularly those severe or chronic enough to warrant referral to mental health services. As outlined above, IDS activation is thought to be a short-term, basic mechanism that evolved to manage competition for evolutionarily meaningful resources (Gilbert & Allan, 1998; Nettle, 2004). Therefore, it is suggested that psychological problems can emerge from the malfunction of the IDS response, characterised by intense, chronic, inflexible or inappropriate IDS activation (e.g., Gilbert, 1998; Nesse, 2000; Nettle, 2004; Sloman et al., 2003; Taylor et al., 2011a). The following discussion relates to defeat, entrapment and the IDS, since defeat and entrapment are thought to be lower-order manifestations of a higher-order IDS construct, and, as discussed previously, the three constructs are thought to form a "depressogenic feedback loop" which underlies some psychological problems (see Figure 1). Whether activation of the IDS becomes a clinical problem for a particular

individual at a particular time, will vary depending on a range of factors, which are now briefly reviewed.

1.4.2. Psychobiology

There is a fairly large body of evidence which has examined the psychobiology of the IDS in animals. Taylor et al (2011a) discusses this evidence and states that many of the psychobiological systems that have been linked to social rank in animal studies (i.e., serotonergic, dopaminergic, hypothalamic–pituitary–adrenal (HPA) axis) are also believed to underpin psychological problems in humans. However, there is currently no research regarding the psychobiology of the IDS in humans, leaving open the possibility that there may be differences between animals and humans.

1.4.3. Physical, social and internal environment

Like all evolutionary processes, the IDS is seen as a reactive mechanism that is sensitive to an individual's physical, social and internal environment (Buss et al., 1998; Tooby & Cosmides, 1990). Therefore, an individual's current context can influence the onset and maintenance of IDS activation. Chronic and excessive IDS activation might arise if, for example, others continue to attack even though an individual has submitted, or escape is blocked because a person is trapped in a defeating experience such as long-term imprisonment, chronic physical illness or a chronic psychological problem (e.g., psychotic experiences). An individual's historical context is seen to be important in potentially conferring vulnerability to unhelpful IDS activation. For example, it is suggested that previous and especially repeated activation of the IDS (e.g. via illness, trauma or stress), will progressively lower the threshold for IDS activation over time (Sloman et al., 2003) (e.g., via habituation). This calibration of the stress system to environmental demands (Lupien, McEwen, Gunnar & Heim, 2009; Teicher, Andersen, Polcari, Anderson, et al., 2003) is seen to be an adaptive response to anticipate and cope with stress and threat, especially in response to childhood maltreatment and adversity (see McCrory, De Brito & Viding, 2010). This process means that the threshold for perceiving defeat or entrapment will vary considerably across individuals and this variation may reach the point where situations or experiences that seem innocuous to some could be interpreted as defeating or entrapping by others (Williams et al., 2005). The

suggestion of a reduced threshold for IDS activation dependent on environmental experiences is consistent with the recurrent nature of psychological problems (e.g., Judd, 1997) and links previous aversive experiences and environments (particularly prolonged periods of distress or arousal) to vulnerability and onset of psychological problems (e.g., Lau et al., 2004).

With regards to beneficial or protective environmental factors, the degree to which (appropriate and positive) social support is available is thought to mediate an individual's experience of defeat and entrapment (Sloman et al., 2003). For example, having friends and family who know, understand and listen to an individual during a significant loss might help that individual to make sense of the experience and, in time, accept it and move on to new goals. Support for the hypothesis that social support may buffer against unhelpful IDS activation comes from various sources outside of the defeat and entrapment literature. For example, two separate reviews have concluded that social support may have beneficial effects and buffer against stressors via social (e.g., stress buffering), psychological (e.g., affective states, perception of control) and behavioural (e.g., health-promoting behaviour) mechanisms (Cohen & Wills, 1985; Uchino, Cacippo & Kiecolt-Glaser, 1996). One of these studies, a meta-analytic review of the relationship between social support and physiological functioning, concluded that social support can reliably cause beneficial effects to multiple aspects of physiological functioning, including the cardiovascular, neuroendocrine and immune systems (Uchino et al., 1996). Since the IDS is purported to be a psychobiological stress and threat-defence response (Sloman, 2000; Sloman et al., 2003), it seems plausible that social support may confer similar beneficial effects for the IDS response, perhaps even directly alleviating IDS activation. Likewise, Baumeister and Leary's (1995) 'need to belong' theory – purported to be a meta-theory encapsulating attachment and social support constructs – suggests that perceiving strong and stable interpersonal relationships (belonging) confers a range of positive effects, including protection against stressors, whilst lack of belonging is linked to a variety of ill effects on health, adjustment and well-being. The three reviews regarding the important benefits of social support and interpersonal belonging provide strong support for the idea that an individual's social environment may mediate experiences of defeat and entrapment.

1.4.4. Societal and cultural factors

Humans live in modern environments that differ profoundly from the ancestral environments to which our hominid ancestors adapted (Buss, 2009; Buss et al., 1998; Confer et al., 2010). Moreover, evolution by natural selection is a very slow process. Thus, the evolutionary mechanisms (such as the IDS) which humans are equipped with to navigate the world can be activated regardless of whether the adaptations currently serve the functions for which they were originally evolved (Buss, 1995; Confer et al., 2010). Modern environmental stimuli may therefore trigger, hijack, or exploit evolved psychological mechanisms (Confer et al., 2010) because of an 'evolutionary mismatch' or 'genome-lag' between the evolved mechanisms and current (Western) sociocultural contexts (Gatward, 2007). Examples of this 'evolutionary mismatch' might include the increased emphasis on competition that is apparent in developed capitalist societies and the role of the mass media in encouraging unreasonably high aspirations and standards (Nesse, 2000). These messages of competition from the environment could result in frequent and prolonged IDS activation, especially if an individual's expectations and values shift to account for these messages. Consistent with these suggestions, an interesting study in the literature found that the relationship between higher levels of defeat and PTSD held only for individuals from independent, typically Western cultures and not for those from interdependent cultures (Jobson & O'Kearney, 2009). As such, defeat in the context of PTSD may be less relevant to interdependent cultures (e.g., African, Asian, and South American) where emphasis is on the individual's dependence on his or her social environment rather than on personal agency and striving for success, as in independent cultures (e.g., Western European, North American; Jobson & O'Kearney, 2009).

1.4.5. Cognitive factors

An individual's particular social values or goals are thought to determine what could potentially be perceived as defeating or entrapping (Sturman & Mongrain, 2008b). This view suggests that perceptions of defeat and entrapment will most commonly arise in relation to events which *matter* to a particular individual at a particular time – whatever those events may be.

There has so far been one study which examined relationships between personality variables and perceptions of defeat and entrapment. Sturman and Mongrain (2008b) examined relationships between personality and perceptions of defeat before and after competitive sporting events. The authors found that individuals who were highly self-critical experienced a greater sense of defeat and an inability to accept defeat following a sporting loss. This finding was attributed to the competitive nature and unrealistically high standards for achievement of self-critical individuals (Sturman & Mongrain, 2008b). Conversely, individuals high in self-efficacy felt the impact of a sporting loss to a lesser degree, and showed greater resilience. The authors suggested that individuals who are confident in their abilities may believe that they will prevail in future encounters and therefore a single loss did not shake their self-belief and induce a perception of defeat (Sturman & Mongrain, 2008b). The novel design of this study was a particular strength, in that sport is thought to represent a ritualised form of the sort of agonistic competitive encounters that are so prevalent for humans. However, a limitation of the study is that it did not examine relationships between these personality variables and perceptions of entrapment.

As suggested above, it seems plausible that personality variables will confer specific vulnerability (or resilience) to perceptions of defeat or entrapment depending on the fit between an individual's personality and their context. For instance, it might be expected that individuals who particularly value goal achievement and individual autonomy (Beck, 1983), and people who are highly perfectionistic (Hewitt & Flett, 1991), would be most likely to perceive defeat and entrapment in relation to the loss or significant disruption of valued individual goals and choice. In contrast, individuals who particularly value interpersonal intimacy and affiliation (Beck, 1983), would be most likely to perceive defeat and entrapment in relation to the loss or significant disruption of a valued interpersonal relationship. Sloman et al (2003) similarly states, but from an attachment perspective, that an individual's attachment security may mediate the threshold for vulnerability to perceptions of defeat or entrapment, since individuals with an insecure attachment would be expected to react more strongly to stress. However, although plausible, these perspectives are purely theoretical at present and require empirical testing.

Another important cognitive factor thought to mediate perceptions of defeat and entrapment concerns individual perceptions of the ability to cope with potentially defeating or entrapping experiences (e.g., the ability to escape from aversive situations via individual agency), as well as perceptions of rescue factors and external sources of coping, often operationalized as social support (O'Connor, 2003; Rasmussen et al., 2010). This perspective – which overlaps with the literature reviewed in relation to self-efficacy and social support – suggests that individuals who perceive a high personal ability to cope and/or the presence of external sources of support, will be buffered against developing perceptions of defeat and entrapment (in effect raising their 'threshold' for perceiving defeat or entrapment).

The manner in which an individual copes with perceptions of defeat and entrapment themselves may also maintain IDS activation. For example, it is now well-established that thought suppression may have the counter-productive effect of making an avoided thought more likely to come to mind (Wenzlaff & Wegner, 2000). Therefore, if individuals employ thought suppression in relation to experiences of defeat and entrapment, it would be expected that these perceptions would increase.

1.5. Specific psychological problems

The manner by which the IDS is thought to contribute specifically to depression, suicidality, anxiety problems and PTSD (according to the model by Taylor et al., 2011a), will now be described.

1.5.1. Depression

Unipolar depression has been the clinical domain where the concepts of defeat and entrapment have so far received the most attention (Taylor et al., 2011a). The social rank model views the relationship between defeat and depression as occurring through activation of the IDS (Sloman, 2000) (see Figure 1). In the model, the IDS is initially activated via perceptions of defeat. Short-term IDS activation following defeat is thought to be adaptive as it acts as a motivator to disengage from a commitment that is not paying off (Nesse, 1998). However, clinical depression is proposed to occur in situations where an individual feels trapped in a defeated state because of low judgments of escapability. For example, an individual who strongly defines their sense of self based on their occupation might be expected to develop

depression if, due to financial cuts, the nature of their job changes significantly and stops being personally rewarding; however, the individual is unable to leave the job because they need to make ongoing mortgage payments. A "depressogenic feedback loop" is hypothesised to link perceptions of defeat, entrapment and IDS activation, wherein ongoing perceptions of entrapment are thought to reciprocally maintain the initial sense of defeat to produce a chronic or excessive IDS response (Gilbert, 2000; Sloman, 2000; Sloman et al., 2003). Within this interlocked state, normally adaptive features of the IDS, including low positive affect, diminished interest in acquiring resources, inhibition of confident or assertive behaviour and negative self-referent cognitions, escalate and stabilise to produce and maintain the characteristic symptoms of clinical depression (Sturman, 2011; Taylor et al., 2011a).

1.5.2. Suicidality

Several theoretical accounts of suicide suggest that suicidality is a response to the presence of perceptions of defeat, entrapment and no rescue (e.g., Baumeister, 1990; Johnson et al., 2008; Williams, 1997; Williams et al., 2005). When these three components are present, they activate what Williams calls a psychobiological "helplessness script". This helplessness script, which is analogous to the IDS, is suggested to be evolutionarily designed to aid survival by facilitating giving up and submission in defeated individuals (Williams, 1997; Williams et al., 2005). Suicide is therefore best understood as a reaction to chronic activation of this usually adaptive script. This process is speculated to occur in particular individuals, such as those who lack effective strategies for eliciting help (Taylor et al., 2011a).

Taylor et al (2011a) suggest that Williams' theory and IDS theory are similar enough for the two models to be collapsed into a single theoretical model. Both theories suggest that the pathway from initial defeat to suicidality follows the same route as for depression, mediated via IDS activation and perceptions of entrapment. However, it is suggested that some individuals develop suicidality as opposed to depression as a result of the presence of preexisting suicidogenic cognitive structures. These may include preexisting mental models for suicidal behaviour, beliefs about suicide, or suicidogenic schema (Johnson et al., 2008; Lau, Segal, & Williams, 2004; Pratt, Gooding, Johnson, Taylor, & Tarrier, 2010; Williams et al., 2005). For example, Williams' suicide theory draws heavily on the differential activation model of

Teasdale (1988), which proposes that particular thought processes can become associated with particular moods throughout the learning history of an individual (see Lau et al., 2004 for a review). For example, depression may occur alongside feelings of hopelessness, entrapment and agitation. These experiences may then become associated with each other for an individual, and from then on be reactivated in the form of a 'suicide schema' whenever the individual experiences a similar low mood. In this model, the 'suicide schema' is thought to be a semantic network of interconnecting stimulus, response and emotional states pertaining to suicide. When activated, this schema will trigger thoughts of suicidal behaviour as an escape strategy from an intolerable emotional or situational state. According to spreading activation theories, each time the suicide schema is activated, it becomes strengthened and elaborated to incorporate further cognitive, emotional or stimulus elements (Teasdale, 1988). Repeated activation of the suicide schema will lead to associations with an increasingly wide range of mood states and contexts and greater potential to be re-activated; thus increasing the risk of suicidal behaviour in the future. Exposure to suicide attempts by other individuals, particularly close associates, is one possible mechanism by which suicidogenic cognitive structures such as suicide schemata are thought to arise (Taylor et al., 2011a).

1.5.3. Anxiety problems

The IDS model suggests that anxiety problems may arise as a result of downstream cognitive, affective, and behavioural consequences of IDS activation which bias an individual's perception towards threat in such a way as to increase the likelihood of experiencing anxiety problems (Sloman, 2000; Taylor et al., 2011a). The possibility that both depressive and anxiety problems may result from the IDS response to defeat is in line with suggestions that depression and anxiety share common evolutionary origins (Nesse, 2000) and high comorbidity and symptom overlap (Mineka, Watson & Clark, 1998). Taylor et al (2011a) suggest two general pathways to link IDS activation, defeat and entrapment to the development and maintenance of anxiety problems.

A cognitive route is suggested, whereby perceptions of defeat and entrapment produce or increase threat appraisals that are themselves thought to be central to anxiety problems (see Butler, Fennell & Hackmann, 2008). This process occurs via

activation of negative cognitions concerning self-worth and adequacy as part of the IDS response (Sloman, 2000; Taylor et al., 2011a). Although these threat appraisals of self and others are adaptive in the short-term, in that they discourage risky behaviours that might result in a loss of status (Sturman & Mongrain 2000b), they may become entrenched if IDS activation is chronic or severe, thereby increasing the likelihood that future events will be (mis)appraised in an anxiety-producing manner.

The second route linking IDS activation, defeat and entrapment to anxiety problems is via the affective and behavioural aspects of the IDS response. These include characteristic features of anxiety such as arousal, hypervigilance for threat, behavioural inhibition and avoidance (Gilbert, 2000; Shively, 1998; Shively et al., 1997; Sloman et al., 2003). The affective and behavioural consequences of IDS activation are likely to have initially been adaptive in previous environments, in that they reduce the likelihood of further harm from others by keeping the individual primed for the risk of attack and ready to submit (Nesse, 1998). However, since such immediate dangers are less prevalent in modern society (Nesse, 1998), this adaptive function may be less well-suited to current contexts and may lead to anxiety responses that are excessive or chronic relative to the objective danger posed.

1.5.4. PTSD

As with other anxiety problems, it is suggested that downstream cognitive, affective and behavioural consequences of IDS activation can lead to PTSD. However, the hypothesized effect of perceptions of defeat and entrapment on PTSD differ from that of other anxiety problems in that it is the individuals' experience of one or more traumatic events, and the meaning and interpretations they have drawn regarding the trauma(s) that contribute to PTSD, rather than biases in the way future events are appraised, as is the case for other anxiety problems (Taylor et al., 2011a).

A cognitive pathway linking perceptions of defeat and IDS activation to PTSD experiences is hypothesised. It is suggested that if an individual experienced or experiences a perception of defeat whilst cognitively processing a trauma, this would engender negative cognitions concerning an individual's self-worth and autonomy, as well as an individual's capacity to cope with future problems and traumas (Dunmore et al., 1999, 2001; Ehlers et al., 2000). These cognitions are similar to those described as part of the IDS response to defeat (Sloman, 2000; Sloman et al., 2003), supporting

the idea that a common mechanism may be operating in both cases (Taylor et al., 2011a). As a consequence of these negative self-appraisals, the individual, rather than viewing the trauma as a discrete and time-limited event, experiences an ongoing sense of threat from the trauma, which is the hallmark of PTSD according to cognitive theories (Ehlers & Clark, 2000). The model also suggests that an individual may feel trapped by the ongoing experience of PTSD (threat) symptoms themselves (e.g., intrusive images, thoughts, flashbacks). A second route is suggested from perceptions of defeat to PTSD, mediated through the use of unhelpful coping strategies (e.g., avoidance of thinking or talking about the trauma, attempting to suppress intrusions), which themselves are thought to maintain PTSD symptoms (Ehlers & Clark, 2000; Ehlers et al., 2000).

1.6. Existing reviews of relationships between defeat, entrapment and psychological problems

To summarise the current literature on defeat and entrapment in humans, Taylor et al. (2011a) recently conducted a comprehensive narrative review of the role of these constructs in depression, suicidality, anxiety problems and PTSD. Their review provided convergent evidence across a range of designs, samples, and measures that perceptions of defeat and entrapment are important contributors to the psychological problems examined. However, it is also apparent from this review that the literature is very much in its infancy in terms of the nature of the empirical studies that have been conducted. These points, and how they relate to clinical interventions as well as the future development of the literature, will be discussed in more detail in chapter four. The main conclusions of the Taylor et al. (2011a) narrative review are summarised below.

1.6.1. Depression

The Taylor et al. (2011a) review found extensive evidence for a link between perceptions of defeat and entrapment and depression across a range of life event studies and self-report measures, and across a range of clinical and nonclinical samples. These relationships held when studies controlled for potential confounding variables (e.g., psychotic symptoms, caregiver stress, pain intensity, hopelessness, health anxiety, rumination, worry, catastrophizing) (Gilbert & Allan, 1998; Gilbert et

al., 2005; Tang et al., 2010). Qualitative investigations were consistent with the quantitative research.

1.6.2. Suicidality

The Taylor et al. (2011a) review found convergent evidence, across a number of clinical and nonclinical samples, that perceptions of defeat and entrapment are associated with an increased risk of suicidality. The studies reviewed were relatively robust in methodology and controlled for a range of confounding variables (e.g., hopelessness, anxiety and depression). Many used multivariate and mediational analyses to demonstrate that defeat and entrapment have a proximal role in the mechanisms underlying suicidality. The qualitative research was consistent with these quantitative findings. However, because only eight studies examined the link between perceptions of defeat and entrapment and suicidality, Taylor et al. (2011a) stated that their conclusions are preliminary.

1.6.3. Anxiety problems

The Taylor et al. (2011a) review found little evidence for a link between perceptions of defeat and entrapment and anxiety problems. Six cross-sectional studies across a variety of clinical and nonclinical samples presented mixed results, so firm conclusions could not be drawn. Further analyses, including partial correlations controlling for depressive symptoms (Gilbert et al., 2002) and multiple regression analyses (Sturman & Mongrain, 2005), failed to identify significant effects, raising the possibility that initial relationships between defeat and entrapment and anxiety problems may have been an artifact of the overlap of depression and anxiety symptoms. Two studies were reviewed which demonstrated that patients with psychosis, who were also classified as being socially anxious, had more extreme perceptions of entrapment than those without comorbid social anxiety, even when covarying for depressive and psychotic symptoms. Two studies presented convergent qualitative and quantitative evidence of a relationship between pain-related defeat and the severity of anxiety symptoms.

1.6.4. PTSD

The Taylor et al. (2011a) review found strong convergent evidence across prospective, cross-sectional and retrospective designs, and both self-report and narrative-based measures, that processing traumatic experiences as psychologically

defeating increased a person's risk of developing PTSD symptoms. As with their review of anxiety problems, Taylor et al. (2011a) noted that a challenge to the validity of their conclusions was the inconsistent control of depressive symptoms in the literature, which means that the relationship between defeat and PTSD may have been confounded with the relationship between defeat and depression. However, Taylor et al. (2011a) state that based on the consensus of the eight studies reviewed, there is evidence to cautiously suggest that the link between defeat and PTSD is not an artifact of depression.

1.7. Longitudinal evidence

The vast majority of the studies included in the Taylor et al. (2011a) narrative review are cross-sectional. This paucity of longitudinal and experimental designs has implications for the validity of the IDS model; a point that is examined further in chapter four. The available longitudinal research will now be reviewed to provide an indication of temporal precedence and potentially draw inferences concerning the direction of causal effects between defeat and entrapment and the four psychological problems (Garber & Hollon, 1991). Characteristics of the available prospective and longitudinal studies are summarised in Table 1.

1.7.1. Depression

Four studies have examined the relationship between perceptions of entrapment and later depression. One of these examined the recurrence of major depressive disorder over a sixteen month period in a sample of formerly depressed students. Baseline scores on a combined IDS variable derived from assessments of perceived entrapment and negative social comparison (the extent to which individuals feel socially attractive relative to others and the degree to which they perceive themselves as an insider or outsider: Allan & Gilbert, 1995), significantly predicted the recurrence of depression at follow-up, after adjusting for number of previous episodes and past depression (Sturman & Mongrain, 2008a). Unfortunately, this study reports unstandardized beta statistics, so it is not possible to determine the size of the predictive effect (Field, 2005a). In a very recent study which used an economically deprived community sample, a combined defeat and entrapment variable significantly predicted increases in depressive symptoms twelve months later when adjusting for

baseline symptoms (Griffiths et al., 2013). This effect (β = .25) was small to medium by Cohen's (1988) criterion.

Two prospective studies have used samples of individuals with a psychosis or bipolar disorder diagnosis. One of these found that appraisals of psychiatric experience (e.g., psychosis) as entrapping at baseline predicted depressive symptoms at follow-up thirty months later, even when covarying for psychotic symptoms, problem-related variables (e.g., duration and age of onset), and treatment-related variables (e.g., medication) (Rooke & Birchwood, 1998). This effect (β = .39) was medium to large by Cohen's (1988) criterion. Change in appraisals of entrapment over time were themselves predicted by the overall number of compulsory admissions to hospital and the number of admissions within the last twelve months, suggesting that perceptions of entrapment originated partly in the experience of certain aspects of psychiatric treatment that promote helplessness and defeat. A second study explored postpsychotic depression (PPD), a subtype of depression emerging after a psychotic episode has subsided (Iqbal et al., 2000). This study tracked individuals following recovery from an initial psychotic episode over a twelve month period and demonstrated that more extreme appraisals of entrapment at baseline increased the risk of subsequently developing PPD. This effect (r = .23) was small to medium by Cohen's (1988) criterion.

1.7.2. Suicidality

One study has examined the relationship between perceptions of defeat and entrapment and suicidality over time. Taylor et al (2011b) found evidence that baseline defeat, but not entrapment, significantly predicted suicidality twelve months later when adjusting for baseline suicidal ideation and depressive symptoms. This effect (β = .57) was large by Cohen's (1988) criterion. In contrast, baseline suicidal ideation did not significantly predict changes in defeat or entrapment. This shows that a unidirectional relationship exists between defeat and suicidal ideation over time. However, some limitations must be noted for the interpretation of these results. First, the sample consisted of predominantly female students, which limits the generalizability of the findings. Moreover, only suicidal ideation was measured. Therefore, it remains unclear whether perceptions of defeat and entrapment also predict suicide attempts or completions.

Table 1. Characteristics of longitudinal studies relating perceptions of defeat and entrapment to depression, suicidality, anxiety problems and PTSD.

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
				Depression				
Griffiths et al. (2013)	Community sample from low SES backgrounds	195	Defeat and Entrapment Scales	Defeat and entrapment	CES-D, STAI: State subscale	36.90 (8.3)	Not reported	UK
Iqbal et al. (2000)	Schizophrenia spectrum disorder	105	Mental defeat rated from narrative	Internal entrapment	BDI	Not reported	Not reported	UK
Rooke & Birchwood (1998)	Schizophrenia spectrum disorder	47	PBIQ	Internal entrapment	BDI	42.1 (12.7)	80.9	UK
Sturman & Mongrain (2008a)	Formerly depressed students	146	Defeat and Entrapment Scales	Internal and external entrapment	SCID: Depression	Not reported	71.9	Canada
				Suicidality				
Taylor et al. (2010b)	University undergraduates with past or current suicidal ideation	93	Defeat and Entrapment Scales	Defeat and entrapment	Suicidal Behaviors Questionnaire–Revised	23.45 (7.1)	81.7	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
				Anxiety problems				
Griffiths et al. (2013)	Community sample from low SES backgrounds	195	Defeat and Entrapment Scales	Defeat and entrapment	CES-D, STAI: State subscale	36.90 (8.3)	Not reported	UK
				PTSD				
Dunmore et al. (2001)	Assault survivors	57	MDTS	Defeat	PTSD Symptom Scale Self-Report	Not reported	54.4	UK
Kleim et al. (2007)	Assault survivors	205	MDTS	Defeat	SCID	35 (11.5)	32.0	UK

Note. BDI = Beck Depression Inventory, CES-D = Center for Epidemiological Studies Depression Scale, MDTS = Mental Defeat during Trauma Scale, PBIQ = Personal Beliefs about Illness Questionnaire, SCID = Structured Clinical Interview for DSM-IV Disorders, STAI-State = State Trait Anxiety Scale – State subscale.

1.7.3. Anxiety problems

A recent study by Griffiths et al (2013) is the only one to have examined whether defeat and entrapment predict anxiety problems over time. Using an economically deprived community sample, Griffiths et al (2013) demonstrated that a combined defeat and entrapment variable significantly predicted increases in state anxiety symptoms twelve months later when adjusting for baseline symptoms (Griffiths et al., 2013). This effect (β = .29) was medium by Cohen's (1988) criterion. One limitation of this study is that it did not examine whether defeat and entrapment predict trait anxiety over time, which has greater clinical relevance.

1.7.4. PTSD

Two longitudinal studies have examined the relationship between mental defeat and later PTSD. One study found that defeat at baseline predicted PTSD severity at nine months following an assault, when controlling for initial severity of PTSD symptoms (Dunmore et al., 2001). This effect (r = .30) was medium by Cohen's (1988) criterion. However, there was no significant relationship between baseline defeat and PTSD symptoms at six months, although the effect size was very similar to that observed for the nine months' follow-up (r = .28). The relatively small sample size (n = 57) raises the possibility that there was not enough power in the analyses to render this slightly smaller effect statistically-significant. A second study examined a variety of biological, cognitive, demographic, and other risk factors for their ability to predict the occurrence of a PTSD diagnosis at six months following trauma, when controlling for baseline acute stress disorder symptoms (Kleim et al., 2007). Appraisals of defeat concerning the trauma experience emerged as a significant predictor of PTSD, with a large (r = .48) effect-size (Cohen, 1988).

1.7.5. Summary of longitudinal evidence

Given the literature's infancy, very few studies to date have examined the longitudinal relationship between defeat and entrapment and the four psychological problems. Three studies (Griffiths et al., 2013; Iqbal et al., 2000; Rooke & Birchwood, 1998; Sturman & Mongrain, 2008a) have demonstrated that entrapment significantly predicts later depression, with small to medium effect sizes (Cohen, 1988). One study (Griffiths et al., 2013) demonstrated that defeat and entrapment significantly predicted later depression, with a small to medium effect size (Cohen, 1988). One study (Taylor

et al., 2011b) demonstrated that defeat, but not entrapment, significantly predicted later suicidality, with a large effect size (Cohen, 1988), and that baseline suicidal ideation did not significantly predict later defeat or entrapment. This is the only study in the literature to have examined the possibility of a bidirectional relationship between defeat and entrapment and the four psychological problems. One study (Griffiths et al., 2013) demonstrated that defeat and entrapment significantly predicted later state anxiety symptoms, with a medium effect-size (Cohen, 1988). Two longitudinal studies (Dunmore et al., 2001; Kleim et al., 2007) demonstrated that defeat significantly predicts later PTSD, with medium to large effect sizes (Cohen, 1988). Overall, there is some preliminary evidence to suggest, consistent with IDS theory, that perceptions of defeat and entrapment potentially cause depression, suicidality, anxiety problems and PTSD. However, it is not clear whether the reverse picture may also be true for some or all of these psychological problems and whether these conclusions are applicable to different populations and clinical presentations (e.g., symptoms versus diagnosis). There are various other limitations to this data, which are examined in chapter four with a view to future research.

1.8. Experimental evidence

There is only one experimental study available in the literature. This study examined whether a depressive mood induction causally influenced perceptions of defeat and entrapment (Goldstein & Willner, 2002), testing the possibility that perceptions of defeat and entrapment may sometimes arise as a consequence of depressed mood. The authors' induced positive and depressed moods in 32 non-depressed[1] female undergraduates. They found that the depressive mood induction caused medium to large-sized (Cohen, 1988) statistically-significant increased perceptions of defeat and entrapment (r = .25 - .46). In contrast, the positive mood induction caused a small/small to medium-sized (Cohen, 1988) decrease in perceptions of defeat and entrapment (r = .15 - .24). This change was statistically-significant for defeat but not internal entrapment, external entrapment or total entrapment score. Interestingly, individuals with elevated depression or internal

[1] Participants were deemed clinically depressed according to the standard cut-off criterion for the Beck Depression Inventory.

entrapment scores prior to the depressive mood induction showed the greatest increases in internal entrapment after the mood induction, whereas individuals with low depression or internal entrapment scores prior to the depressive mood induction showed very little increase following the negative mood induction. These finding are consistent with more recent theories of depression which suggest a reciprocal relationship between symptoms of depression (e.g., mood) and negative cognitions. They are also consistent with the IDS model's suggestion of a "depressogenic feedback loop", whereby a depressed mood strengthens perceptions of defeat and entrapment and vice-versa; representing a potentially important mechanism for the maintenance of a depressive episode. Two limitations with this study are noteworthy. First, the entirely female sample raises the possibility that these findings may not be generalizable to men. Second, the fact that only depressive and positive mood induction conditions were tested does not comment on the potential role of other moods that are considered clinically important, such as anxiety and disgust (e.g., Davey & Bond, 2006).

1.9. Why focus on depression, suicidality, anxiety problems and PTSD?

The present meta-analysis confined its focus to quantifying relationships between defeat and entrapment and four common psychological problems (depression, suicidality, anxiety problems and PTSD) for three reasons. First, from a clinical standpoint, depression and anxiety problems are the commonest mental health problems in adults (e.g., Bromet, Andrade, Hwang, Sampson, et al., 2011; Kessler, Petukhova, Sampson, Zaslacsky, et al., 2012; Kessler & Wang, 2008) and suicidality is an extremely concerning psychological experience (World Health Organisation, 2002). For example, suicide was the 10th leading cause of death among all age groups in 2010 (Centers for Disease Control and Prevention, 2012). Therefore, prioritising examination of these particular psychological problems will maximise the potential benefit to NHS mental health services. Second, theory linking defeat and entrapment to psychological problems is strongest for depression, suicidality, anxiety problems and PTSD (e.g., Taylor et al., 2011a), providing testable hypotheses (see below) and a clear theoretical framework to guide the meta-analysis. Lastly, since very few studies have examined the role of defeat and entrapment in other psychological problems

(e.g., psychosis), it would not be possible to examine potential moderator variables for these studies (Borenstein, Hedges, Higgins & Rothstein, 2009).

1.10. The present study

The present meta-analytic review extends the contribution of the narrative review by Taylor et al. (2011a) in several important respects. Most importantly, it quantifies for the first time, the size and consistency of the population effect size (the 'true' effect) for each of the relationships between defeat and entrapment and depression, suicidality, anxiety problems and PTSD. Although Taylor et al. (2011a) concluded that there was a "strong evidence base" for relationships between perceptions of defeat and entrapment and these psychological problems (p. 415), meta-analysis uniquely allows the population effect sizes for these relationships to be estimated.

Next, as discussed above, the conceptualisation of defeat and entrapment has been a matter of some debate and the two constructs have more recently been conceptualised as lower-order manifestations of a single higher-order latent IDS variable (see Sturman, 2011; Taylor et al., 2009, 2011a). This meta-analysis therefore examines relationships between perceptions of defeat and entrapment and the four psychological problems both when defeat and entrapment are combined and when these variables are separated as defeat, internal entrapment and external entrapment. In addition, it is not yet known whether defeat and entrapment have different-sized correlations with the four psychological problems, as suggested by IDS theory, and whether particular moderator variables attenuate or accentuate the consistency of population effect size estimates. Answering these questions may help progress current theoretical explanations and guide the future expansion of the literature. Potential findings would also have direct clinical relevance for incorporating perceptions of defeat and entrapment into clinical assessment, case conceptualisation, intervention and prevention. The current meta-analysis provides the first direct test of these questions. From a theoretical perspective, depression is thought to arise directly through IDS activation, where depression is simply an IDS that has been active for longer than is functionally useful (Price et al., 1994; Sloman, 2000; Sturman, 2011; Taylor et al., 2011a). In contrast, relationships between defeat and entrapment and

anxiety problems and PTSD are thought to arise as a consequence of IDS activation. Therefore, it was expected that relationships between defeat and entrapment and depression would be stronger than relationships between defeat and entrapment and suicidality, anxiety problems and PTSD.

Three sample characteristics are examined as potential moderators: mean age, gender composition of samples (i.e. percentage of the sample female), and clinical status of samples (i.e. community versus clinical sample). IDS theory makes very little comment regarding the potential influence of demographic variables; however, it was expected that perceptions of defeat and entrapment would be more common or stronger in females due to greater exposure to adversity and differing reactions to stressors (Nolen-Hoeksema, 1990, 2001). No specific a priori hypotheses were made in relation to age and these analyses were therefore undertaken on an exploratory basis. It was expected that perceptions of defeat and entrapment would be equal in clinical and community samples since these variables are thought to be continuous and to have linear relationships with psychological problems. It was also important to examine measurement instrument differences as a potential moderator, since varying precision of the scores obtained from particular measurement instruments could produce spurious effect size differences (Baguley, 2009). There were enough effect sizes to examine measures of defeat and entrapment and depression as potential moderators. There was no strong theoretical or empirical reason to make a priori directional hypotheses regarding particular measures so these analyses were also undertaken on an exploratory basis. Lastly, year of publication was examined as a moderator because some research suggests that date of publication may influence effect sizes (Abramowitz, 1997). If more recent studies, for example, produced larger effect sizes, it would be important to consider why this was the case and to account for this in future research designs.

1.10.1. Aims of the present study

The present study has three major aims:

(1) To utilise a meta-analytic approach to quantify the size and consistency of the population effect size for relationships between perceptions of defeat and entrapment and depression, suicidality, anxiety problems and PTSD.

(2) To examine whether moderator variables attenuate or accentuate the strength of these relationships.

(3) To examine the extent to which publication bias may have inflated population effect size estimates.

CHAPTER TWO: METHOD

2.1. Selection of articles

The studies included in this meta-analysis were obtained from two literature searches: the results of the literature search by Taylor et al. (2011a), covering research until April 2010, and an additional search conducted by the author between April 2010 and November 2012.

2.1.1. Search terms

The keyword search terms used by Taylor et al. (2011a) in their literature search were also used here. These included combinations of the following keyword terms *defeat, entrapment,* and *trapped,* in combination with keywords indexing *anxiety, PTSD, depression,* and *suicide* (*depres$, anxi$, suicid$, stress, symptoms, distress*). *$* denotes the use of truncated search terms, which broaden the search by looking for all words that begin with the same stem but have different endings. Abstracts, keywords and titles were searched.

2.1.2. Databases searched

Three psychological and medical literature databases (PsycINFO, MEDLINE and Web of Knowledge) were searched to identify English-language articles reporting a relationship between defeat and/or entrapment and the four psychological variables:

(1) PsycINFO (1880s onwards) is an electronic abstracting and indexing database compiled by the American Psychological Association. It searches behavioural science and mental health literatures and has more than 3.4 million records which are updated on a weekly basis. PsycINFO covers journal articles (approximately 2,500 journals), books and dissertations from more than fifty countries.

(2) MEDLINE (1946 onwards) is the leading electronic bibliographic database of articles in the life sciences, with a concentration on biomedicine and health. It is compiled by the National Library of Medicine in the United States and has over 19 million records from over 5,600 worldwide journals, with weekly updates.

(3) Web of Knowledge (1945 onwards) is provided by Thomson Reuters and is the largest accessible citation database. It provides access to multiple databases, cross-disciplinary research, and in-depth exploration of specialized subfields, encompassing 11,261 journals and 12,000 conferences each year.

2.1.3. Additional search strategies

To ensure that the literature search was as comprehensive as possible, a number of additional search strategies were also undertaken. Secondary sources such as review articles, book chapters and the reference sections of selected articles were examined. An additional database search was also conducted for articles that had developed measures of defeat or entrapment. To reduce the effect of publication bias, researchers with one or more publications involving defeat and/or entrapment were emailed to request unpublished research (e.g., forthcoming papers) for potential inclusion. A search of Abstracts from conferences was also conducted to potentially locate additional unpublished work in the area.

2.1.4. Outcome of the literature search

The literature searches yielded a preliminary database of 281 published studies. This included the 51 studies included in the Taylor et al. (2011a) narrative review and two forthcoming studies (Griffiths et al., 2013; Troop, 2013). This initial pool of studies was then reviewed to determine eligibility for inclusion in the meta-analysis.

2.1.5. Screening potential studies for inclusion

Initially, the Abstracts of all 281 identified articles were read to determine whether each study met the inclusion criteria. In instances where more information was required, the full text of the article was also read. To ensure decision-rule consistency, all eligible studies were carefully reviewed by two authors (Andy Siddaway and Dr Peter Taylor, University of Manchester) using the inclusion and exclusion criteria described below, with 100% agreement.

2.2. Inclusion and exclusion criteria

Inclusion criteria for quantitative studies were that they were (1) original research articles; (2) used adult, human participants (18 years+); (3) were written in English; (4) included a quantitative measure of subjective defeat, entrapment or defeat

and entrapment combined; (5) included a symptom-based or diagnostic measure of depression, suicidality, anxiety problems or PTSD; (6) employed measures with adequate reliability and validity, as demonstrated by publication of psychometric properties in a peer-reviewed journal; (7) reported single-*df*/bivariate relationships (Rosenthal, 1991); and (8) reported the correlation coefficient *r* or else provided sufficient statistical information that could be used to compute this statistic (as per Borenstein et al., 2009). Authors of papers with unclear statistical information were contacted to request further information. If this was not provided, these papers were excluded from the analysis. No dissertations were identified for inclusion; a publication bias analysis is conducted later to address the important concern that unpublished data from dissertations or other research studies might have findings that could alter the conclusions of the meta-analysis (Vevea & Woods, 2005).

The inclusion and exclusion criteria adopted here meant that some studies that were included in the Taylor et al. (2011a) narrative review were not suitable for inclusion here. For example, LeBlanc, Driscoll and Pearlin (2004) was excluded because it employed unvalidated measures, Park, Ryu, Han, Kwon, et al. (2010) was excluded because it used a youth sample and Kendler et al. (2003) was excluded because it reported non-bivariate relationships. Overall, the inclusion and exclusion criteria meant that thirteen studies were excluded from the current meta-analysis which had been included in the Taylor et al. (2011a) narrative review (Birchwood et al., 2005; Broadhead & Abas, 1998; Brown et al., 1995; Ehlers et al., 1998; Gilbert et al., 2001; Hagen, 2002; Kendler et al., 2003; Kidd, 2006; Leblanc et al., 2004; O'Connor, 2003; Park et al., 2010; Rooke & Birchwood, 1998; Sturman & Mongrain, 2005). Nine studies were included in the meta-analysis that were not included in the Taylor et al. (2011a) review (Garcia-Campayo, Rodero, Lopez del Hoyo, Luciano, et al., 2010; Griffiths et al., 2013; Panagioti, Gooding, Taylor & Tarrier, 2012; Sturman, 2011; Taylor, Wood, Gooding & Tarrier, 2011b; Trachsel, Krieger, Gilbert & Holtforth, 2010; Troop, 2013; Troop & Hiskey, in press; Troop, Andrews, Hiskey & Treasure, 2013).

2.2.1. Final pool of studies included in the meta-analysis

Details of the literature sifting process are shown in Figure 2. Of the 281 published articles yielded by the literature searches, 38 studies (0.21%) met all of the

requirements for inclusion in the meta-analysis. Studies included in the meta-analysis are identified with an asterisk in the Reference section and described in detail in Table 2.

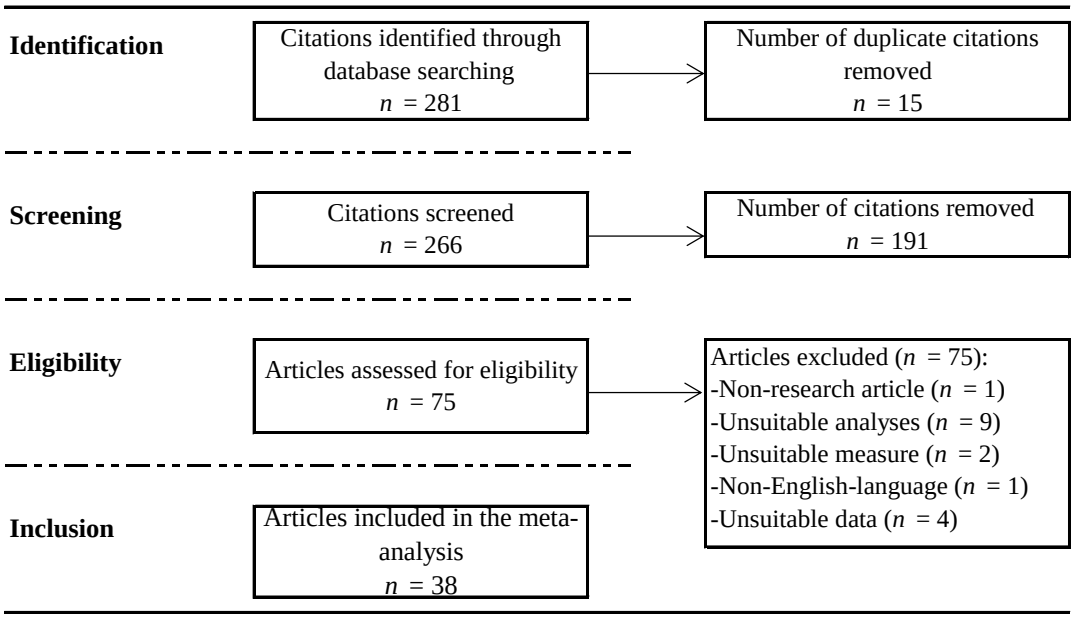

Figure 2. Flow diagram of the study selection procedure.

2.3. Multiple effect sizes and samples from the same study

Because individual effect sizes were very similar (Marascuillo, Busk & Serlin, 1988), the simple mean was computed when studies reported multiple effect sizes for the same relationship (e.g., different measures of depression and their respective relationships with defeat) (Rosenthal, 1991). Two of the included studies reported multiple effect sizes for the same relationship based on two different sample sizes but not different samples. This was due to different group comparisons (Dunmore et al., 1999) or different formats of a measure (paper and pencil vs. online: Trachsel et al., 2010). For these studies, a weighted mean was computed to account for the sampling accuracy of each effect size. Two studies (Gilbert & Allen, 1998; Gilbert et al., 2002) each contributed two separate samples (undergraduate and clinical). Different articles reporting analyses from the same dataset were included if the studies provided effect size estimates for different relationships. Cross-sectional data from Time 1 only was used from longitudinal studies which presented multiple assessments of symptoms over time.

20 of the 38 included studies contributed multiple effect sizes. For example, Garcia-Campayo et al. (2010) contributed an effect size for the relationship between defeat and depression and an effect size for the relationship between defeat and anxiety problems. Inclusion of multiple effect sizes from the same study violates the statistical assumption underpinning meta-analyses that data points are independent, potentially introducing a slight bias (Borenstein et al., 2009). Multivariate meta-analysis (e.g., multi-level modelling) provides the optimal solution to this problem. However, none of the standard meta-analysis packages offers an option for 'clustered data sets' of this nature, so it is not possible to take the dependency between some of the data points into account here. The potential impact of this source of bias for the interpretation of results was noted and is taken into account in the discussion[2].

2.4. Methodological considerations

2.4.1. Standardised effect sizes

Effect sizes provide a measure of the size and direction of an effect or association between two or more variables. Standardised effect sizes are the metric used in meta-analysis because (1) they are relatively resistant to sample size influence and thus provide a truer measure of the magnitude of effects than null-hypothesis significance testing (Ferguson, 2009), and (2) they enable comparison of different studies that have measured variables using different scales of measurement (Baguley, 2009; Lipsey & Wilson, 2001).

2.4.2. Interpreting the size of Pearson's *r*

Pearson's correlation coefficient *r* was used as the effect size metric in this meta-analysis. Cohen (1988) provided the following widely adopted guidelines for interpreting *r* in the social sciences *r* = .10 (a small effect, indicating 1% overlap in variance between two variables), *r* = .30 (a medium effect, indicating 9% overlap in variance between two variables), *r* = .50 (a large effect, indicating 25% overlap in

[2] Chapter three shows that meta-analyses were performed when defeat and entrapment were combined as a single variable (analyses used multiple effect sizes from the same study) and when defeat and entrapment were separated (analyses did not involve using multiple effect sizes from the same study). Section 4.3.1. discusses the fact that the similarity in the two sets of results provides some confidence that the results were not substantially biased by the inclusion of multiple effect sizes. For the manuscript to be submitted for publication, it is hoped that the author's principal supervisor will be able to use a customised approach to multi-level modelling to correct for this potential bias.

variance between two variables). Although these guidelines can be a useful rule of thumb to assess the importance of an effect (regardless of its statistical significance), the fact that they are guidelines means that they should not be interpreted rigidly; it is necessary to evaluate the effect size within the context of the research domain in which it is being used in order to assess its importance or practical significance (Rosenthal, 1991).

2.5. Meta-analytic models

There are two ways to conceptualize meta-analysis: fixed-effect and random-effects models (see Borenstein et al., 2009; Hedges, 1992; Hedges & Vevea, 1998; Hunter & Schmidt, 2000). The fixed-effect conceptualization assumes that the studies included in a meta-analysis are sampled exhaustively from a population of interest with a fixed but unknown effect size. Consequently, sample effect sizes are expected to be homogenous because they come from the same population which has a fixed average effect. Therefore, any variation in the distribution of effect sizes is thought to be due to sampling error alone (Cohn & Becker, 2003). A fixed effect meta-analytic model might be appropriate if all studies included in a meta-analysis were performed by the same researcher using the same population and methods. An example might be if a pharmaceutical company enrolled 1000 patients for a clinical trial and divided them among ten cohorts of 100 patients each. If these ten cohorts are known to be identical in all important respects, it would be reasonable to assume that the true effect (the population effect size) would be the same for all ten studies (Borenstein et al., 2009) and to apply a fixed-effect model.

The random-effects conceptualisation assumes that population effect sizes vary randomly from study to study; that is, the studies included in a meta-analysis are thought to each be sampled from a distribution of population effect sizes that naturally vary in their average effect sizes (Hedges & Vevea, 1998; Hunter & Schmidt, 2000), related to factors such as the methods used and the context of the research (Cohn & Becker, 2003; Field, 2003; Hunter & Schmidt, 2000; National Research Council, 1992). In this sense, the studies included in a meta-analysis can be thought of as being sampled from a "superpopulation" of possible effects (Hedges, 1992) and the overall effect size is therefore assumed to be an estimate of the mean of the superpopulation's

effect size distribution. Returning to the pharmaceutical company example, even though all studies to be included in the theoretical meta-analysis were performed by the same pharmaceutical researchers using the same population and methods, it is likely that there will be some differences from study to study such that the true effect differs between studies (Borenstein et al., 2009). If differences do exist between studies and could exert an influence on the population effect size estimate, then a random-effects model is a better fit of the data (Borenstein et al., 2009).

From a statistical viewpoint, the main difference between the two meta-analytic models is in the source of error that is accounted for. In fixed-effect models, there is within-study error as a result of sampling studies from a population of studies. This error also exists in random-effects models but, in addition, between-study error is also assumed as a result of sampling studies from individual sub-populations that make up a superpopulation.

2.5.1. Rationale for using a random-effects model

Following recommendations by Borenstein et al. (2009), Lipsey and Wilson (2001) and Field and Gillett (2010), a random-effects model of meta-analysis was used here for two principal reasons. First, real-world social science research is typically conducted by a range of researchers using different populations and methods (Borenstein et al., 2009) and therefore contains variability in effect sizes as the norm (Barrick & Mount, 1991; Borenstein et al., 2009; Field, 2003, 2005b; Hunter & Schmidt, 2000; National Research Council, 1992; Osburn & Callender, 1992). Furthermore, given the variability in the methods, settings and recruitment procedures of the included studies, it is difficult to conceive of one true effect size for each of the relationships between defeat and entrapment and the four psychological problems. Second, fixed-effect models are appropriate for making inferences that extend only to the studies included in the meta-analysis. Since social science researchers hope to generalise their conclusions to studies that may be done subsequently, could have been done earlier, or may have already been done but are not included among the

observed studies, a random-effects model is indicated (Borenstein et al., 2009; Cohn & Becker, 2003; Hedges & Vevea, 1998; Hunter & Schmidt, 2000)[3].

2.6. Determining between-study heterogeneity

Two sources of variability might cause heterogeneity among the studies included in a meta-analysis. One is variability due to sampling error (within-study variability). This variability is always present in a meta-analysis because every study uses a different sample (Huedo-Medina, Sánchez-Meca, Marín-Martínez & Botella, 2006). The other source of variability is between-studies variability, which can appear in a meta-analysis when there are true differences among the population effect sizes estimated by individual studies. Between-studies variability is due to the influence of a potentially large number of characteristics that vary among studies, such as participants, measures, treatment conditions, study design, and so on (Cohn & Becker, 2003; Field, 2003; Hunter & Schmidt, 2000; National Research Council, 1992).

2.6.1. The *Q* test

The *Q* test is the most commonly used method of assessing whether there is true (i.e. between-study) heterogeneity in a meta-analysis. The basic principle is that similar studies will have drawn subjects from the same population (or superpopulation) and so will have similar effect sizes. Therefore, statistical heterogeneity is assumed if there is greater variation across studies than would be expected via sampling error alone. A statistically significant *Q* test indicates significant between-study heterogeneity. The source of this variation can then be examined by using distinct features of the studies (e.g., average age of samples) as moderator variables.

2.6.2. I squared (I^2)

A shortcoming of the *Q* statistic is that it has poor power to detect true heterogeneity among studies when there are a small number of studies (as is the case here), and excessive power to detect negligible variability when there are a large number of studies (see Huedo-Medina et al., 2006). Moreover, the *Q* statistic does not

[3] It is no longer accepted practice to use the results of heterogeneity tests to determine the model of meta-analysis because (i) homogeneity tests have low power to detect genuine variation in population effect sizes (Hedges & Pigott, 2001) and (ii) as discussed, social science researchers wish to make inferences that extend beyond the studies included in meta-analyses.

inform researchers of the *extent* of true heterogeneity, only of its statistical significance. I^2 was recently proposed as a useful alternative to Q and τ^2 (τ^2 estimates between-study variance). I^2 provides a way of gauging the magnitude and the statistical significance of heterogeneity in a meta-analysis. I^2 is easily interpretable as the percentage of the total variability in a set of effect sizes that is due to true heterogeneity (i.e. between-studies inconsistency) (see Hedges & Pigott, 2001; Higgins & Thompson, 2002; Higgins, Thompson, Deeks & Altman, 2003; Huedo-Medina et al., 2006). For example, a meta-analysis with $I^2 = 0$ indicates that all the variability in effect size estimates is due to the sampling error within studies. Evidence of true heterogeneity is apparent when the confidence interval around I^2 does not include the 0% value (Higgins & Thompson, 2002).

2.7. Moderator variables

2.7.1. Replacement of missing data

Differences across studies in terms of the sample and study characteristics that were reported meant that there was some missing data for two of the moderator variables. Ten data points were missing for the gender composition moderator variable and eleven data points were missing for the mean age moderator variable (see Table 2). Moderator analyses for these two variables involved replacing missing values with the median from other included studies for each variable. Running the analyses with and without replacing the missing data points with the median value showed that the outcome was unchanged if missing values were not replaced.

2.7.2. Number of effect sizes for categorical moderator analyses

To ensure robust conclusions could be drawn, the recommendations by Borenstein et al. (2009) concerning sub-group sizes for categorical moderator analyses were followed. This recommendation states that moderators need to have ≥6 effect sizes in each sub-group in order to yield an acceptably precise estimate of between-study variance to make analyses meaningful.

2.7.3. Moderator information coded for each included study

To examine the potential role of different moderator variables in attenuating or accentuating the strength of relationships between defeat and/or entrapment and

depression, suicidality, anxiety problems and PTSD, the following information from each included study was coded and used in the analyses:

(1) The defeat and entrapment construct itself was examined as a categorical moderator variable by coding effect sizes into defeat and entrapment categories (defeat, internal entrapment, external entrapment, total internal and external entrapment). However, depression was the only psychological problem for which there were enough effect sizes in each of the defeat and entrapment categories to meet the recommendation by Borenstein et al. (2009) regarding the minimum size of moderator sub-groups. Therefore, moderator analyses of this variable for suicidality, anxiety problems and PTSD groups involved collapsing the entrapment sub-groups (internal entrapment, external entrapment and total internal and external entrapment) to form two moderator categories (defeat versus entrapment).

(2) Mean age was examined as a continuous moderator variable using the mean age in years (to two decimal places) of each included sample.

(3) Sample gender composition was examined as a continuous moderator variable using the percentage of the sample that were female (to two decimal places) from each included sample.

(4) Clinical status was examined as a categorical moderator variable by coding effect sizes into clinical and community categories. Samples were categorised according to the nature of the psychological problems experienced, rather than by some other criterion such as inpatient versus outpatient status, or whether samples were currently sectioned under the Mental Health Act, for example. There were not enough effect sizes to examine more than two categories of clinical status. Community samples consisted of undergraduates and office workers, both of whom had a low incidence and severity of psychological problems. Clinical samples consisted of samples that were (i) specifically recruited because of the presence of one or more severe psychological

problems (e.g., samples of individuals currently experiencing depression or psychosis; undergraduates currently experiencing suicidality) or (ii) at an elevated risk of experiencing one or more psychological problems (e.g., a community sample of individuals who had experienced a traumatic event).

(5) Study design was examined as a categorical moderator variable by coding effect sizes into cross-sectional and 'other' (longitudinal, prospective) categories.

(6) Measure of defeat and entrapment was examined as a categorical moderator variable by coding effect sizes into Defeat and Entrapment Scales (Gilbert & Allan, 1998) and 'other'. Other consisted of the Personal Beliefs about Illness Questionnaire, Mental Defeat During Trauma Scale, Pain Self Perception Scale, Custom Interview Concerning Entrapment, Mental Defeat Rated from Narrative, Carer's Entrapment Scale and the Carer Burden Scale – Entrapment subscale (see Table 2).

(7) Measure of depression was examined as a categorical moderator variable by coding effect sizes into Beck Depression Inventory (Beck, 1988; Beck, Steer & Brown, 1996), Center for Epidemiological studies Depression Scale (Radloff, 1977), Hospital Anxiety and Depression Scale (Zigmond, & Snaith, 1983) and 'other'. Other consisted of the Mood and Anxiety Symptoms Questionnaire, Structured Clinical Interview for DSM-IV Disorders, Calgary Depression Scale for Schizophrenia and the Self-Rating Depression Scale (see Table 2).

(8) Year of publication was examined as a continuous moderator variable using the year that each study was published.

Other sample and study characteristics such as the ethnic composition of samples and the percentage of the sample employed were only sporadically reported.

Therefore, there were not enough effect sizes to examine these as potential moderator variables.

2.8. Publication bias

Publication bias, or the 'file drawer problem' (Rosenthal, 1979), describes the tendency for the availability of research to depend on the results (Begg, 1994; Vevea & Woods, 2005). In a simple (and extreme) case, publication bias might manifest itself if only studies with results that are statistically-significant are published, and all other studies are not published (Rosenthal, 1979). Such a situation might arise either because researchers are less likely to write up and submit those results, or because journal editors and reviewers are less likely to accept them for publication (Vevea & Woods, 2005). If studies are published depending on the statistical-significance of their results, then it is likely that the studies not included in a meta-analysis will have different results from those that have been included. This would result in meta-analyses over-estimating population effect sizes, potentially leading to inappropriate conclusions being drawn (Vevea & Woods, 2005). Given the potentially serious implications of publication bias – especially if meta-analytic conclusions are used to make practical recommendations for medical or psychological interventions – a number of authors have suggested strategies for eliminating or preventing publication bias in the long-term, as well as statistical methods for detecting and correcting for it in the context of meta-analysis (Vevea & Woods, 2005).

2.8.1. Assessing publication bias

Numerous statistical procedures have been developed to test for the presence of publication bias and to assess the impact of such a bias. Most are based on the assumption that, for a given substantive area, studies with small samples should yield a relatively wide range of effect sizes, whereas studies with large samples should yield estimates near to the population effect size.

2.8.2. Assessing publication bias in the current meta-analysis

Two methods were used to assess for and address publication bias in the present meta-analysis. First, the risk of publication bias was assessed using funnel plots. A funnel plot is a scatterplot of effect sizes graphed against sample size (or an expression of sampling uncertainty such as standard error) (Light & Pillemer, 1984).

A central line indicates the population effect size estimate. An unbiased sample will ideally show a cloud of data points that is symmetric around the population effect size and has the shape of a funnel (reflecting greater variability in effect sizes from small studies). A sample showing publication bias will deviate from the funnel shape because studies based on small samples that showed small effects will be less likely to be published than studies based on the same-size samples but that showed larger effects (Macaskill, Walter & Irwig, 2001; Vevea & Woods, 2005). The literature recommends that funnel plots should only be used as a first step before further analyses because there are factors that can cause asymmetry other than publication bias (Egger, Smith, Schneider & Minder, 1997; Field & Gillett, 2010) and funnel plots leave open the question of how to proceed if publication bias is suspected (Vevea & Woods, 2005).

Next, Vevea and Woods' (2005) sensitivity analysis procedure was performed. This method is argued to be superior to the other available methods for examining publication bias, since it allows the user to apply various 'weights' that represent different types and severities of selection effects in order to explore different theoretical publication bias scenarios (Vevea & Woods, 2005). This method involves 'correcting' the population effect size estimate for publication bias using a priori weights to model the process through which the likelihood of a study being published varies (based on a criterion such as the significance of a study). Exploring different theoretical publication bias scenarios in this way allows the user to explore whether population effect size estimates are robust to the effects of various forms and severities of selection bias; various weights are applied because the presence and extent of publication bias in any given research area is unknown (Vevea & Woods, 2005). The Vevea and Woods (2005) sensitivity analysis method is argued to be particularly useful because it estimates bias in the population effect size itself, rather than being dependent on significance testing: it is more useful to know the effect of publication bias on population effect size estimates, and to correct for it, than to know how many studies would be needed to reverse a conclusion (Vevea & Woods, 2005). Moreover, this method can be applied to relatively small samples of studies, such as is the case here.

2.9. Statistical analysis in the current meta-analysis

Analyses were conducted using the meta-analytic procedures recommended by Borenstein et al. (2009), Lipsey and Wilson (2001) and Field and Gillett (2010). Field and Gillett's (2010) meta-analysis syntax were conducted using SPSS 19 and R2.15.3 (R Development Core Team, 2010).

A number of meta-analyses were carried out to investigate the separate and combined relationships between defeat and entrapment and the four psychological problems. Effect sizes were first transformed using Fisher's Z-transformation. Then, Hedges and Vevea's (1998) random effects method was used to obtain estimates and their standard errors. The estimates were then back-transformed into Pearson's *r* (see Field, 2005b; Lipsey & Wilson, 2001; Overton, 1998). Using this method, each effect size was weighted by a value reflecting the within study variance ($1/n-3$ for correlation coefficients in which n is the sample size) and the between study variance (τ^2). In both the main analyses and moderator analyses, between-study variance was estimated noniteratively (e.g., Dersimonian & Laird, 1986).

Moderator analyses were conducted using a random-effects general linear model in which the effect of a particular moderator variable (i.e. predictor) on each *z*-transformed effect size was estimated (represented by regression coefficient, *b*). The moderator effect, *b*, was estimated using generalised least squares (GLS). Analogue ANOVAs were conducted for categorical moderator variables and meta-regression was conducted for continuous moderator variables. The regression coefficient *b* and its associated 95% confidence interval are reported for continuous moderator variables; *b* is the unstandardized regression parameter for the moderator effect, where a positive *b*-value indicates a positive moderator effect and a negative *b*-value indicates a negative moderator effect. For interpretation purposes, *b* is reported in Fisher's Z*r* units (not Pearson's *r* units). Spearman's rho correlation coefficients are presented for all continuous moderator analyses in order to provide an indication of the size of the moderating relationship. Where statistically significant and borderline significant relationships were found for continuous moderators, scatterplots were generated to explore these relationships further. For a technical overview of the GLS moderator analysis that was employed here see Overton (1998) or Field and Gillett (2010).

2.10. Quality criteria of the meta-analysis

This meta-analysis adhered to the Assessment of Multiple Systematic Reviews (AMSTAR) (Shea, Grimshaw, Wells, Boers, et al., 2007; Shea, Hamel, Wells, Bouter, et al., 2009) quality criteria (see Appendix 1), and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Moher, Liberati, Tetzlaff, Altman, et al., 2009) reporting criteria (see Appendix 2) for meta-analytic reviews. All AMSTAR criteria apart from number seven were met. AMSTAR criterion seven states:

'Was the scientific quality of the included studies assessed and documented? 'A priori' methods of assessment should be provided (e.g., for effectiveness studies if the author(s) chose to include only randomized, double-blind, placebo controlled studies, or allocation concealment as inclusion criteria); for other types of studies alternative items will be relevant.'

All PRISMA criteria apart from number twelve were met. PRISMA criterion twelve states:

'Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis).'

These two criteria describe the practice of conducting a formal quality assessment of studies included in a meta-analysis in order to then examine this variable as a potential moderator. These criteria were not met in the current meta-analysis because the stringent inclusion and exclusion criteria and the nature of the literature meant that there was not enough between-study variation in terms of study quality to make this analysis meaningful. For example, the checklist for determining study quality by Mirza and Jenkins (2004) consists of five criteria: (1) clear study aims, (2) sample representative of population, (3) clear inclusion and exclusion criteria, (4) validated predictor variable measure (in this case defeat and entrapment), and (5) appropriate statistical analysis. Although there was some variation between the

included studies in terms of criteria one and three of Mirza and Jenkins' (2004) checklist, there was not enough variation to form meaningful sub-groups for moderation analyses. It is also worth noting that all but two of the studies included in the meta-analysis (these are forthcoming studies) were published in peer-reviewed journals, which provides some degree of reassurance that their quality would have been assessed and scrutinised by experts in the field before a decision was taken to accept them for publication.

CHAPTER THREE: RESULTS

3.1. Overview

This chapter begins by describing the characteristics of studies included in the meta-analysis and presenting some descriptive statistics regarding the distribution of included effect sizes. Next, random-effects meta-analyses of relationships between defeat and entrapment combined and the four psychological problems are presented. These are followed by random-effects meta-analyses of the separate relationships between defeat, internal entrapment, external entrapment and total internal and external entrapment, and the four psychological problems. Statistical tests of between-study heterogeneity and of the statistical significance of the obtained population effect size estimates are presented. Next, moderator analyses are reported where there are sufficient numbers of effect sizes in sub-groups. The potential moderating role of eight different sample and study characteristics are examined. The chapter finishes with two different methods of publication bias analysis.

3.2. Study characteristics

Thirty-eight studies contributed ninety-four effect sizes for inclusion in the meta-analysis; with a total of 11,343 participants. The sample sizes used in statistical analyses in each individual included study ranged from 9 (Clare & Singh, 1994) to 311 (Yoon, 2003) (M = 120.67, SD = 72.02). Five of the studies used a prospective or longitudinal design (12.05% of total effect sizes). Of the twelve studies that provided information on ethnicity (M = 17.20%), one (Birchwood et al., 2007) included a majority sample of non-white participants (54.4% non-white). Two studies (Jobson & O'Kearny, 2009; Karatzias et al., 2007) used categorical (diagnostic) measures of psychological problems. Characteristics of the studies included in the meta-analysis are presented in Table 2.

Table 2. Characteristics of studies included in the meta-analysis.

Article	**Sample details**	**N**	**Measure of defeat and/or entrapment**	**Defeat and/or entrapment data**	**Measure(s) of psychological problems**	**Mean age (SD)**	**Percentage of sample female**	**Study location**
Allan & Gilbert (2002)	University undergraduates	197	Defeat and Entrapment Scales	External entrapment	CES-D	23.40 (8.0)	62.9	UK
Birchwood et al. (1993)	Medicated; mixed psychosis sample	84	PBIQ	Internal entrapment	BDI	48.05 (13.2)	35.7	UK
Birchwood et al. (2007)	First-episode schizophrenia spectrum disorder	79	PBIQ	Internal entrapment	Social Interaction Anxiety Scale	Not reported	22.8	UK
Clare & Singh (1994)	Medicated; Mixed psychosis and other affective disorders	11	PBIQ	Internal entrapment	BDI	35.00 (Not reported)	27.3	UK
Dunmore et al. (1997)	Mixed physical and sexual assault victims	20	MDTS	Defeat	PTSD Symptom Scale Self-Report	38.10 (11.4)	75.0	UK
Dunmore et al. (1999)	Mixed physical and sexual assault victims	92	MDTS	Defeat	PTSD Symptom Scale Self-Report	38.60 (16.2)	47.8	UK
Dunmore et al. (2001)	Assault survivors	57	MDTS	Defeat	PTSD Symptom Scale Self-Report	Not reported	54.4	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
Garcia-Campayo et al. (2010)	Chronic pain (Fibromyalgia) outpatients	250	PSPS	Defeat	HADS	44.90 (7.2)	91.6	Spain
Gilbert & Allan (1998) Sample 1	University undergraduates	302	Defeat and Entrapment Scales	Defeat and entrapment	BDI	22.90 (8.0)	77.2	UK
Gilbert & Allan (1998) Sample 2	Depressed patients	90	Defeat and Entrapment Scales	Defeat and entrapment	BDI	22.90 (8.0)	77.2	UK
Gilbert et al. (2002) Sample 1	University undergraduates	193	Defeat and Entrapment Scales	Defeat and entrapment	MASQ	22.90 (7.7)	76.7	UK
Gilbert et al. (2002) Sample 2	Mixed psychiatric inpatients	81	Defeat and Entrapment Scales	Defeat and entrapment	MASQ	36.80 (13.0)	60.5	UK
Gilbert et al. (2004)	Depressed inpatients and outpatients	50	Custom interview concerning entrapment	External entrapment	BDI-II	43.45 (Not reported)	46.0	UK
Gilbert et al. (2005)	University undergraduates	166	Defeat and Entrapment Scales	Internal and external entrapment	CES-D	22.07 (7.2)	83.1	UK
Goldstein & Willner (2002)	University undergraduates	32	Defeat and Entrapment Scales	Defeat and entrapment	BDI	Not reported	100.0	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
Griffiths et al. (2013)	Community sample from low SES backgrounds	195	Defeat and Entrapment Scales	Defeat and entrapment	CES-D, STAI: State subscale	36.90 (8.3)	Not reported	UK
Gumley et al. (2004)	Schizophrenia spectrum disorder	38	PBIQ	Internal entrapment	Brief Symptoms Interview: Social Anxiety	34.35 (8.4)	26.3	UK
Iqbal et al. (2000)	Schizophrenia spectrum disorder	105	Mental defeat rated from narrative	Internal entrapment	BDI	Not reported	Not reported	UK
Jobson & O'Kearny (2009)	Community sample: traumatic experiences	106	Mental defeat rated from narrative	Defeat	Post-Traumatic Stress Diagnostic Scale	37.21 (13.4)	69.1	Australia
Karatzias et al. (2007)	Schizophrenia spectrum disorder	138	PBIQ	Internal entrapment	SCID: Comorbid Anxiety or Affective Disorder	36.60 (9.8)	28.3	UK
Martin et al. (2006)	Caregivers of Alzheimer disease patients	70	CES	External entrapment	CES-D	Not reported	Not reported	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
Panagioti et al. (2012)	Community sample: experienced a traumatic event	56	Defeat and Entrapment Scales	Defeat and entrapment	Suicidal Behaviors Questionnaire–Revised, Post-Traumatic Diagnostic Scale	29.10 (11.5)	82.1	UK
Rasmussen et al. (2010)	Individuals who had attempted suicide	103	Defeat and Entrapment Scales	Defeat and entrapment	Suicide Probability Scale, HADS	34.92 (13.4)	59.0	UK
Stommel et al. (1990)	Caregivers of elderly relatives	307	CBS-E	External entrapment	CES-D	Not reported	Not reported	America
Sturman (2011)	University undergraduates	119	ISQ	Defeat and entrapment	CES-D, Social Anxiety Interaction Scale and Social Phobia Scale	19.00 (Not reported)	79.8	Canada
Sturman & Mongrain (2008a)	Formerly depressed students	146	Defeat and Entrapment Scales	Internal and external entrapment	SCID: Depression	Not reported	71.9	Canada
Tang et al. (2007)	Chronic pain patients	302	PSPS	Defeat	HADS	46.10 (12.3)	72.7	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
Tang et al. (2010)	Chronic pain patients	133	PSPS	Defeat	HADS	46.10 (Not reported)	Not reported	UK
Taylor et al. (2010a)	Schizophrenia spectrum disorder	78	Defeat and Entrapment Scales	Defeat and entrapment	Beck Scale of Suicidal Ideation	42.50 (11.8)	25.6	UK
Taylor et al. (2010b)	University undergraduates with past or current suicidal ideation	93	Defeat and Entrapment Scales	Defeat and entrapment	Suicidal Behaviors Questionnaire–Revised	23.45 (7.1)	81.7	UK
Taylor et al. (2011)	University undergraduates		Defeat and Entrapment Scales	Defeat and entrapment	BDI-II, Suicidal Behaviors Questionnaire–Revised	19.61 (4.5)	83.5	UK
Trachsel et al. (2010)	Community sample (general population)	540	Defeat and Entrapment Scales	Internal and external entrapment	CES-D	Not reported	63.2	Germany
Troop (2013)	Eating disorder inpatient and outpatients	114	Defeat and Entrapment Scales	Defeat and entrapment	Post-Traumatic Diagnostic Scale	33.70 (10.3)	96.5	UK
Troop & Baker (2008)	Female office workers	74	Defeat and Entrapment Scales	Defeat and entrapment	BDI-II	24.60 (7.6)	100.0	UK

Article	Sample details	N	Measure of defeat and/or entrapment	Defeat and/or entrapment data	Measure(s) of psychological problems	Mean age (SD)	Percentage of sample female	Study location
Troop & Hiskey (in press)	Community sample recruited from stress and trauma-related websites	275	Defeat and Entrapment Scales	Defeat and entrapment	Post-Traumatic Diagnostic Scale	31.60 (11.4)	75.0	UK
Troop et al. (2013)	Eating disorder history	189	Defeat and Entrapment Scales	Defeat and entrapment	BDI-II	35.50 (9.9)	96.0	UK
White et al. (2007)	Schizophrenia spectrum disorder	100	PBIQ	Internal and external entrapment	Calgary Depression Scale for Schizophrenia	39.40 (11.2)	22.0	UK
Willner & Goldstein (2001)	Mothers of children with special educational needs	76	Defeat and Entrapment Scales	Defeat and entrapment	BDI	40.20 (7.2)	Not reported	UK
Wyatt & Gilbert (1998)	University undergraduates	113	Defeat and Entrapment Scales	Defeat	CES-D	24.88 (8.3)	77.9	UK
Yoon (2003)	Caregivers of family member with functional and/or cognitive impairment	311	CBS-E	External entrapment	Self-Rating Depression Scale	56.10 (15.6)	81.0	Korea

Note. BDI = Beck Depression Inventory, BDI-II = Beck Depression Inventory – II, CBS-E = Caregiver Burden Scale – Entrapment subscale, CES = Caregiver's Entrapment Scale, CES-D = Center for Epidemiological Studies Depression Scale, HADS = Hospital

Anxiety and Depression Scale, MASQ = Mood and Anxiety Symptoms Questionnaire, MDTS = Mental Defeat during Trauma Scale, PBIQ = Personal Beliefs about Illness Questionnaire, PSPS = Pain Self Perception Scale, SCID = Structured Clinical Interview for DSM-IV Disorders, STAI-State = State Trait Anxiety Scale – State subscale.

3.3. Exploratory data analyses

The distribution of effect sizes included in the meta-analysis was examined using multiple standard methods, as described by Field (2005a). The resulting descriptive statistics are displayed in Table 3. Means are unweighted at this stage of this analysis. Table 3 shows a modest degree of skewness and kurtosis for two of the effect size groups. This occurred when all variables were combined (i.e. all four psychological problems combined) and for the depression effect-sizes. The extent of skewness and kurtosis, although modest, is statistically significant for these two groups when compared against values that would be expected by chance alone (i.e., when compared against known values for the normal distribution after first converting the skewness and kurtosis statistics to *z*-scores) (Field, 2005a). Kolmogorov-Smirnov tests likewise confirmed that the distributions for these two effect size groups were significantly different from a normal distribution.

Table 3. Descriptive statistics for defeat and entrapment combined and the four psychological problems.

	Psychological variables				
Descriptive statistics	All variables combined	Depression	Anxiety	PTD	Suicidality
N	94	53	17	12	12
Skewness	.81**	-1.43*	.81	-.91	-.07
Kurtosis	1.02*	2.67***	.17	.03	-.96
Mean (*SD*)	.62 (.13)	.66 (.14)	.56 (.10)	.56 (.11)	.61 (.09)
Median	.63	.67	.59	.60	.60
Min-Max	.23 - .88	.23 - .88	.33 - .70	.33 - .70	.45 - .75

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

One study (Ehlers et al., 2000) was excluded because it introduced an extreme outlier (r = .18, >3 interquartile ranges) to the pool of effect sizes for the relationship between defeat and depression (Table 3 was created with this study removed). The decision was taken to exclude this study on methodological grounds because it differed substantially from the other included trauma samples in terms of the time period between trauma and assessment: in this study, measures were taken an average

of 21.3 years after the trauma, whereas in all other included trauma samples, measures were taken between one month and two years post-trauma.

Next, boxplots were constructed in order to explore the identified skewness further. Figure 3 displays boxplots of the effect sizes between defeat and entrapment combined and each of the four psychological problems. There are various outliers on both sides of the depression distribution, although none of these can be considered extreme outliers (>3 interquartile ranges). The fact that these outliers appear on both sides of the distribution suggests that the average depression effect size has probably not been substantially biased by outliers. This hypothesis is confirmed by the similarity in values for the mean and median for each problem group (Table 3).

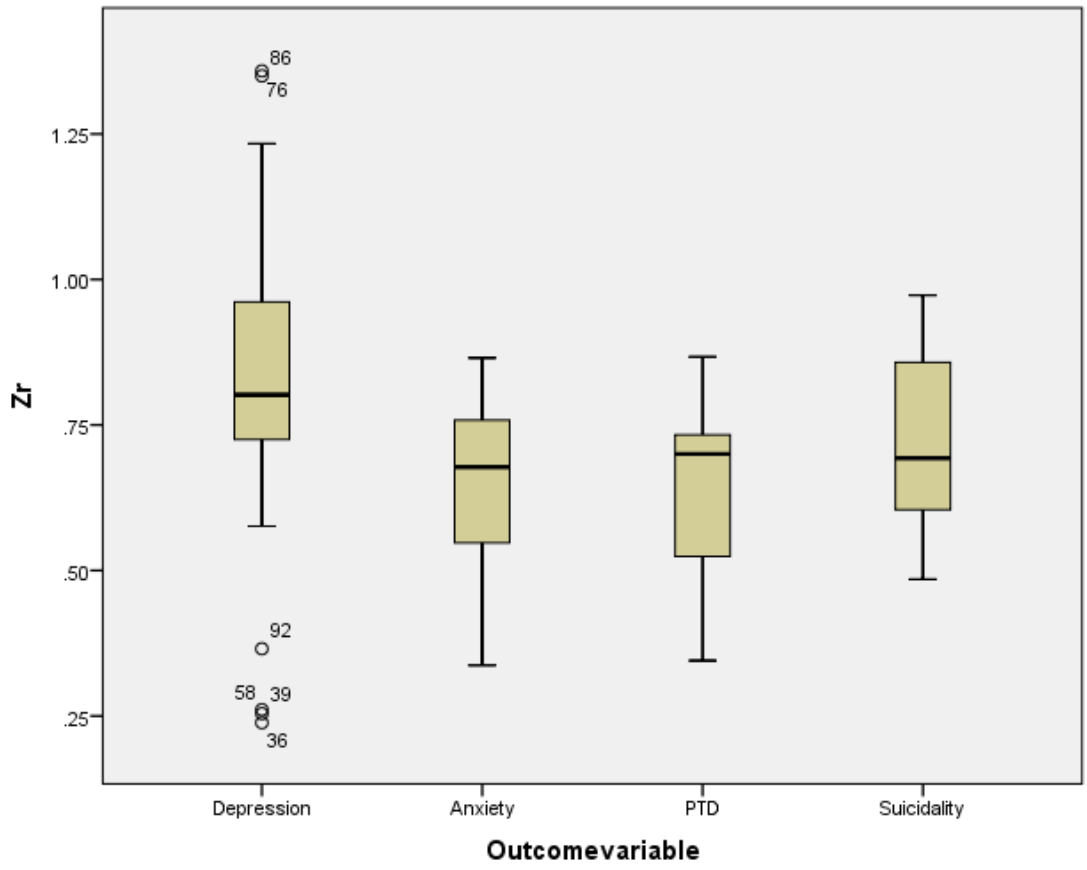

Figure 3. Boxplots of effect sizes between defeat and entrapment combined as a single variable and type of psychological problem.

Following Rosenthal's (1995) advice regarding visually displaying effect sizes, Table 4 shows stem and leaf plots of the included effect sizes listed by the type of psychological problem. The mode of all four distributions is .6, after which a fairly even number of effect sizes cluster around the intervals between .5 and .8. There is a clear negative skew in all four stem and leaf plots.

Table 4. Stem and leaf plots of included effect sizes listed by type of psychological problem.

Depression		Suicidality	
Stem	Leaf	Stem	Leaf
.2	3, 5, 6	.2	
.3	5	.3	
.4		.4	5, 9
.5	2, 4, 4, 7, 8, 8	.5	7, 6, 2
.6	0, 0, 2, 2, 2, 3, 3, 4, 4, 4, 5, 5, 6, 7, 7, 8, 9, 9	.6	0, 0, 3, 9
.7	0, 1, 2, 2, 3, 3, 3, 3, 4, 4, 5, 6, 6, 7, 7, 7, 8	.7	0, 1, 5
.8	0, 1, 2, 3, 4, 7, 8	.8	

Anxiety problems		PTSD	
Stem	Leaf	Stem	Leaf
.2		.2	
.3	3	.3	3
.4	1, 5, 9	.4	2, 4
.5	0, 2, 3, 6, 9	.5	2, 5
.6	0, 2, 3, 4, 5, 6	.6	0, 1, 2, 2, 3, 6
.7	0, 2	.7	0
.8		.8	

Next, a normal probability plot (NPP) was created for all effect sizes included in the meta-analysis (Figure 4). The NPP is a graphical technique for assessing whether a dataset is approximately normally distributed. Observed data (the black dots) are plotted against data that would be expected if the distribution were perfectly normal (the solid diagonal line). Figure 4 shows that the ninety-four effect sizes included in the meta-analysis deviate minimally from what would be expected if the distribution were perfectly normal. Confidence bands around normality are also displayed (the parallel dotted lines). None of the effect sizes breaches this confidence

band. One outlier is apparent in the depression group (the leftmost point in the chart). However, the fact that this outlier does not fall outside of the confidence bands indicates that this cannot be considered an extreme outlier.

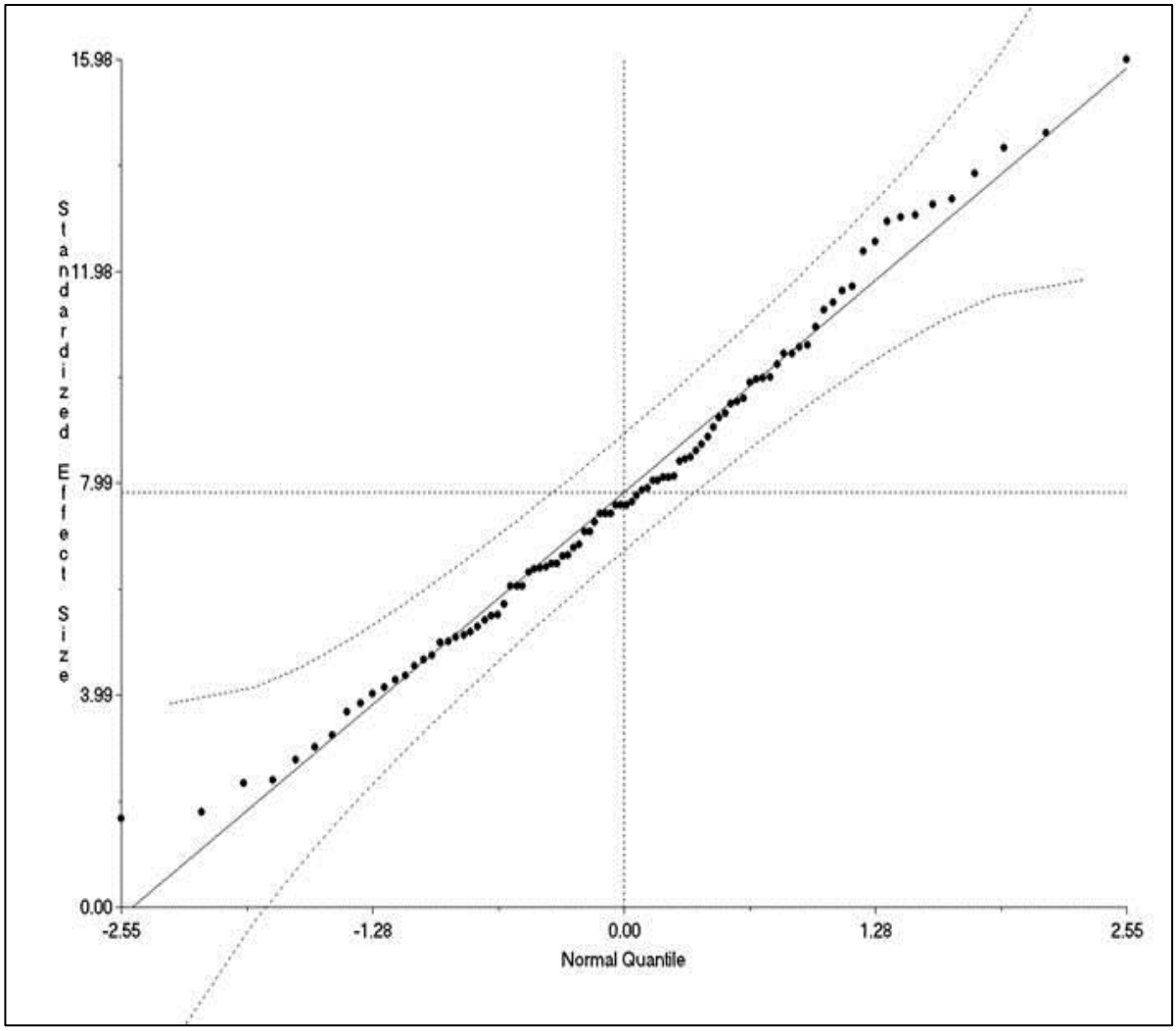

Figure 4. Normal probability plot of all effect size correlations.

3.3.1. Summary of exploratory data analyses

Using multiple methods, exploratory data analyses established that the ninety-four effect sizes included in the meta-analysis predominantly adhere to assumptions of normality and form a reasonably symmetrical distribution. There were some minor outliers for the depression effect sizes, which were apparent on both sides of the depression distribution. However, these were not extreme outliers (>3 interquartile

ranges) and they do not appear to have substantially skewed the distribution of depression effect sizes.

3.4. Population effect size estimates

3.4.1. Combining psychological problem groups

As has been outlined in chapter one, there is evidence to suggest that the strength of relationships between defeat and entrapment and psychological problems might differ considerably depending on the type of psychological problem. This hypothesis was examined by running an Analogue ANOVA (Lipsey & Wilson, 2001) to test the overall Null-Hypothesis that the average effect size correlations regarding defeat and entrapment would be equal between the four types of psychological problems (depression, suicidality, anxiety problems and PTSD). This moderator analysis revealed that differences in effect sizes across psychological problem categories was a significant moderator ($Q(3) = 13.91$, $p = .003$). In light of this moderator effect, analyses are reported separately for the four psychological problem groups.

3.4.2. Defeat and entrapment combined

Four random-effects meta-analyses were conducted to explore the relationships between defeat and entrapment combined as a single variable and depression, suicidality, anxiety problems and PTSD. Table 5 shows that large (Cohen, 1988), statistically significant effect sizes were observed for depression (explaining 45% of the variance in depression scores), anxiety problems (explaining 32% of the variance in anxiety problem scores), PTSD (explaining 34% of the variance in PTSD scores) and suicidality (explaining 37% of the variance in suicidality scores). Table 5 also shows that depression effect sizes significantly differ (because of non-overlapping confidence intervals) from anxiety problem and PTSD effect sizes.

There was no significant between-study heterogeneity in the distribution of effect sizes within each meta-analysis. Linking the heterogeneity results to the exploratory data analyses presented earlier, this suggests that the between-study heterogeneity – which was apparent for the depression boxplot at both ends of the distribution (Figure 3) – was relatively trivial and was therefore accounted for by the

use of a random-effects meta-analytic model. The absence of significant between-study heterogeneity is consistent with the results of the NPP plot (Figure 4).

Table 5. Meta-analyses of relationships between defeat and entrapment combined as a single variable and the four psychological problems.

Analysis	*k*	*Q*	I^2 (95% Confidence Interval)[a]	95% Confidence Interval for *r*			*z*	r_{pb}
				Lower	Mean	Upper		
Grand mean[b]	94	98.97	.06 (.00, .28)	.62	.64	.67	35.16***	.64
Depression[b]	53	54.32	.04 (.00, .30)	.64	.68	.71	25.81***	.68
Anxiety problems[b]	17	16.12	.01 (.00, .51)	.53	.58	.62	18.98***	.57
PTSD	12	11.99	.08 (.00, .62)	.54	.59	.63	18.08***	.59
Suicidality	12	11.14	.01 (.00, .59)	.56	.61	.66	16.61***	.61

Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$, *k* = number of studies, r_{pb} = estimate of the population effect size under severe two-tailed publication bias (Vevea & Woods, 2005), PTSD = Post-Traumatic Stress Disorder.

[a] 95% confidence intervals are calculated as proposed by Higgins and Thompson (2002).

[b] These meta-analyses included a study by Sturman (2011) which combined measures of defeat and entrapment into one variable. Removal of this study would reduce the population effect size estimate by .002 for depression and .008 for anxiety problems.

3.4.3. Defeat and entrapment analysed separately

Next, thirteen random-effects meta-analyses explored the separate relationships between defeat, internal entrapment, external entrapment and total internal and external entrapment, and the four psychological problems (Table 6). Again, large (Cohen, 1988), statistically significant effect sizes were observed in all meta-analyses and there was no significant between-study heterogeneity in the distribution of effect sizes within each meta-analysis. Table 6 shows that defeat and entrapment correlated most strongly with depression (with one exception). There was a particularly large relationship between defeat and depression, with little variation (relatively narrow confidence intervals), which explained 55% of the variance in depression scores. Correlations between defeat and entrapment and suicidality,

anxiety problems and PTSD groups, were generally of a similar-size (with the exception of internal entrapment).

Table 6. Meta-analyses of the separate relationship between defeat, internal entrapment, external entrapment and total internal and external entrapment, and the four psychological problems.

Analysis	*k*	*Q*	I^2 (95% Confidence Interval)[a]	95% Confidence Interval for *r*			*z*	r_{pb}
				Lower	Mean	Upper		
Defeat								
Depression	16	14.96	.00 (.00, .52)	.69	.74	.78	18.32***	.74
Anxiety problems	7	5.97	.00 (.00, .71)	.54	.58	.63	20.36***	.58
PTSD	7	6.30	.05 (.00, .72)	.48	.58	.66	9.64***	.58
Suicidality	5	4.03	.07 (.00, .79)	.50	.57	.64	12.50***	.57
Internal Entrapment								
Depression	13	14.09	.15 (.00, .54)	.56	.65	.73	10.44***	.64
Anxiety problems	4	2.43	.00 (.00, .81)	.30	.48	.62	4.94***	.47
PTSD	2	.044		.54	.61	.67	13.77***	.61
External Entrapment								
Depression	16	12.72	.00 (.00, .44)	.57	.64	.70	14.58***	.64
Anxiety problems	3	1.64	.00 (.00, .87)	.45	.53	.60	11.28***	.53
PTSD	2	.190		.47	.54	.61	11.88***	.54
Total Internal and External Entrapment								
Depression	7	6.38	.22 (.00, .66)	.51	.63	.72	8.66***	.63
Anxiety problems	2	.521		.61	.68	.74	13.94***	.68
Suicidality	5	4.02	.05 (.00, .79)	.51	.62	.72	8.37***	.62

Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$, *k* = number of studies, r_{pb} = estimate of the population effect size under severe two-tailed publication bias (Vevea & Woods, 2005), PTSD = Post-Traumatic Stress Disorder.

[a] 95% confidence intervals are calculated as proposed by Higgins and Thompson (2002); missing confidence intervals are due to $N<3$.

3.5. Moderator analyses

3.5.1. Sensitivity analyses regarding the strength of continuous moderator variables

Field & Gillett's (2010) SPSS syntax for conducting moderator analysis reports unstandardized beta units (*b*) for continuous moderator variables. In order to explore the strength of continuous moderator variables, it is therefore necessary to compute Pearson's *r* or Spearman's rho correlation coefficients (or standardised betas). Pearson's *r* can be dramatically affected by outliers in small samples, such as is the case here. Therefore a sensitivity analysis was undertaken to explore which of the two types of correlation coefficient would be most appropriate for the data included in the meta-analysis. This consisted of two steps. First, scatterplots were generated to explore the shape and distribution of all continuous moderator variables. Scatterplots are presented below for statistically significant and borderline-significant continuous moderator variables. Second, both Spearman's rho and Pearson's *r* correlation coefficients were computed for all continuous moderator variables. The size and statistical significance of each respective correlation coefficient was then compared. Appendix 4 shows that the two types of correlation coefficient do not differ much for depression (because of its larger sample size) but differ markedly for the other three psychological problems. On the basis of exploratory scatterplots and the discrepancy between correlation coefficients presented in Appendix 4, Spearman's rho correlation coefficients are reported for all continuous moderator analyses.

3.5.2. Reminder about statistical analysis for moderator analyses

For continuous moderator variables, the regression coefficient *b* and its associated 95% confidence interval are reported; *b* is the unstandardized regression parameter for the moderator effect, where a positive *b*-value indicates a positive moderator effect and a negative *b*-value indicates a negative moderator effect. For interpretation purposes, *b* is reported in Fisher's Z*r* units (not Pearson's *r* units).

3.5.3. Depression moderator analyses

3.5.3.1. Defeat and entrapment categories

Differences across defeat and entrapment categories was a significant moderator of depression effect sizes, $Q(3) = 8.35$, $p = .039$. Table 7 shows that effect sizes for the relationship between defeat and depression were significantly larger than

those found between each of the entrapment variables and depression. Table 6 shows that the population effect size estimates for defeat and depression (r = .74) were larger than those obtained for internal entrapment (r = .65), external entrapment (r = .64) and total internal and external entrapment (r = .63). It also reveals that population effect size estimates obtained for all four defeat and entrapment categories were significantly different from zero.

Table 7. Moderators of the impact of defeat and entrapment sub-groups on depression.

				95% Confidence Interval for *b*			
Moderator	Groups	*k*	*b*	Lower	Upper	*t*	*p*
Defeat and entrapment variable	Defeat vs. internal entrapment	29	.86	.78	.95	20.17	.000
	Defeat vs. external entrapment	32	.85	.77	.92	23.25	.000
	Defeat vs. total internal and external entrapment	23	.88	.79	.97	4.89	.027
Measure of depression	BDI/BDI-II vs. CES-D	37	.88	.81	.94	27.29	.000
	BDI/BDI-II vs. HADS	35	.87	.80	.95	24.77	.000
	BDI/BDI-II vs. Other	36	.85	.76	.94	19.65	.000

Note. *k* = number of studies, *b* = regression parameter for the moderator effect, *t* = test of the moderation effect, BDI = Beck Depression Inventory, BDI-II = Beck Depression Inventory – II, CES-D = Center for Epidemiological studies Depression Scale, HADS = Hospital Anxiety and Depression Scale, Other consisted of the Mood and Anxiety Symptoms Questionnaire, Structured Clinical Interview for DSM-IV Disorders, Calgary Depression Scale for Schizophrenia and the Self-Rating Depression Scale.

3.5.3.2. Sample gender composition

Sample gender composition emerged as a significant moderator of depression effect sizes (b = .007 (95CI, .004, .010), p = .000, r_s = .55), indicating that samples made up of a higher proportions of females showed a stronger relationship between defeat and entrapment combined and depression. A scatterplot (Figure 5) was

generated to explore this positive, statistically-significant (p = .000) Spearman's rho correlation further. Although there is some scatter in the data points, a generally positive linear relationship is apparent. The coefficient of determination (R^2 = .22) indicated that 22% of the variance in depression effect sizes was accounted for by the sample gender composition. In interpreting R^2, it should be noted that although the sample gender composition can account for 22% of the variance in the depression effect sizes (or vice-versa), this does not necessarily mean that one variable caused variation in the other variable (Field, 2005a).

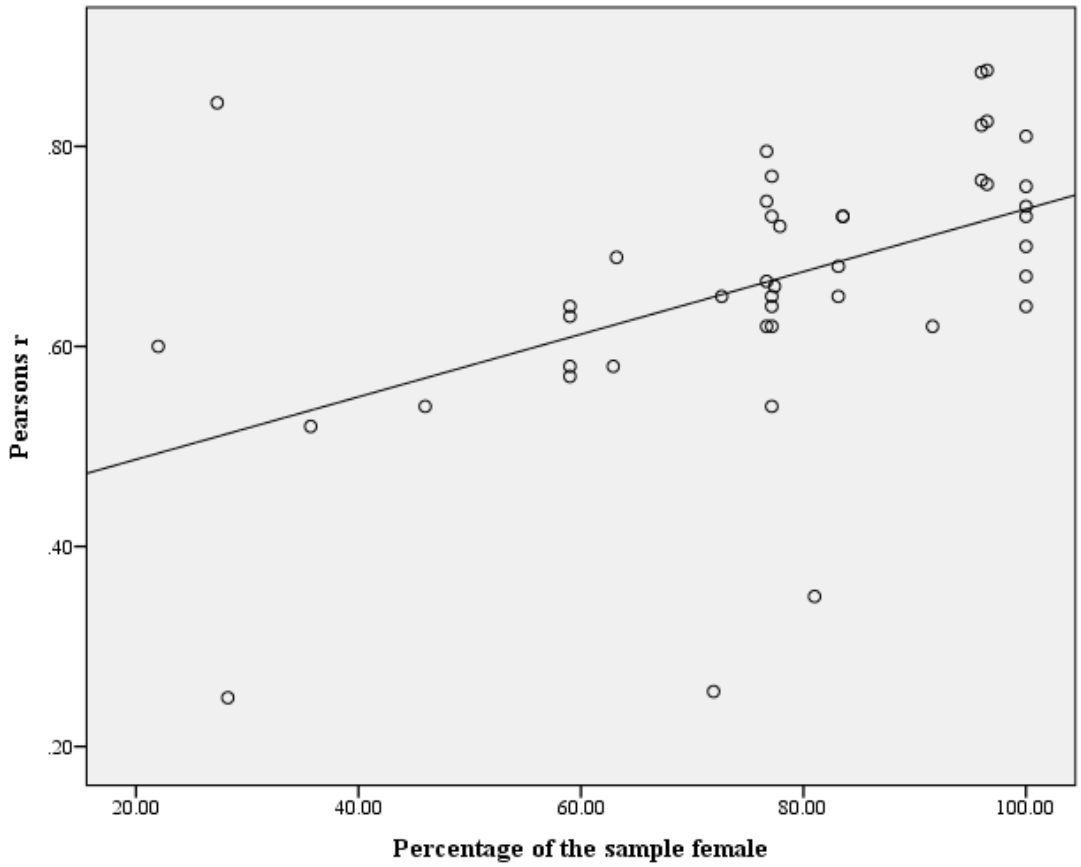

Figure 5. Scatterplot showing the relationship between gender composition of samples and depression effect sizes when defeat and entrapment are combined.

3.5.3.3. Depression measure

Measure of depression also significantly moderated the relationship between defeat and entrapment combined and depression effect sizes, $Q(3) = 13.53$, $p = 004$. Table 7 shows that effect sizes obtained using the Beck Depression Inventory differed

significantly to those obtained via the Center for Epidemiological Studies Depression Scale (CES-D), Hospital Anxiety and Depression Scale (HADS) and 'other' depression measures. Table 8 shows that the population effect size estimates obtained by all depression measures were significantly different from zero and that the population effect size estimates obtained for depression using the Beck Depression Inventory (r = .72) were larger than those obtained via the CES-D (r = .65), HADS (r = .62) and 'other' depression measures (r = .56). The effect sizes obtained using the BDI were significantly larger (because of non-overlapping confidence intervals) than those obtained using the HADS.

Table 8. Moderators of the impact of measure of depression on the relationship between defeat and entrapment combined and depression.

		95% Confidence Interval for *r*			
Groups	*k*	Lower	Mean	Upper	z
BDI/BDI-II	29	.68	.72	.76	20.44***
CES-D	9	.62	.65	.68	31.26***
HADS	7	.58	.62	.66	21.95***
Other	8	.40	.56	.69	5.86***

Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$, k = number of studies, BDI = Beck Depression Inventory, BDI-II = Beck Depression Inventory – II, CES-D = Center for Epidemiological Studies Depression Scale, HADS = Hospital Anxiety and Depression Scale, Other consisted of Mood and Anxiety Symptoms Questionnaire, Structured clinical interview for DSM-IV disorders, Calgary Depression Scale for Schizophrenia and the Self-Rating Depression Scale.

3.5.3.4. Defeat and entrapment measure

Lastly, measure of defeat and entrapment significantly moderated the relationship between defeat and entrapment combined and depression effect sizes, $Q(1) = 13.54$, $p = .000$. Table 9 shows that effect sizes obtained using all defeat and entrapment measures were significantly different from zero and that the effect size estimates obtained for depression using the Defeat and Entrapment Scales (r = .70) were significantly larger (because of non-overlapping confidence intervals) than those obtained using other defeat and entrapment measures (r = .55).

Table 9. Moderators of the impact of measure of defeat and entrapment on the relationship between defeat and entrapment combined and depression.

		95% Confidence Interval for *r*			
Groups	*k*	Lower	Mean	Upper	z
Defeat and Entrapment Scale	40	.67	.70	.74	26.54***
Other scales	12	.46	.55	.63	10.30***

Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$, *k* = number of studies, Other consisted of Personal Beliefs about Illness Questionnaire, Mental Defeat During Trauma Scale, Pain Self Perception Scale, Custom Interview Concerning Entrapment, Mental Defeat Rated from Narrative, Carer's Entrapment Scale and the Carer Burden Scale – Entrapment subscale.

3.5.3.5. Other moderators of depression

Year of publication ($b = .010$, $p = .07$, $r_s = .18$), mean age ($b = -.005$, $p = .124$, $r_s = -.17$) and clinical status of sample ($Q(1) = 2.71$, $p = .100$) did not significantly moderate the relationship between defeat and entrapment combined and depression effect sizes.

3.5.4. Anxiety moderator analyses

3.5.4.1. Year of publication

Year of publication emerged as a significant moderator of anxiety problem effect sizes ($b = .024$ (95CI, .009, .038), $p = .004$, $r_s = .78$), indicating that more recently published studies showed a stronger relationship between defeat and entrapment combined and anxiety problems. A scatterplot (Figure 6) was generated to explore this positive, statistically-significant ($p = .000$) Spearman's rho correlation further. Although two data points (at the bottom of the scatterplot) are somewhat different from the others, a generally positive linear relationship is apparent. The coefficient of determination ($R^2 = .42$), indicated that 42% of the variance in anxiety problem effect sizes was accounted for by the year of publication. In interpreting R^2, it is again noted that although the year of publication can account for 42% of the variance in the anxiety problem effect sizes (or vice-versa), this does not necessarily mean that one variable caused variation in the other variable (Field, 2005a). Given that there are a relatively small number of effect sizes, the nature and size of the

relationship between year of publication and anxiety problem effect sizes should be interpreted with caution.

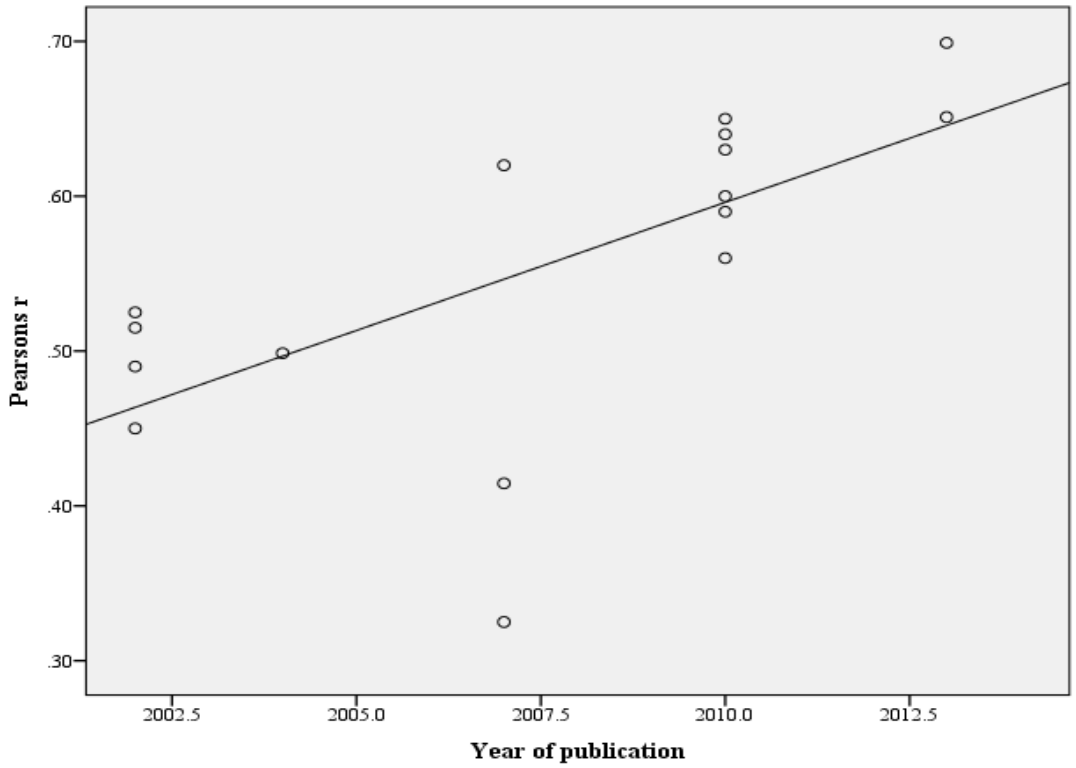

Figure 6. Scatterplot showing the relationship between year of publication and anxiety problem effect sizes when defeat and entrapment are combined.

3.5.4.2. Mean age

Mean age did not significantly moderate the relationship between defeat and entrapment combined and anxiety problem effect sizes (b = .007, p = .157, r_s = .53). However, the Spearman's correlation for mean age was statistically significant (p = .034). This could suggest that a larger sample size may reveal that mean age is a significant moderator of anxiety problem effect sizes. A scatterplot (Figure 7) was generated to explore this correlation further. It is apparent in the scatterplot that there is a great deal of scatter of data points. For this reason, the moderate-size Spearman's rho correlation should be interpreted very tentatively as it is entirely possible that a larger sample size could reveal no relationship. The coefficient of determination (R^2 = .14), indicated that 14% of the variance in anxiety problem effect sizes was accounted for by the mean age of samples.

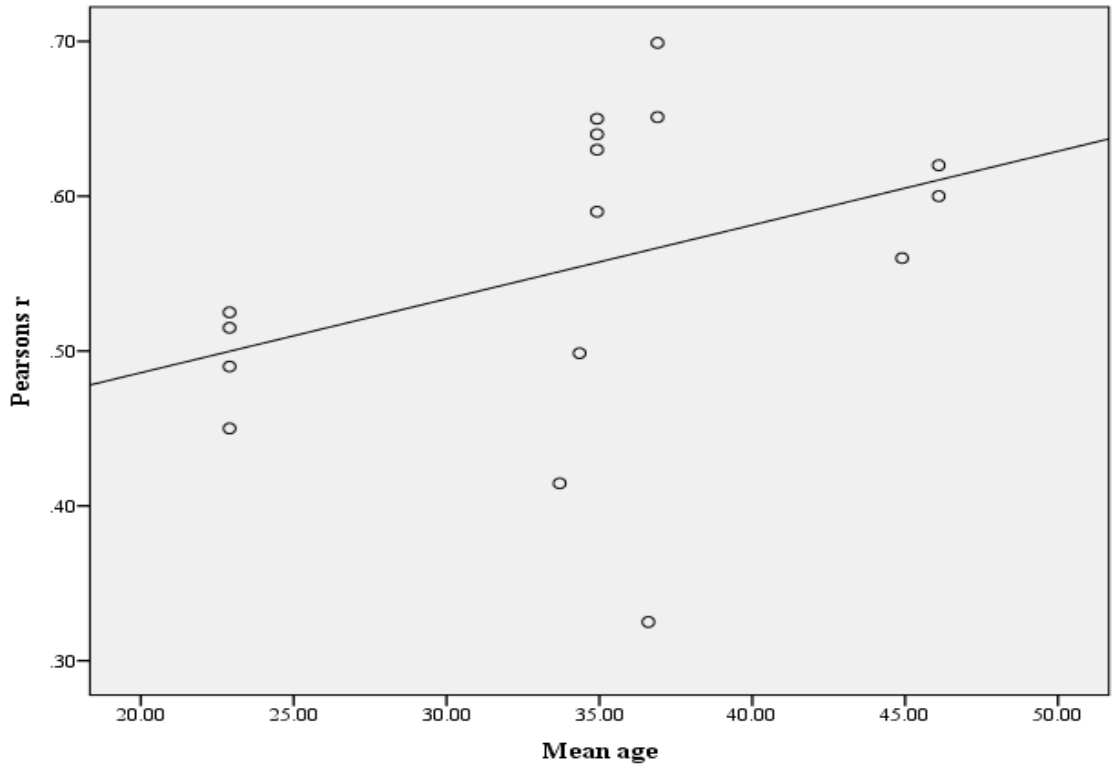

Figure 7. Scatterplot showing the relationship between mean age of sample and anxiety problem effect sizes when defeat and entrapment are combined.

3.5.4.3. Other moderators of anxiety problems

Differences in effect size across defeat and entrapment categories ($Q(1) = .49$, $p = .486$), sample gender composition ($b = .003$, $p = .07$, $r_s = .35$) and measure of defeat and entrapment ($Q(1) = 2.28$, $p = .131$) did not significantly moderate the relationship between defeat and entrapment combined and anxiety problem effect sizes.

3.5.5. PTSD moderator analyses

Differences in effect size across defeat and entrapment categories ($Q(1) = .04$, $p = .843$), year of publication ($b = .008$, $p = .295$, $r_s = .43$), sample gender composition ($b = .002$, $p = .470$, $r_s = .15$), mean age ($b = -.029$, $p = .08$, $r_s = -.65$) and clinical status of sample ($Q(1) = .94$, $p = .334$), did not significantly moderate the relationship between defeat and entrapment combined and PTSD effect sizes. The Spearman's correlation between defeat and entrapment combined and mean age was moderate to large (Cohen, 1988) and statistically significant ($p = .022$). This could suggest that a

larger sample size may reveal that mean age is a significant moderator of PTSD effect sizes. A scatterplot (Figure 8) was generated to explore this correlation further. It is apparent in the scatterplot that there is a fair degree of scatter in data points. The two data points on the lower right of the scatterplot may explain the nature of the correlation. For these reasons, the moderate to large Spearman's rho correlation should be interpreted very tentatively as it is entirely possible that a larger sample size could reveal no relationship. The coefficient of determination ($R^2 = .35$) indicated that 35% of the variance in PTSD effect sizes was accounted for by the mean age of samples.

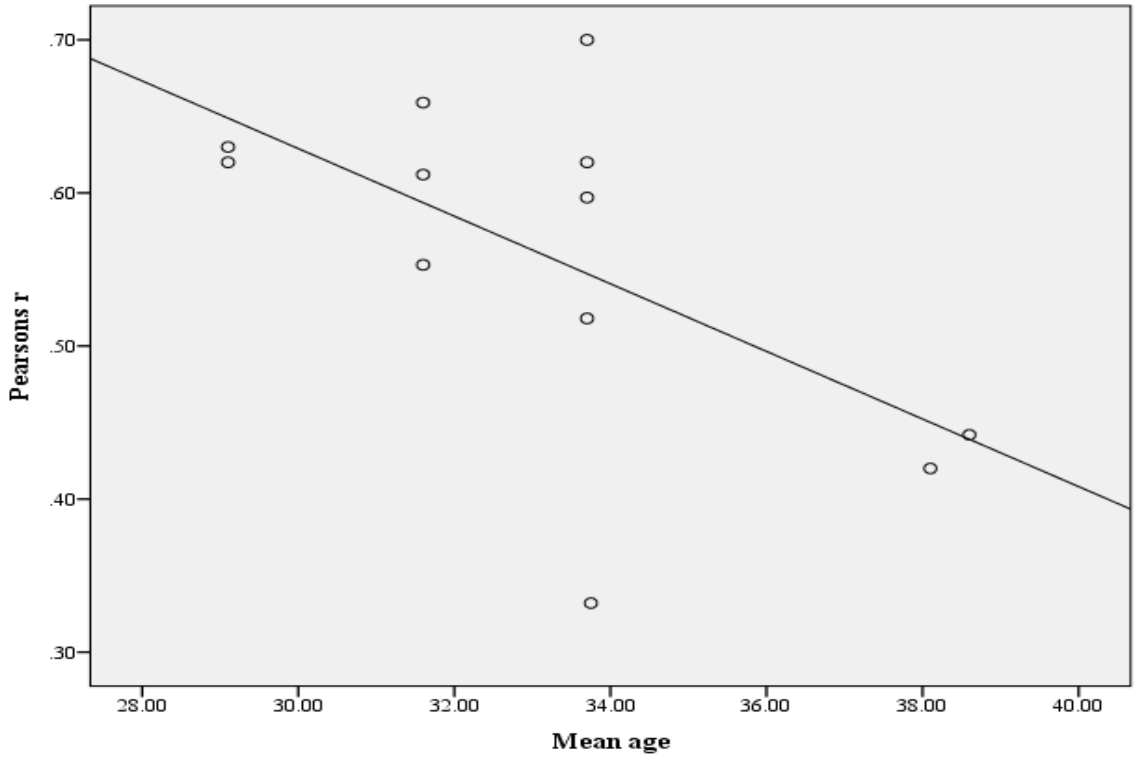

Figure 8. Scatterplot showing the relationship between mean age of samples and PTSD effect sizes when defeat and entrapment are combined.

3.5.6. Suicidality moderator analyses

Differences in effect size across defeat and entrapment categories ($Q(1) = 1.24$, $p = .266$), year of publication ($b = .075$, $p = .08$, $r_s = .53$), sample gender composition ($b = .001$, $p = .715$, $r_s = .24$) and mean age ($b = .003$, $p = .657$, $r_s = .04$) did not significantly moderate the relationship between defeat and entrapment

combined and suicidality effect sizes. The Spearman's correlation between defeat and entrapment combined and year of publication was moderate and approached statistical significance (p = .078). This could suggest that a larger sample size may reveal that year of publication is a significant moderator of suicidality effect sizes. A scatterplot (Figure 9) was generated to explore this correlation further. The scatterplot indicates virtually no relationship between the data points. The two data points on the right of the scatterplot therefore probably explain the nature of the correlation. For these reasons, the moderate to large-size Spearman's rho correlation should be discounted as it appears to be a spurious relationship arising because of two outliers. The coefficient of determination (R^2 = .35) indicated that 35% of the variance in suicidality effect sizes was accounted for by the year of publication.

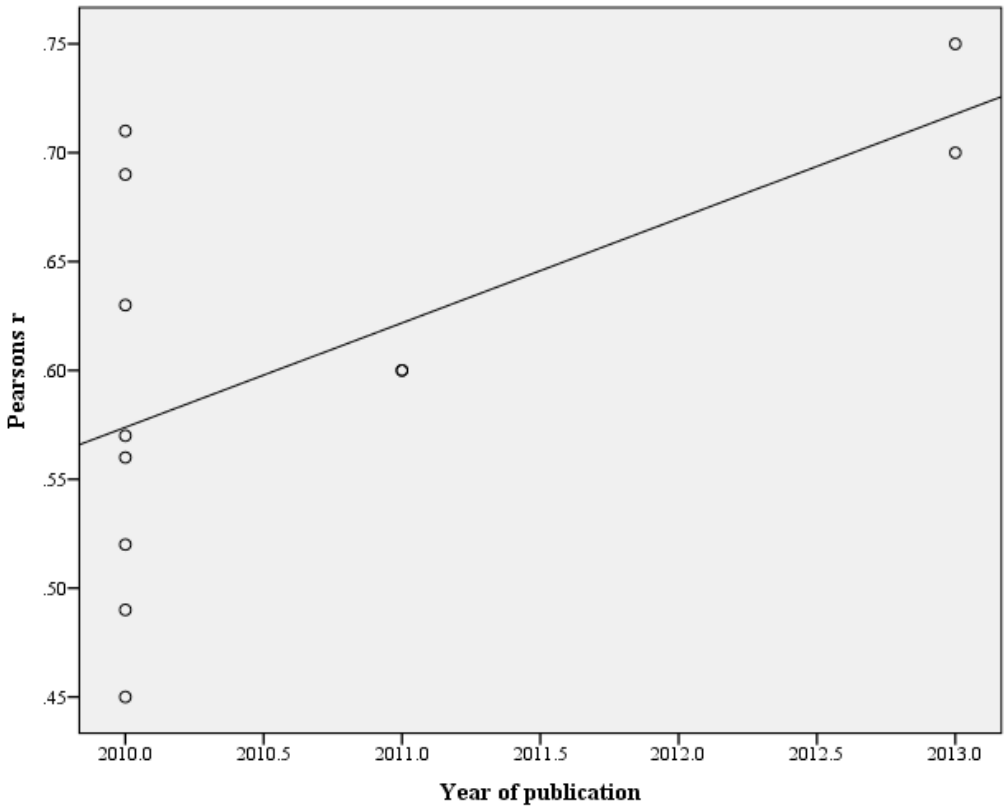

Figure 9. Scatterplot showing the relationship between year of publication and suicidality effect sizes when defeat and entrapment are combined.

3.5.7. Summary of moderator analyses

Differences across defeat and entrapment categories was a significant moderator of depression effect sizes. Gender composition of the sample, measure of depression and measure of defeat and entrapment all significantly moderated the relationship between defeat and entrapment combined and depression effect sizes. Year of publication significantly moderated the relationship between defeat and entrapment combined and anxiety problem effect sizes. All other moderator analyses were not significant. Overall, consistent with the fact that there was no between-study heterogeneity, most moderators did not exert a significant influence on the effect sizes included in the meta-analysis (i.e., moderators were examined to explain a small and unreliable amount of between-study heterogeneity). These results need to be interpreted tentatively, given that the relatively small number of effect sizes may have resulted in low power to detect the presence of moderators and an increased probability of falsely identifying moderators when they were not present (Hunter & Schmidt, 2004).

3.6. Publication bias

3.6.1. Funnel plots

In keeping with the recommended minimum number of effect sizes for using funnel plots (>10) (Sterne, Egger & Moher, 2008), funnel plots were created for seven of the meta-analyses. Figure 10 presents funnel plots of the relationships between defeat and entrapment combined and each of depression (k = 52), anxiety problems (k = 16), PTSD (k = 12) and suicidality (k = 12). There are a few outliers for the suicidality, anxiety problems and PTSD funnel plots. Given that some degree of asymmetry is to be expected in funnel plots with relatively few data points (Sterne, Sutton, Loannidis, Terrin, Jones, Lau, et al., 2011), the funnel plots for suicidality, anxiety problems and PTSD generally appear fairly symmetrical and funnel-shaped. The funnel plot for depression shows that the literature contains very few small (imprecise) studies. This pattern could be indicative of a one-tailed publication bias (Vevea & Woods, 2005) in which smaller studies are less likely to report relationships between defeat and entrapment and depression. The funnel plot for depression also shows that there are numerous outliers on both sides of the distribution of depression

effect sizes. This may indicate a two-tailed publication bias (Vevea & Woods, 2005) in which statistically-significant correlations in either direction were favoured for publication in larger studies. This latter result is consistent with the results of the boxplots conducted during exploratory data analysis, where there are various outliers on both sides of the depression distribution.

Figure 11 presents funnel plots of the relationships between depression and each of defeat (k = 16), internal entrapment (k = 13) and external entrapment (k = 16). There are a few outliers for all three funnel plots. Again recognising that some degree of asymmetry is to be expected in funnel plots with relatively few data points (Sterne et al., 2011), the funnel plots for defeat and external entrapment generally appear fairly symmetrical and funnel-shaped. The funnel plot for internal entrapment shows that the literature contains very few small studies, which could be indicative of a one-tailed publication bias (Vevea & Woods, 2005) in which smaller studies are less likely to report relationships between internal entrapment and depression. Overall, the seven funnel plots provide two indications of potential publication bias.

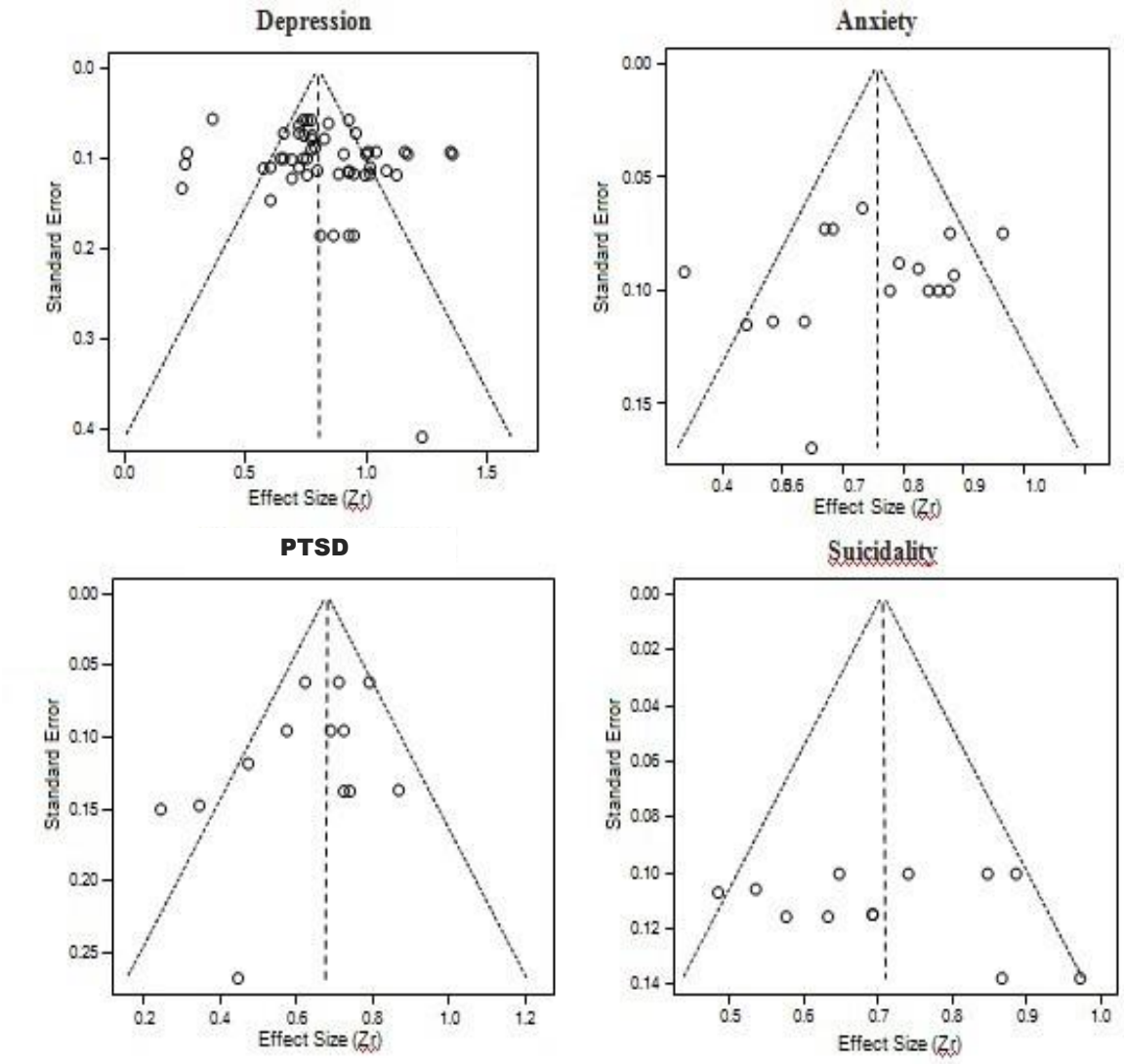

Figure 10. Funnels plots of relationships between defeat and entrapment combined and each of depression, suicidality, anxiety problems and PTSD. The vertical line is the population effect size and the diagonal line displays the 95% confidence interval.

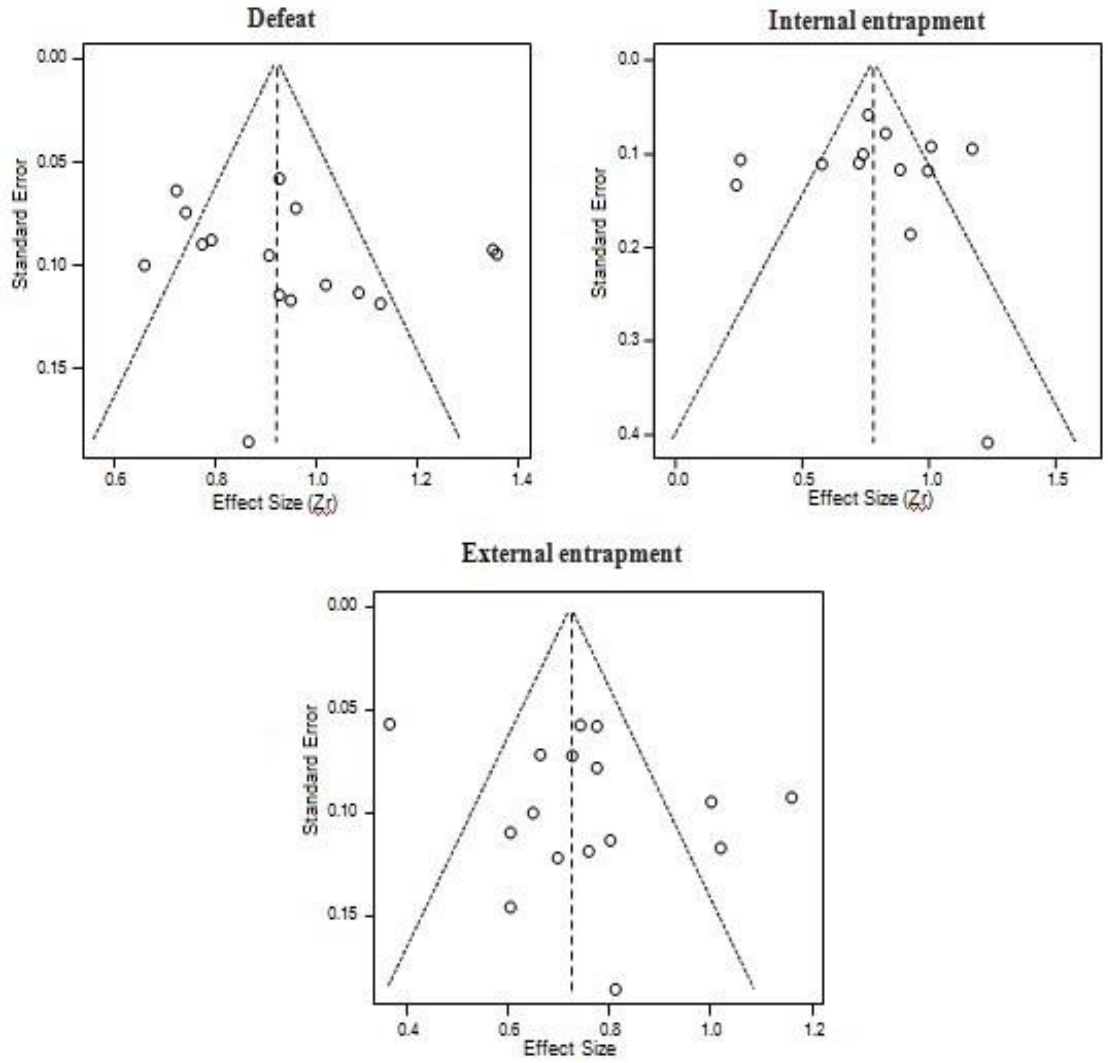

Figure 11. Funnels plots of relationships between each of defeat, internal entrapment and internal entrapment and depression. The vertical line is the population effect size and the diagonal line displays the 95% confidence interval.

3.6.2. Vevea and Woods' (2005) sensitivity analyses

To quantify the likely effect of publication bias, Vevea and Woods' (2005) sensitivity analysis using a priori weight functions was conducted for all meta-analyses. In Tables 5 and 6, r_{pb} is reported, which is an estimate of the population effect size when corrected for severe two-tailed publication bias. Severe two-tailed publication bias refers to a weighting function that simulates a hypothetical scenario in which studies publishing correlations near zero are less likely to be published and included in a meta-analysis, while significant correlations in either direction are more

likely to be published and therefore included in a meta-analysis (see Vevea and Woods (2005). Vevea and Woods' (2005) weight function model of publication bias was also used to calculate population effect size estimates under moderate and severe one- and two-tailed selection bias scenarios, but the results remained consistent with the reported model (see Appendix 5). In Tables 5 and 6, if r and r_{pb} are similar, then publication bias has had little effect. In all cases, even correcting for severe two-tailed publication bias has only very trivial effects on the population effect size estimates and certainly would not change the existing interpretations of them. These findings provide confidence that the population effect size estimates included in this meta-analysis are robust and have not been severely inflated by unpublished studies not included in the meta-analysis.

CHAPTER FOUR: DISCUSSION

4.1. Summary of findings

This meta-analysis quantitatively summarised the findings from 38 studies (11,343 participants) which examined relationships between perceptions of defeat and entrapment and four psychological problems commonly encountered in NHS clinical services: depression, suicidality, anxiety problems and PTSD. All correlations between defeat and entrapment and the psychological problems were large by Cohen's (1988) criterion, and there were significantly different-sized relationships depending on the type of psychological problem. Specifically, and consistent with IDS theory (e.g., Gilbert & Allan, 1998; Rohde, 2001; Sloman, 2000; Sloman et al., 2003), correlations between defeat and entrapment and depression were the largest, and were significantly larger than those for anxiety problems and PTSD. Correlations between defeat and entrapment and each of suicidality, anxiety problems and PTSD were generally of a similar size. There was no significant between-study heterogeneity in the distribution of effect sizes within each meta-analysis. A sensitivity analysis using a priori weight functions suggested that the population effect size estimates are robust and were not severely inflated by unpublished studies not included in the meta-analysis. Overall, these results are consistent with the earlier narrative review of Taylor et al (2011a), but additionally contribute to the literature by (a) bringing the literature review up to date through the inclusion of recent, important studies, (b) quantifying for the first time the relative size and consistency of the population effect size (the 'true' effect) for each of these relationships, (c) testing for statistical differences across psychological problems, (d) examining potential moderator variables, and (e) examining the potential for publication bias in the literature.

4.2. Discussion of findings

Guidelines regarding the interpretation of effect sizes suggest that the magnitude of the meta-analytic results can be considered large (Ahadi & Diener, 1989; Cohen, 1988; Kraemer, Morgan, Leech, Gliner, et al., 2003; Rosnow & Rosenthal, 2003). Indeed, Rosnow and Rosenthal (2003) suggest that effect sizes in the social sciences are oftentimes very small, and Cohen (1988) argued that when two variables measure different constructs, $r = .3$ is typical and $r = .5$ is about as large as

correlations get. In terms of interpreting the practical importance of these findings within the context of other mental health research (Ferguson, 2009; Kraemer et al., 2003; Prentice & Miller, 1992; Rosenthal, 1991; Rosnow & Rosenthal, 2003), in two meta-analyses of risk factors for adult PTSD (Brewin, Andrews & Valentine, 2000; Ozer, Best, Lipsey & Weis, 2003), for example[4], there were no large effects (effect sizes above $r = .50$: Cohen, 1988). Since even the moderate effect sizes from these previous meta-analyses were considered to have important clinical implications (Brewin et al, 2000; Ozer et al., 2003), the observed correlations in the current study can be considered both theoretically and clinically important.

The magnitude of the meta-analytic results introduces the possibility that defeat and entrapment, and perhaps other involuntary subordination constructs, may be integral components or driving forces behind depression, suicidality, anxiety problems and PTSD, rather than, for example, increasing the risk of these psychological problems via other mediating mechanisms. This postulation would suggest that perceptions of defeat and entrapment may be distal (involved in the aetiology of problems) and/or proximal (involved in maintaining problems) variables that are somehow integral to the four psychological problems examined; perhaps representing transdiagnostic processes that are common across various psychological problems (Harvey et al., 2004). IDS theory makes three broad hypotheses along these lines.

First, it suggests that perceptions of defeat act distally in the four psychological problems examined by initially activating the IDS, whereas perceptions of both defeat and entrapment act proximally in these psychological problems. The latter process occurs through the hypothesised formation of a self-reinforcing "depressogenic feedback loop" in which defeat, entrapment and the IDS reinforce each other continuously in a vicious circle (Taylor et al., 2011a). Two of the meta-analytic results are consistent with these theoretical hypotheses: (i) separate analyses for defeat, internal entrapment and external entrapment revealed a particularly large (Cohen, 1988) and consistent relationship between defeat and depression – the largest in the meta-analysis, and (ii) moderator analyses showed that the correlation between

[4] It was not possible to locate a meta-analysis of risk factors for depression.

defeat and depression (r = .74) was significantly larger than the correlations between sub-types of entrapment and depression (r = .63-.65). Although these results support the hypothesis that defeat acts distally in the four psychological problems by initially activating the IDS, it is possible that the particularly large relationship between perceptions of defeat and depression may have been artificially inflated as a result of measurement instrument differences. Specifically, it is possible that measures of defeat may have slightly different properties to measures of entrapment, which could lead to varying precision in the scores obtained from particular measurement instruments and potentially introduce spurious effect size differences (Baguley, 2009). It was not possible to test this possibility by examining differences across defeat and entrapment measures as a moderator variable. However, all included studies employed measures with adequate published psychometric properties. Therefore, if measurement instrument differences did introduce a bias to these results, this bias would be expected to be small.

Second, IDS theory suggests a reciprocal relationship between defeat and entrapment and the psychological problems, whereby perceptions of defeat and entrapment may influence the onset and maintenance of psychological problems and psychological problems, in turn, may influence the onset and maintenance of perceptions of defeat and entrapment (e.g., Gilbert & Allen, 1998; Taylor et al., 2011a). Although the meta-analytic results are consistent with this possibility, their cross-sectional nature means that it was not possible to statistically test this hypothesis. Furthermore, at this stage, the available longitudinal and experimental evidence is not able to clarify these questions either, since only one longitudinal study to date (Taylor et al., 2011b) has examined the possibility of a bidirectional relationship between defeat and entrapment and one of the four psychological problems (suicidality). Likewise, to date, one experimental study (Goldstein & Willner, 2002) has examined the relationship between a depressive mood induction and perceptions of defeat and entrapment. However, the reverse relationship not been investigated experimentally and mood inductions representing the other three psychological problems are needed.

The third broad hypothesis IDS theory makes regarding how defeat and entrapment may potentially act as distal or proximal drivers behind depression,

suicidality, anxiety problems and PTSD, is in predicting different-sized relationships across psychological problems. The theory suggests that depression arises directly through IDS activation, where depression is simply an IDS that has been active for longer than is functionally useful (Price et al., 1994; Sloman, 2000; Sturman, 2011; Taylor et al., 2011a). This suggests a close association and potentially more of a distal relationship between perceptions of defeat and entrapment and depression. In contrast, the association between perceptions of defeat and entrapment and each of anxiety problems and PTSD is thought to arise as a consequence of IDS activation (Taylor et al., 2011a), suggesting more of a proximal relationship between these variables. The results of this meta-analysis were in agreement with these theoretical suggestions, as correlations between defeat and entrapment and depression were significantly larger than those for anxiety problems and PTSD. With regard to suicidality, IDS theory suggests that the presence of a "depressogenic feedback loop" plus the availability of beliefs about the use of suicide as an escape strategy will lead to suicidality (Taylor et al., 2011a). The correlations observed here between defeat and entrapment and suicidality were large and of a similar-size to those for anxiety problems and PTSD. This potentially suggests more of a proximal relationship between defeat and entrapment and suicidality.

One potentially important challenge to the validity of the meta-analytic results, is the possibility that the large correlations between defeat and entrapment and suicidality, anxiety problems and PTSD arise merely as a result of the comorbidity of these three psychological problems with depression (see Mineka et al., 1998; Watson, 2009). However, the literature provides some evidence to refute this potential challenge. For example, Taylor et al. (2011b) found that defeat significantly predicted suicidality (twelve months later) when controlling for depressive symptoms (Taylor et al., 2011b); Birchwood et al. (2007) and Gumley et al. (2004) found that entrapment significantly predicted social anxiety when controlling for depressive and psychotic symptoms; and Jobson and O'Kearney (2009) found that defeat significantly predicted PTSD when controlling for depression. Together, these and a number of other studies in the literature provide preliminary evidence to suggest that the correlations between defeat and entrapment and suicidality, anxiety problems and PTSD are substantive (Podsakoff, Mackenzie, Lee & Podsakoff, 2003).

4.2.1. Discussion of effect size moderators

An important aim of this study was to examine whether particular moderator variables attenuate or accentuate the consistency of the population effect sizes. Given that there was no significant between-study heterogeneity in the distribution of effect sizes for any of the psychological problems, moderator analyses were undertaken on an exploratory basis in order to potentially improve methodological reporting in the literature and identify areas for future research.

Moderator analysis revealed that the gender composition of samples significantly moderated depression effect sizes, whereby samples made up of a higher percentage of females showed a stronger relationship with depression. This result is consistent with the well-established finding that in early adolescence and adulthood, women are twice as likely as men to experience depression (Kessler, McGonagle, Swartz, Blazer, et al., 1993; Nolen-Hoeksema, 1990, 2001; Weissman, Bland, Canino, Faravelli, et al., 1996). The literature concerning psychological problem gender differences attributes differential rates of depression across genders as being due to greater exposure to adversity and differing reactions to stressors. With regards to greater exposure to adversity, it is suggested that because of the nature of their social roles relative to men, women experience more chronic strains such as poverty, harassment, lack of respect and constrained choices (Nolen-Hoeksema, 2001; Nolen-Hoeksema & Girgus, 1994). Moreover, as a result of less power and status (Nolen-Hoeksema, 1990, 2001), women experience certain traumas, particularly sexual abuse, more often than men (Tolin & Foa, 2006). Additionally, even when women and men experience the same stressors, women may be more likely to develop depression (and anxiety problems) because of gender differences in biological responses to stressors (Breslau, Davis, Andreski, Peterson & Schultz, 1997). For example, an intriguing hypothesis is that women are more likely than men to develop a dysregulated HPA response to stress, which would make them more likely to develop depression in response to stress (Weiss, Longhurst & Mazure, 1999). Women may be more likely to have a dysregulated HPA response because they are more likely to have suffered traumatic events, which are known to contribute to HPA dysregulation. At present, the majority of these hypotheses remain untested, thereby highlighting the need to

investigate the relationship between demographic variables such as gender in future research.

Moderator analysis also revealed that measure of depression significantly moderated depression effect sizes. Depression effect sizes obtained using the Beck Depression Inventory were larger than those obtained using the Center for Epidemiological studies Depression Scale, the Hospital Anxiety and Depression Scale and other depression measures. It is not immediately clear why this would be the case, since the different depression measures have comparable psychometric properties (e.g., Beck, Steer & Garbin, 1988; Mykletun, Stordal & Dahl, 2001; Weissman, Sholomskas, Pottenger, Prusoff, et al., 1977). However, the significant moderator result suggests that careful consideration needs to be used when selecting depression scales for use in defeat and entrapment research.

Measure of defeat and entrapment also significantly moderated depression effect sizes. Depression effect sizes estimates obtained using the Defeat and Entrapment Scales were significantly larger than those obtained using alternative measures of defeat and entrapment. Because of low numbers of effect sizes, the other measures were aggregated to form one group consisting of the Personal Beliefs about Illness Questionnaire, Mental Defeat During Trauma Scale, Pain Self Perception Scale, Custom Interview Concerning Entrapment, Mental Defeat Rated from Narrative, Carer's Entrapment Scale and the Carer Burden Scale – Entrapment subscale. Taylor et al. (2011a) provide a useful overview of the psychometric properties of defeat and entrapment scales. It is noteworthy that many of the self-report instruments which operationalize defeat and entrapment (and therefore underpin the literature) were developed in ways that did not follow standard scale development practices (e.g., beginning with a broad, representative item pool, employing exploratory factor analysis: Clark & Watson, 1995; Floyd & Widaman, 1995; Worthington & Whittaker, 2006). This fact may explain this significant moderator effect, in that measures of varying quality may be in use in the defeat and entrapment literature. For example, the Defeat and Entrapment Scales (Gilbert & Allan, 1998) are the longest measure in the literature, introducing the possibility that these scales measure important aspects of defeat and entrapment that alternative measures of defeat and entrapment do not. However, it may alternatively be the case

that differing formats of measures (e.g., questionnaire versus narrative report) explain these differences. Clarifying this issue further represents an important area for future research.

Year of publication, mean age and clinical status of sample (community versus clinical samples) did not significantly moderate depression effect sizes. The latter result could be explained by the possibility that the relationship between defeat and depression is not any stronger in clinical groups (i.e., that perceptions of defeat and entrapment are continuous variables which have a linear relationship with depression). However, it may be that the coding system used to categorise samples into community versus clinical groups somehow masked important differences.

With the exception of the moderating role of year of publication on anxiety problem effect sizes, no other moderator variables significantly moderated suicidality, anxiety problem and PTSD effect sizes. These results are not surprising given the absence of between-study heterogeneity, meaning that moderators were examined to explain a small/unreliable amount of variability. Given the potential for low statistical power because of very low numbers of effect sizes (Hunter & Schmidt, 2004), it is important to recognise that failure to obtain a statistically significant difference among subgroups was not interpreted as evidence that the effect is the same across subgroups (Borenstein et al., 2009). For these reasons, moderator analyses for suicidality, anxiety problem and PTSD should be interpreted tentatively.

4.3. Limitations and recommendations for future research

This meta-analysis illustrates the promising and exciting nature of this area of research. However, it also indicates that much remains to be learned and highlights areas of research where future work is needed.

4.3.1. Meta-analytic methodology

Several aspects of the meta-analytic methodology warrant discussion. Most notable is the fact that the meta-analyses for suicidality, anxiety problems and PTSD were based on a small number of effect sizes. Drawing firm conclusions in such circumstances is not possible because the effect sizes included could reflect idiosyncrasies in the included studies, sampling bias, or may simply not be generalizable. Related to this point is the fact that 20 of the 38 included studies contributed multiple effect sizes, thereby violating the statistical assumption

underpinning meta-analyses that data points are independent (Borenstein et al., 2009). However, the similarity in results between meta-analyses when defeat and entrapment were combined and when these variables were separated into sub-categories, provides some degree of confidence that the results were probably not substantially biased.

It is also important to note the heavy reliance on self-report measures that characterizes the literature on which this meta-analysis is based. This approach is understandable given the highly subjective and idiosyncratic nature of perceptions of defeat and entrapment, and the relative infancy of the literature. However, it may be possible to develop alternative methods of measuring perceptions of defeat and entrapment. The development of narrative-based measures (e.g., Ehlers et al., 1998) as well as nonverbal behavioural measures (Sturman, 2011) of perceptions of defeat and entrapment highlight the possibility that future research will develop alternatives to self-report that may provide additional benefits in understanding the phenomenology of these experiences. Equally, future theory and research may indicate the development of refined self-report measures or the improvement of the psychometric quality of existing measures.

The literature reviewed here also heavily relies on cross-sectional designs. There is therefore a pressing need for additional longitudinal and experimental studies that have the potential to establish temporal precedence and causality, and to isolate the mechanisms responsible for observed effects. Although expensive and complex to analyse, experience sampling designs offer particular promise for examining the extent to which a range of dynamic moment-by-moment factors (e.g., thoughts, feelings, behaviour, interpersonal interactions, environment) may contribute to fluctuations in perceptions of defeat and entrapment, and whether these fluctuations lead to changes in psychological symptoms or functioning.

The literature search strategy used here was restricted to publications in English language, and it is not known to what extent this limitation may have influenced the findings (although the sensitivity analyses provide reassurance that this possibility was unlikely). For example, it is possible that sociocultural differences, which may manifest through language, could explain individual differences in perceptions of defeat and entrapment. Examining defeat and entrapment further in more diverse ethnic and cultural groups therefore represents an important area for

future research. Furthermore, this meta-analysis restricted its focus to adult samples because, to date, only two cross-sectional studies have used adolescent samples (Kidd, 2006; Park et al., 2010). This likewise highlights the importance of studying defeat and entrapment in children and adolescents, which may be particularly useful in clarifying questions around vulnerability to and onset of IDS malfunction.

In keeping with theory (Taylor et al., 2011a), this meta-analysis assumed that different stressors (triggering circumstances) are interchangeable and homogeneous in bringing about experiences of defeat or entrapment. For example, perceptions of entrapment by psychotic experiences were treated as being equivalent to perceptions of entrapment through a caregiving role. However, it remains an empirical question for future research to determine whether this assumption is accurate. It seems likely that a complex, multi-faceted relationship exists between triggering circumstances and the onset and maintenance of perceptions of defeat and entrapment, mediated by cognitive, systemic, environmental, sociocultural and perhaps other factors.

Conducting this review highlighted three recurrent shortcomings of the literature in terms of reporting conventions. First, it was often the case that studies did not report an effect size for every relationship examined, or sufficient statistical information that could be used to compute an effect size (e.g., reporting only that a finding was not statistically-significant). Second, presentation of descriptive statistics for all variables (rather than just those that were statistically-significant), was inconsistent. Third, sample, design and individual difference variables were inconsistently reported. These issues, which had direct bearing on the nature of the current meta and moderator analyses, can be easily remedied by researchers, reviewers and journal editors in future research.

4.3.2. IDS theory

The IDS theory (e.g., Gilbert, 1998; Nesse, 2000; Nettle, 2004; Sloman et al., 2003; Taylor et al., 2011a) provides a theoretical attempt to account for the onset and maintenance of specific psychological problems in terms of the malfunction of an evolutionarily-adaptive psychobiological mechanism. However, the theory has developed somewhat independently of empirical testing and various questions are raised which future iterations of the theory need to address.

A fundamental concern with the current theory is the lack of conceptual clarity regarding the factor structure of the IDS. Some preliminary attempts have been made to address this issue (e.g. Sturman, 2011; Taylor et al., 2009) but there is a pressing need to establish the bounds of the IDS construct (e.g., via exploratory and confirmatory factor analyses). In addition to the variables examined by Sturman (2011), it would be instructive to examine whether learned helplessness, depression and perhaps other variables, load onto a single higher-order involuntary subordination construct. For example, according to learned helplessness theory (Abramson, Seligman & Teasdale, 1978; Abramson, Metalsky & Alloy, 1989; Maier & Seligman, 1976; Peterson & Seligman, 1984), experience of uncontrollable events can lead an expectation of impotence. This expectation of a lack of control has been found to lead to motivational deficits (lowered response initiation and persistence), cognitive deficits (inability to perceive existing opportunities to control outcomes), and emotional deficits (sadness and lowered self-esteem), which are collectively known as learned helplessness. Therefore, learned helplessness appears to have face validity in conceptually overlapping with involuntary subordination constructs, but this possibility requires empirical testing. Once the factor structure of the IDS has been clearly defined, it will be useful to continue to examine relationships between the IDS and other psychological problems in order to extend current theory and inform clinical interventions.

As an evolutionary construct, the IDS is thought to be a reactive and adaptive mechanism that is sensitive to an individual's physical, social and internal environment (Buss et al., 1998; Tooby & Cosmides, 1990). However, the current literature has focused predominantly on individual cognitive experiences. Further exploration of the interplay of individual, interpersonal and sociocultural variables is therefore indicated in order to identify where clinical resources are most efficiently focused. For example, if empirical investigations reveal that perceptions of social support and belonging confer strong buffering effects against IDS activation, or perhaps directly de-activate the IDS, interventions focusing on improving a defeated or trapped individual's social environment would seem to be a clinical priority. Moreover, the theoretical hypothesis that targeting perceptions of defeat and

entrapment will lead to commensurate changes in associated (or non-associated) psychological problems, or vice-versa, remains untested.

Another major concern is that IDS theory is currently under-specified in terms of accounting for suicidality, PTSD and anxiety problems. For example, the link between perceptions of defeat and entrapment and each of anxiety problems and PTSD is thought to arise as a consequence of IDS activation (Taylor et al., 2011a). Depression is thought to arise directly through IDS activation, where depression is simply an IDS that has been active for longer than is functionally useful (Price et al., 1994; Sloman, 2000; Sturman, 2011; Taylor et al., 2011a). Taken together, these two suggestions imply that anxiety problems and PTSD are always comorbid with depression. However, this suggestion is inconsistent with the fact that anxiety and depression do not always co-occur (e.g., Mineka et al., 1998). The model therefore seems to have little specificity in explaining why anxiety problems and PTSD could occur in the absence of depression; nor does the theory make clear when, why and for whom IDS activation will lead to anxiety problems.

Similarly, the theory suggests that the presence of a "depressogenic feedback loop" plus the availability of beliefs about the use of suicide as an escape strategy will lead to suicidality. This appears to be an overly-simplistic explanation that fails to capture clinical complexity or the abundant theoretical and empirical base that underpins suicidality research. For example, IDS theory does not account for individual differences in the desire and the ability to die by suicide (as suggested by the interpersonal-psychological theory of suicide: Joiner, 2005); nor does it account for various key risk factors such as impulsivity (Kingsbury, Hawton, Steinhardt, & James, 1999), childhood adversity (Joiner, Sachs-Ericsson, Wingate, Brown, et al., 2007) and hopelessness (Brown, Beck, Steer, & Grisham, 2000). IDS theory also does not provide any detail regarding the formation and maintenance of beliefs about suicide as a potential escape strategy, which are seen to be central to theories of suicide (Johnson et al., 2008; Lau et al., 2004; Pratt et al., 2010; Williams et al., 2005).

The issue of model under-specification is apparent in several other respects. For example, the potential role of unhelpful methods of coping with clinical symptoms is acknowledged as a mediator between defeat and PTSD only (Taylor et

al., 2011a). However, there is no reason to believe that this process would not also be apparent for other psychological problems. Research regarding rumination (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008; Watkins, 2008), thought suppression (Wenzlaff & Wegner, 2000) and experiential avoidance (Hayes, Strosahl & Wilson, 1999), for example, has demonstrated that these methods of coping are transdiagnostic (Aldao & Nolen-Hoeksema, 2010; Harvey et al., 2004). The presence of unidirectional arrows in the IDS figure diagram by Taylor et al (2011a) also seems to be inconsistent with the textual description of the model (e.g., that psychological problems have a transactional relationship with perceptions of defeat and entrapment and the IDS). Perhaps these concerns are best resolved through the development of both a generic IDS model at the maximum level of abstraction, which would satisfy theorists and basic scientists, as well as more specific models (e.g., to explain specific psychological problems), which would offer practical utility in clinical and research settings (Dalgleish, 2004).

4.4. Clinical implications

The results of this meta-analysis suggest that perceptions of defeat and entrapment are strong risk factors for depression, suicidality, anxiety problems and PTSD. It is therefore important that clinicians are aware of the potential importance of these states and incorporate them into clinical assessment, formulation, intervention and evaluation.

Assessing for perceptions of defeat and entrapment is likely to have several clinical benefits. For example, defeat and entrapment have been shown to share variance with depression, suicidality, anxiety problems and PTSD that is not captured by other notable psychological risk factors (see Taylor et al., 2011a). This importantly suggests that routinely assessing for perceptions of defeat and entrapment will enable clinicians to describe and explain important aspects of individual phenomenological experience that would not otherwise be described. Likewise, incorporating perceptions of defeat and entrapment into clinical risk assessments may enhance the capacity of such instruments to identify at-risk individuals (Taylor et al., 2011a). For example, in the case of suicidality, measures of defeat and entrapment add predictive value over and above measures of depressive symptoms and hopelessness (e.g., Kidd, 2006; Park et al., 2010; Taylor et al., 2010a, b), suggesting that risk assessments incorporating

these constructs may be more accurate. Moreover, change on measures of defeat and entrapment over time may illustrate meaningful clinical change for an individual, thereby suggesting the potential usefulness of these constructs in monitoring therapeutic change and evaluating the outcome of therapy.

The evolutionary basis of defeat and entrapment lends itself to providing compassionate and normalising conceptualisations of presenting problems. For example, explicit in IDS theory is the suggestion that all humans have an evolved sensitivity to signals of social status and competition and that every individual has a set of social values involving the positive attention of others (Sloman et al., 2003; Sturman & Mongrain, 2008b). Conceptualising psychological problems and well-being in this manner has the potential to locate psychological problems on a continuum with other human experiences. Such an approach would also be thought to reduce stigma around mental health problems because it would, in effect, send the message that everyone is 'human' and that use of mental health services does not therefore indicate a flawed or defective character or a permanent change for the worse. The triggering circumstances that led to the individual's perceptions of defeat and entrapment could be positively reframed as providing useful information about what the individual values most in life. This information could then be used to plan interventions to directly address the individual's triggering circumstances. It is common for individuals in psychological distress to form unhelpful appraisals of psychological problems (Wells, 2008), or to experience them from family members or society (Dallos & Draper, 2010). Since unhelpful appraisals of psychological problems (e.g., "I am abnormal", "I am weird", "Things are never going to change") are thought to be a core maintaining process across psychological problems (e.g., Wells, 2008), providing an evolutionary-based conceptualisation is likely to make a therapeutic contribution in and of itself by introducing a more helpful narrative regarding individual experiences. This alternative story might be expected to reduce stigma, normalise experiences, make experiences more understandable (and therefore predictable and controllable) and instil hope that change is possible.

A clinical case conceptualisation which incorporates defeat, entrapment and the IDS has the potential to explain a wide range of interacting factors. For example, these constructs could be used to link an individual's presenting problems to their

current sociocultural and interpersonal context, including their interpersonal interactions. Linking these variables to previous contexts would potentially provide the opportunity to make psychological problems seem more understandable. Such a conceptualisation would provide a validating and normalising base from which to explore maintaining factors such as unhelpful ways of coping (e.g., too quick to back down, submit, or fight) and particular ways of thinking that are unhelpful in the individual's current context (e.g., seeing oneself as inferior to others, unattractive, incompetent, unwanted, etc). The idea that IDS malfunction involves intense, chronic, inflexible or inappropriate IDS activation (e.g., Gilbert, 1998; Nesse, 2000; Nettle, 2004; Sloman et al., 2003; Taylor et al., 2011a) can be used to explore with clients how to respond more *flexibly* to their environment, make sense of things and behave. Indeed, consistent with this idea, a recent review suggested that psychological flexibility is a core mechanism of therapeutic change that involves the ability to (i) recognize and adapt to various situational demands, (ii) shift mindsets or behavioural repertoires when these strategies compromise personal or social functioning, (iii) maintain balance among important life domains, and (iv) be aware, open, and committed to behaviours that are congruent with deeply held values (Kashdan & Rottenberg, 2010).

Although defeat and entrapment have different-sized correlations with the four psychological problems examined, the manner in which both states are thought to be activated, and therefore alleviated, is thought to be similar (Taylor et al., 2011a). Therefore, at present, it is suggested that clinicians focus equally on ameliorating perceptions of defeat and entrapment (Johnson et al., 2008; Rohde, 2001; Sloman et al., 2003; Tarrier, 2010). Perceptions of defeat and entrapment could be therapeutically addressed using several different but nevertheless complimentary approaches, which are now described.

Since an individual's interpersonal and sociocultural context are thought to influence the onset and maintenance of IDS activation, working with an individual and/or other significant people in their lives (e.g., other professionals, school, work colleagues) to meaningfully change the individual's environment, would be expected to have therapeutic benefits. For example, in a situation in which others continue to attack even though a particular individual has submitted (leading to perceptions of

defeat and entrapment), the clinician might work with the individual or the systems around the person to problem-solve useful ways to change this environment. Examples of such situations might include bullying, child abuse or domestic violence.

Likewise, the degree to which (appropriate and positive) social support is available is thought to mediate an individual's experience of defeat and entrapment (Sloman et al., 2003) and perhaps even buffer against unhelpful IDS activation. For example, having friends and family who know, understand and listen to an individual during a significant loss might help that individual to make sense of the experience and, in time, accept it and move on to new goals. It may therefore be helpful for clinicians to work with individuals to try to (realistically) enhance their level of social support and social interactions if the absence of these is seen to be contributory to their current problems.

An individual's historical context is seen to be important in potentially conferring vulnerability to perceptions of defeat and entrapment via repeated activation of the IDS (e.g. via illness, trauma or stress), which is thought to progressively lower the threshold for IDS activation over time (Sloman et al., 2003). This highlights the critical role that preventative and early interventions have in identify individuals experiencing excessive or chronic IDS activation and changing their context. Since perceptions of defeat and entrapment are strong risk factors for depression, suicidality, anxiety problems and PTSD, policies which directly reduce such perceptions would seem to be imperative. For example, anti-bullying and anti-harassment policies in schools and workplaces, and policies regarding working conditions which enable quality of life and optimise functioning (quality of life).

Along these lines, some authors (e.g., Gilbert, 2009; Seligman, 1998) have argued that the very nature of modern Western society contributes profoundly to the onset and maintenance of psychological problems. For example, Seligman (1998) suggests that modern society involves a strong emphasis on individuality, freedom, choice and positive moods, which he terms the "waxing of the self". Although these values are positive in many respects, they also, for example, lead to expectations that cannot be met, which means that individuals are faced on a daily basis with the fact that their expectations do not match reality (e.g., moods change over time; sometimes patience is necessary; it is not possible for everyone to be the most attractive, the

richest or the best). Seligman (1998) also suggests that modern society involves a diminished sense of community (e.g., reduced or no interactions with neighbours; long-distance commuting to work; increased divorce rates) and a reduction in a sense of higher purpose (e.g., God, the nation, family), which he terms the "waning of the commons". These factors mean that the human commitment to larger entities has weakened, causing people to look inward more for identity, meaning and coping. The findings of the current meta-analysis are consistent with Seligman's (1998) theory regarding the potential link between society and psychological problems. This link is consistent with IDS theory and has various clinical implications. For example, it would be expected that societal changes which promote a sense of higher purpose (e.g., religion, an orientation towards one's community), frequent and stable relationships with others (Baumeister & Leary, 1995) and realistic expectations (e.g., regarding mood, self, identity, aspirations) would be thought to reduce the frequency and severity of IDS activation and therefore reduce psychological problems. To give an example, some clinical problems (e.g., body dysmorhpia, eating problems) have been linked to messages regarding unhelpful and unrealistic cultural norms (e.g., achieving thinness is suggested to be profoundly meaningful). However, these sociocultural messages take place within a society which provides choice and availability in ways never seen before and the media puts forward images and stories of events and people from across the world, making very rare occurrences seem normal. In such instances, some of the focus of clinical interventions would be on broadening the individual's sense of self (e.g., away from physical image and weight, shape or control) and finding more helpful goals, values and expectations (e.g., Waller, Cordery, Corstorphine, Hinrichsen, et al., 2007).

Of course sometimes it will not be practical or useful to work towards changing an individual's environment. In these circumstances, interventions could be targeted at unhelpful cognitive or behavioural processes in order to alleviate perceptions of defeat and entrapment. For example, the therapeutic focus for an individual trapped in a defeating experience such as long-term imprisonment, chronic physical illness or a chronic psychological problem (e.g., psychotic experiences) might be in adjusting to this new reality and finding alternative, more constructive ways to relate to and cope with these experiences. This might involve shifting social

goals, values and expectations; in effect raising the individual's threshold to signals of defeat and entrapment (Johnson et al., 2008). Exploring new ways to define one's sense of self and meaning in relation to others (Rohde, 2001; Sloman et al., 2003) so that there is less discrepancy between these and an individual's environment would also be expected to reduce perceptions of defeat and entrapment. For example, an individual with unrealistic standards concerning personal success at work may benefit from a shift in emphasis to other personal roles (e.g., hobbies, sport, family, friends). Likewise, interventions designed to address unhelpful ways of coping (e.g., rumination, thought suppression) or thinking (e.g., unhelpful meta-cognitions) that may be maintaining perceptions of defeat and entrapment, would be expected to reduce IDS activation and assist in alleviating clinical problems. Based on previous experiences, an individual's threshold for IDS activation may have become relatively low, leading to this normally evolutionarily-adaptive process being inappropriately triggered. The extent to which an individual's IDS is (inappropriately) triggered could be addressed by working collaboratively with the individual to increase their assertiveness, self-esteem, self-efficacy and optimism, whilst reducing their self-criticalness (Carver, Scheier & Segersrom, 2010; Sloman et al., 2003; Sturman & Mongrain, 2008), for example. These changes might be achieved by examining the evidence and usefulness of the individual's self-perceived social rank or through other cognitive changes methods such as guided discovery (Beck, 1995).

4.5. Conclusion

This meta-analysis revealed large and consistent correlations between defeat and entrapment and each of depression, suicidality, anxiety problems and PTSD, introducing the possibility that defeat and entrapment, and perhaps other involuntary subordination constructs, may be integral components or driving forces behind these psychological problems; perhaps representing transdiagnostic processes that are common across various psychological problems. The results suggest that it is important for clinicians to be made aware of defeat, entrapment and the IDS constructs, and to incorporate these variables into clinical assessment, formulation, intervention and evaluation. They also suggest that wider society and policy-makers would benefit from an awareness of these constructs and other evolutionary ideas,

since they provide an important rationale for policies and social norms to facilitate prevention and early intervention.

The current theory which underpins this literature, the Involuntary Defeat Strategy (IDS) theory, has various strengths. However, this review also discussed a number of specific limitations. It is hoped that the weaknesses of the current theory and of the literature itself will be addressed by future research. Changes of this nature would be expected to make defeat and entrapment theory and research more clinically-relevant, ultimately leading to improved clinical interventions and NHS mental health outcomes.

REFERENCES

*** References marked with an asterisk indicate studies included in the meta-analysis.**

Abramowitz, J. S. (1997). Effectiveness of psychological and pharmacological treatments for obsessive–compulsive disorder: A quantitative review. *Journal of Consulting and Clinical Psychology, 65*, 44− 52.

Abramson, L.Y., Metalsky, G. I., & Alloy, L.B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96,* 359-372.

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*, 49-74.

Ahadi, S., & Diener, E. (1989). Multiple determinants and effect size. *Personality Process and Individual Differences, 56,* 398-406.

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

Allan, S., & Gilbert, P. (1995). A social comparison scale: Psychometric properties and relationship to psychopathology. *Personality and Individual Differences, 19,* 293–299.

*Allan, S., & Gilbert, P. (2002). Anger and anger expression in relation to perceptions of social rank, entrapment and depressive symptoms. *Personality and Individual Differences, 32,* 551–565.

Baguley, T. (2009). Standardized or simple effect size: What should be reported? *British Journal of Psychology, 100,* 603-617.

Barrick, M. R., & Mount, M. K. (1991). The big five personality dimensions and job performance: A meta-analysis. *Personnel Psychology, 44,* 1–26.

Baumeister, R. F. (1990). Suicide as escape from self. *Psychological Review, 97,* 90–113.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117, 497–529.*

Beck, A. T. (1983). Cognitive therapy of depression: New perspectives. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depression: Old controversies and new approaches* (pp. 265-290). New York, NY: Raven Press.

Beck, A. T. (1988). *Beck Depression Inventory*. New York: The Psychological Corporation.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York, NY: Guilford Press.

Beck, A.T., Steer, R.A., & Brown, G.K. (1996). *Beck Depression Inventory, 2nd Edition Manual*. San Antonio: The Psychological Corporation.

Beck, A. T., Steer, R. A., & Garbin, M. (1988). Psychometric properties of the Beck Depression Inventory: 25 years of evaluation. *Clinical Psychology Review, 8*¸ 77-100.

Begg, C. B. (1994). Publication bias. In H. Cooper & L. V. Hedges (Eds.), *The Handbook of Research Synthesis* (pp. 399−409). New York: Russell Sage Foundation.

Birchwood, M., Iqbal, Z., & Upthegrove, R. (2005). Psychological pathways to depression in schizophrenia: Studies in acute psychosis, post psychotic depression and auditory hallucinations. *European Archives of Psychiatry and Clinical Neuroscience, 255,* 202–212.

*Birchwood, M., Mason, R., MacMillan, F., & Healy, J. (1993). Depression, demoralization and control over psychotic illness: A comparison of depressed and non-depressed patients with a chronic psychosis. *Psychological Medicine, 23,* 387–395.

*Birchwood, M., Trower, P., Brunet, K., Gilbert, P., Iqbal, Z., & Jackson, C. (2007). Social anxiety and the shame of psychosis: A study in first episode psychosis. *Behaviour Research and Therapy, 45,* 1025–1037.

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis.* New York, NY: Wiley.

Breslau, N., Davis, G. C., Andreski, P., Peterson, E. L., & Schultz, L. (1997). Sex differences in posttraumatic stress disorder. *Archives of General Psychiatry, 54,* 1044–1048.

Brewer, M. B., & Caporael, L. R. (1990). Selfish genes versus selfish people: Sociobiology as origin myth. *Motivation and Emotion*, *14*, 237-243.

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

Broadhead, J. C., & Abas, M. A. (1998). Life events, difficulties and depression among women in an urban setting in Zimbabwe. *Psychological Medicine, 28*, 29–38.

Bromet, E., Andrade, L. H., Hwang, I., Sampson, N. A., Alonso, J., de Girolamo, D., et al. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine, 9*, 1-16.

Brown, G., Beck, A. T., Steer, R., & Grisham, J. (2000). Risk factors for suicide in psychiatric outpatients: A 20-year prospective study. *Journal of Consulting and Clinical Psychology, 68*, 371–377.

Brown, G. W., Harris, T. O., & Hepworth, C. (1995). Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison. *Psychological Medicine, 25*, 7-21.

Buss, D. M. (1995). Evolutionary psychology: A new paradigm for psychological science. *Psychological Inquiry, 6*, 1-30.

Buss, D. M. (2009). The great struggles of life: Darwin and the emergence of evolutionary psychology. *American Psychologist, 64*, 140-148.

Buss, D. M., Haselton, M. G., Shackelford, T. K., Bleske, A., & Wakefield, J. C. (1998). Adaptations, exaptations, and spandrels. *American Psychologist, 53*, 533-548.

Butler, G., Fennell, M., & Hackmann, A. (2008). *Cognitive behavioural therapy for anxiety disorders: Mastering clinical challenges*. New York: The Guilford Press.

Carver, C., Scheier, M. F., & Segersrom, S. C. (2010). Optimism. *Clinical Psychology Review, 30*, 879-889.

Centers for Disease Control and Prevention (2012). National Center for Injury Prevention and Control, suicide fact sheet. Available at

http://www.cdc.gov/ncipc/dvp/suicide/suicidedatasheet.pdf (accessed May 5, 2013).

*Clare, L., & Singh, K. (1994). Preventing relapse in psychotic illness: A psychological approach to early intervention. *Journal of Mental Health, 3,* 541–550.

Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment, 7,* 309-319.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cohen, S., & Wills, T. A. (1985). Stress, social support and the buffering hypothesis. *Psychological Bulletin, 98,* 310-357.

Cohn, L. D. & Becker, B. J. (2003). How meta-analysis increases statistical power. *Psychological Methods*, 8, 243-253.

Confer, J. C., Easton, J. E., Fleischman, D. S., Goetz, C., Lewis, D. M., Perilloux, C., & Buss, D. M. (2010). Evolutionary psychology: Controversies, questions, prospects, and limitations. *American Psychologist, 65,* 110-126.

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

Dallos, R., & Draper, R. (2010). *An introduction to family therapy: Systemic theory and practice* (3rd ed.). Maidenhead, UK: Open University Press.

Davey, G. C. L., & Bond, N. (2006). Using controlled comparisons in disgust psychopathology research: The case of disgust, hypochondriasis and health anxiety. *Journal of Behavior Therapy and Experimental Psychiatry*, *37*, 4-15.

Dersimonian, R., & Laird, N. (1986). Meta-analysis in clinical-trials. *Controlled Clinical Trials*, *7*(3), 177−188.

Dixon, A. K. (1998). Ethological strategies for defence in animals and humans: Their role in some psychiatric disorders. *British Journal of Medical Psychology, 71,* 417–445.

Dixon, A. K., Fisch, H. U., Huber, C., & Walser, A. (1989). Ethological studies in animals and man: Their use of psychiatry. *Pharmacopsychiatry, 22*, 44-50.

*Dunmore, E., Clark, D. M., & Ehlers, A. (1997). Cognitive factors in persistent versus recovered post-traumatic stress disorder after physical or sexual assault: A pilot study. *Behavioural and Cognitive Psychotherapy, 25*, 147–159.

*Dunmore, E., Clark, D. M., & Ehlers, A. (1999). Cognitive factors involved in the onset and maintenance of posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy, 37*, 809–829.

Dunmore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy, 39*, 1063–1084.

Egger, M., Smith, G. D., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical tests. *British Medical Journal, 315*, 629-634.

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

Ehlers, A., Clark, D. M., Dunmore, E., Jaycox, L., Meadows, E., & Foa, E. B. (1998). Predicting response to exposure treatment in PTSD: The role of mental defeat and alienation. *Journal of Traumatic Stress, 11*, 457–471.

Ehlers, A., Maercker, A., & Boos, A. (2000). Posttraumatic stress disorder following political imprisonment: The role of mental defeat, alienation, and perceived permanent change. *Journal of Abnormal Psychology, 109*, 45–55.

Ferguson, C. J. (2009). An effect size primer: A guide for clinicians and researchers. *Professional Psychology: Research and Practice, 40*, 532-538.

Field, A. P. (2003). The problem in using fixed-effects models of meta-analysis on real-world data. *Understanding Statistics, 2*, 77−96.

Field, A. P. (2005a). *Discovering statistics using SPSS: And sex and drugs and rock 'n' roll* (2nd ed.). London: Sage.

Field, A. P. (2005b). Is the meta-analysis of correlation coefficients accurate when population effect sizes vary? *Psychological Methods, 10*, 444−467.

Field, A. P. & Gillett, R. (2010). How to do a meta-analysis. *British Journal of Mathematical and Statistical Psychology, 63*, 665–694.

Floyd, F. J., & Widaman, K. F. (1995). Factor analysis in the development and refinement of clinical assessment instruments. *Psychological Assessment, 7*, 286-299.

Garber, J., & Hollon, S. D. (1991). What can specificity designs say about causality in psychopathology research? *Psychological Bulletin, 110,*129–136.

*Garcia-Campayo., Rodero, B., Lopez del Hoyo, Y., Luciano, JV., Alda, M., & Gili, M. (2010). Validation of a Spanish language version of the pain self-perception scale in patients with fibromyalgia. *BMC Musculoskeletal Disorders, 11*, 255-262.

Gatward, N. (2007). Anorexia nervosa: An evolutionary puzzle. *European Eating Disorders Review, 125,* 1-12.

Gilbert, P. (1989). *Human nature and suffering*. London: Lawrence Erlbaum Associates.

Gilbert, P. (1992). *Depression: The evolution of powerlessness.* New York, NY: Guilford Press.

Gilbert, P. (1998). Evolutionary psychopathology: Why isn't the mind better designed than it is? *British Journal of Medical Psychology, 71*, 353-373.

Gilbert, P. (2000). Varieties of submissive behavior as forms of social defense: Their evolution and role in depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 3–45). Mahwah, NJ: Erlbaum.

Gilbert, P. (2001). Depression and stress: A biopsychosocial exploration of evolved functions and mechanisms. *Stress, 4,* 121–135.

Gilbert, P. (2009). *The compassionate mind: A new approach to life's challenges*. London: Constable.

Gilbert, P., & Allan, S. (1994). Assertiveness, submissive behaviour and social comparison. *British Journal of Clinical Psychology, 33,* 295–306.

*Gilbert, P., & Allan, S. (1998). The role of defeat and entrapment (arrested flight) in depression: An exploration of an evolutionary view. *Psychological Medicine, 28,* 585–598.

*Gilbert, P., Allan, S., Brough, S., Melley, S., & Miles, J. N. V. (2002). Relationship of anhedonia and anxiety to social rank, defeat, and entrapment. *Journal of Affective Disorders, 71,* 141–151.

Gilbert, P., Birchwood, M., Gilbert, J., Trower, P., Hay, J., Murray, B., . . . Miles, J. N. V. (2001). An exploration of evolved mental mechanisms for dominant and subordinate behaviour in relation to auditory hallucinations in schizophrenia and critical thoughts in depression. *Psychological Medicine, 31,* 1117–1127.

*Gilbert, P., Cheung, M., Irons, C., & McEwan, K. (2005). An exploration into depression-focused and anger-focused rumination in relation to depression in a student population. *Behavioural and Cognitive Psychotherapy, 33,* 273–283.

Gilbert, P., & Gilbert, J. (2003). Entrapment and arrested fight and flight in depression: An exploration using focus groups. *Psychology and Psychotherapy: Theory, Research and Practice, 76,* 173–188.

*Gilbert, P., Gilbert, J., & Irons, C. (2004). Life events, entrapments and arrested anger in depression. *Journal of Affective Disorders, 79,* 149– 160.

*Goldstein, R. C., & Willner, P. (2002). Self-report measures of defeat and entrapment during a brief depressive mood induction. *Cognition & Emotion, 16,* 629–642.

*Griffiths, A. W., Wood, A. M., Maltby, J., & Taylor, P. J. (in submission). The prospective role of defeat and entrapment in depression, anxiety, psychological well-being and coping. *Psychiatry Research*

*Gumley, A., O'Grady, M., Power, K., & Schwannauer, M. (2004). Negative beliefs about self and illness: A comparison of individuals with psychosis with or without comorbid social anxiety disorder. *Australian and New Zealand Journal of Psychiatry, 38,* 960–964.

Hagen, E. H. (2002). Depression as bargaining: The case postpartum. *Evolution and Human Behavior, 23,* 323–336.

Harvey, A., Watkins, E., Mansell W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. Oxford: Oxford University Press.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Hedges, L. V. (1992). Meta-analysis. *Journal of Educational Statistics, 17,* 279–296.

Hedges, L. V., & Pigott, T. D. (2001). The power of statistical tests in meta-analysis. *Psychological Methods, 6,* 203-217.

Hedges, L. V., & Vevea, J. L. (1998). Fixed- and random-effects models in meta-analysis. *Psychological Methods, 3,* 486−504.

Hewitt, P. L., & Flett, G. L. (1991). Perfectionism in the self and social contexts: Conceptualization, assessment, and association with psychopathology. *Journal of Personality and Social Psychology, 60,* 456–470.

Higgins, J. P. T., & Thompson, S. G. (2002). Quantifying heterogeneity in a meta-analysis. S*tatistics in Medicine, 21,* 1539-1558.

Higgins, J. P. T., & Thompson, S. G., Deeks, J. J., Altman, D. G. (2003). Measuring inconsistency in meta-analyses. *British Medical Journal, 327,* 557-60.

Huedo-Medina, T. B., Sánchez-Meca, J., Marín-Martínez, F., & Botella, J. (2006). Assessing heterogeneity in meta-analysis: *Q* statistic or I^2 *index? Psychological Methods, 11,* 193-206.

Hunter, J. E., & Schmidt, F. L. (2000). Fixed effects vs. random effects meta-analysis models: Implications for cumulative research knowledge. *International Journal of Selection and Assessment, 8,* 275−292.

Hunter, J. E., & Schmidt, F. L. (2004). *Methods of meta-analysis: Correcting error and bias in research findings* (2nd ed.). Newbury Park, CA: Sage.

*Iqbal, Z., Birchwood, M., Chadwick, P., & Trower, P. (2000). Cognitive approach to depression and suicidal thinking in psychosis. 2. Testing the validity of a social ranking model. *British Journal of Psychiatry, 177,* 522–528.

*Jobson, L., & O'Kearney, R. T. (2009). Impact of cultural differences in self on cognitive appraisals in posttraumatic stress disorder. *Behavioural and Cognitive Psychotherapy, 37,* 249 –266.

Johnson, J., Gooding, P., & Tarrier, N. (2008). Suicide risk in schizophrenia: Explanatory models and clinical implications. *Psychology and Psychotherapy: Theory, Research and Practice, 81,* 55-77.

Joiner, T. E. (2005). *Why people die by suicide*. Cambridge, MA: Harvard University Press.

Joiner, T. E., Jr., Sachs-Ericsson, N. J., Wingate, L. R., Brown, J. S., Anestis, M. D., & Selby, E. A. (2007). Childhood physical and sexual abuse and lifetime number of suicide attempts: A persistent and theoretically important relationship. *Behaviour Research and Therapy, 45,* 539–547.

Judd, L. J. (1997). The clinical course of unipolar major depressive disorders. *Archives of General Psychiatry, 54,* 989–991.

*Karatzias, T., Gumley, A., Power, K., & O'Grady, M. (2007). Illness appraisals and self-esteem as correlates of anxiety and affective comorbid disorders in schizophrenia. *Comprehensive Psychiatry, 48,* 371–375.

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review, 30,* 865-878.

Kendler, K., Hettema, J., Butera, F., Gardner, C., & Prescott, C. (2003). Life event dimensions of loss, humiliation, entrapment, and danger, in the prediction of onsets of major depression and generalised anxiety. *Archives of General Psychiatry, 60,* 789-796.

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey I: Lifetime prevalence, chronicity, and recurrence. *Journal of Affective Disorders, 29,* 85–96.

Kessler, R.C., Petukhova, M., Sampson, N.A., Zaslavsky, A.M., Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the US. *International Journal of Methods in Psychiatric Research, 21,* 169-184.

Kessler, R. C., & Wang, P. S. (2008). The descriptive epidemiology of commonly occurring mental disorders in the United States. *Annual Review of Public Health, 29,* 115-129.

Kidd, S. A. (2006). Factors precipitating suicidality among homeless youth: A quantitative follow-up. *Youth & Society, 37,* 393–422.

Kingsbury, S., Hawton, K., Steinhardt, K. M., & James, A. (1999). Do adolescents who take overdoses have specific psychological characteristics? A

comparative study with psychiatric and community controls. *Journal of the American Academy of Child & Adolescent Psychiatry, 38,* 1125–1131.

Kleim, B., Ehlers, A., & Glucksman, E. (2007). Early predictors of chronic post-traumatic stress disorder in assault survivors. *Psychological Medicine, 37,* 1457–1467.

Kraemer, H. C., Morgan, G. A., Leech, N. L., Gliner, J. A., Vaske, J. J., & Harmon, J. J. (2003). Measures of clinical significance. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 1524-1529.

Lau, M. A., Segal, Z. V., & Williams, J. M. G. (2004). Teasdale's differential activation hypothesis: Implications for mechanisms of depressive relapse and suicidal behaviour. *Behaviour Research and Therapy, 42,* 1001–1017.

Leblanc, A. J., Driscoll, A. K., & Pearlin, L. I. (2004). Religiosity and the expansion of caregiver stress. *Aging & Mental Health, 8,* 410–421.

Light, R. J., & Pillemer, D. B. (1984). *Summing up: The science of reviewing research*. Cambridge, MA: Harvard University Press.

Lipsey, M.W., & Wilson, D.B. (2001). *Practical meta-analysis*. Thousand Oaks, CA: Sage.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10,* 434–445.

Macaskill, P., Walter, S. D., & Irwig, L. (2001). A comparison of methods to detect publication bias in meta-analysis. *Statistics in Medicine, 20,* 641-654.

Maier, S. E, & Seligman, M. E. P. (1976). Learned helplessness: Theory and evidence. *Journal of Experimental Psychology: General, 105,* 3-46.

Marascuillo, L.A., Busk, P.L., & Serlin, R.C. (1988). Large sample multivariate procedures for comparing and combining effect sizes within a single study. *Journal of Experimental Education, 58,* 69-85.

*Martin, Y., Gilbert, P., McEwan, K., & Irons, C. (2006). The relation of entrapment, shame and guilt to depression, in carers of people with dementia. *Aging and Mental Health, 10,* 101–106.

McCrory, E., De Brito, S. A., & Viding, E. (2010). The neurobiology and genetics of maltreatment and adversity. *The Journal of Child Psychology and Psychiatry, 51,* 1079-1095.

Mineka, D., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49,* 377-412.

Mirza, R., & Jenkins, R. (2004). Risk factors, prevalence, and treatment of anxiety and depressive disorders in Pakistan: Systematic review. *British Medical Journal, 328,* 794−797.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & The PRISMA Group. (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses: The PRISMA Statement. *British Medical Journal, 339,* 332-336.

Mykletun, A., Stordal, E., & Dahl, A. A. (2001). Hospital Anxiety and Depression (HAD) scale: Factor structure, item analysis and internal consistency in a large population. *British Journal of Psychiatry, 179,* 540-544.

National Research Council (1992). *Combining information: Statistical issues and opportunities for research*. Washington, D.C.: National Academy Press.

Nesse, R. (1998). Emotional disorders in evolutionary perspective. *British Journal of Medical Psychology, 71,* 397–415.

Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry, 57,* 14–20.

Nettle, D. (2004). Evolutionary origins of depression: A review and reformulation. *Journal of Affective Disorders, 81,* 91–102.

Nolen-Hoeksema, S. (1990). *Sex differences in depression*. Stanford, CA: Stanford University Press.

Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Science, 10,* 173-176.

Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression in adolescence. *Psychological Bulletin, 11,* 424–443.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3,* 400-425.

O'Connor, R. C. (2003). Suicidal behavior as a cry of pain: Test of a psychological model. *Archives of Suicide Research, 7,* 297-308.

Osburn, H. G., & Callender, J. (1992). A note on the sampling variance of the mean uncorrected correlation in meta-analysis and validity generalization. *Journal of Applied Psychology, 77*, 115-122.

Overton, R. C. (1998). A comparison of fixed-effects and mixed (random-effects) models for meta-analysis tests of moderator variable effects. *Psychological Methods, 3,* 354−379.

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weis, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin, 129,* 52–73.

Park, Y.-J., Ryu, H., Han, K., Kwon, J. H., Kim, H. K., Kang, H. C., et al. (2010). Suicidal ideation in adolescents: An explanatory model using LISREL. *Western Journal of Nursing Research, 32,* 168–184.

*Panagioti, M., Gooding, P. A., Taylor, P. J., & Tarrier, N. (2012). Negative self-appraisals and suicidal behavior among trauma victims experiencing PTSD symptoms: The mediating role of defeat and entrapment. *Depression & Anxiety, 29,* 187-194.

Peterson, C., & Seligman, M. E. P. (1984). Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review, 91,* 347-374.

Podsakoff, P. M., MacKenzie, S. B., Lee, J-Y., & Podsakoff, N. P. (2003). Common method biases in behavioral research: A critical review of the literature and recommended remedies. *Journal of Applied Psychology, 88,* 879–903.

Pratt, D., Gooding, P., Johnson, J., Taylor, P., & Tarrier, N. (2010). Suicide schemas in non-affective psychosis: An empirical investigation. B*ehaviour Research and Therapy, 48,* 1211–1220.

Preacher, K.J., Rucker, D. D., MacCallum, R. C., & Nicewander, W. A. (2005). Use of the extreme groups approach: A critical reexamination and new recommendations. *Psychological Methods, 10,* 178-192.

Prentice, D. A., & Miller, D. T. (1992). When small effects are impressive. *Psychological Bulletin, 112,* 160-164.

Price, J., Sloman, L., Gardner, R., Jr., Gilbert, P., & Rohde, P. (1994). The social competition hypothesis of depression. *British Journal of Psychiatry, 164,* 309–315.

Radloff, L. S. (1977). The CES-D scale: a new self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385–401.

R Development Core Team (2010). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing (Retrieved from http://www.R-project.org)

*Rasmussen, S. A., Fraser, L., Gotz, M., MacHale, S., Mackie, R., Masterton, G., & O'Connor, R. C. (2010). Elaborating the cry of pain model of suicidality: Testing a psychological model in a sample of first-time and repeat self-harm patients. *British Journal of Clinical Psychology, 49,* 15–30.

Rohde, P. (2001). The relevance of hierarchies, territories, defeat for depression in humans: Hypotheses and clinical predictions. *Journal of Affective Disorders, 65*, 221–230.

Rooke, O., & Birchwood, M. (1998). Loss, humiliation and entrapment as appraisals of schizophrenic illness: A prospective study of depressed and non-depressed patients. *British Journal of Clinical Psychology*, *37*, 259-268.

Rosenthal, R. (1979). The 'file drawer problem' and tolerance for null results. *Psychological Bulletin, 86,* 638−641.

Rosenthal, R. (1991). *Meta-analytic procedures for social research* (revised ed.). Newbury Park, CA: Sage.

Rosenthal, R. (1995). Writing meta-analytic reviews. *Psychological Bulletin, 118,* 183−192.

Rosnow, R., & Rosenthal, R. (2003). Effect sizes for experimenting psychologists. *Canadian Journal of Experimental Psychology, 57*, 221–237.

Seligman, M. E. P. (1998). *Learned optimism: How to change your mind and your life*. New York, NY: Free Press.

Selten, J.-P., & Cantor-Graae, E. (2005). Social defeat: Risk factor for schizophrenia? *The British Journal of Psychiatry, 187*, 101–102.

Selten, J.-P., & Cantor-Graae, E. (2007). Hypothesis: Social defeat is a risk factor for schizophrenia? *The British Journal of Psychiatry, 191*, s9–s12.

Shea, B. J., Grimshaw, J. M., Wells, G. A., Boers, M., Andersson, N., Hamel, C., Porter, A. C., Tugwell, P., Moher, D., & Bouter, L. M. (2007). Development

of AMSTAR: A measurement tool to assess the methodological quality of systematic reviews. *BMC Medical Research Methodology, 7,* 10-17.

Shea, B. J., Hamel, C., Wells, G. A., Bouter, L. M., Kristjansson, E., Grimshaw, J., Henry, D. A., & Boers, M. (2009). AMSTAR is a reliable and valid measurement tool to assess the methodological quality of systematic reviews. *Journal of Clinical Epidemiology, 62,* 1013-1020.

Shively, C. A. (1998). Social subordination stress, behavior, and central monoaminergic function in female cynomolgus monkeys. *Biological Psychiatry, 44,* 882–891.

Shively, C. A., Laber-Laird, K., & Anton, R. F. (1997). Behavior and physiology of social stress and depression in female cynomolgus monkeys. *Biological Psychiatry, 41,* 871– 882.

Sloman, L. (2000). How the involuntary defeat strategy relates to depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 47–67). Mahwah, NJ: Erlbaum.

Sloman, L., Gilbert, P., & Hasey, G. (2003). Evolved mechanisms in depression: The role and interaction of attachment and social rank in depression. *Journal of Affective Disorders, 74,* 107–121.

Sterne, J. A. C., Egger, M., & Moher, D. (2008). Addressing reporting biases. In: J. P. T. Higgins & S. Green (Eds.), *Cochrane handbook for systematic reviews of interventions.* Version 5.0.1 [updated September 2008]. The Cochrane Collaboration, 2008. Available from www.cochrane-handbook.org.

Sterne, J. A. C., Sutton, A. J., Loannidis, J. P. A., Terrin, N., Jones, D. R., Lau, J., et al. (2011). Recommendations for examining and interpreting funnel plot asymmetry in meta-analyses of randomised controlled trials. *British Medical Journal, 343,* 302-307.

*Stommel, M., Given, C. W., & Given, B. A. (1990). Depression as an overriding variable explaining caregiver burdens. *Journal of Aging and Health, 2,* 81–102.

Stowkowy, J., & Addington, J. (2012). Maladaptive schemas as as mediator between social defeat and positive symptoms in young people at clinical high risk for psychosis. *Early Intervention in Psychiatry*, *6*, 87-90.

*Sturman, E. D. (2011). Involuntary subordination and its relation to personality, mood, and submissive behavior. *Psychological Assessment, 23*, 262-276.

Sturman, E. D., & Mongrain, M. (2005). Self-criticism and major depression: An evolutionary perspective. *British Journal of Clinical Psychology, 44*, 505–519.

*Sturman, E. D., & Mongrain, M. (2008a). Entrapment and perceived status in graduate students experiencing a recurrence of major depression. *Canadian Journal of Behavioural Science, 40,* 185–188.

Sturman, E. D., & Mongrain, M. (2008b). The role of personality in defeat: A revised social rank model. *European Journal of Personality, 22,* 55–79.

*Tang, N. K. Y., Goodchild, C. E., Hester, J., & Salkovskis, P. M. (2010). Mental defeat is linked to interference, distress and disability in chronic pain. *Pain, 149*, 547–554.

*Tang, N. K. Y., Salkovskis, P. M., & Hanna, M. (2007). Mental defeat in chronic pain: Initial exploration of the concept. *Clinical Journal of Pain, 23,* 222–232.

*Taylor, P. J., Gooding, P. A., Wood, A. M., Johnson, J., Pratt, D., & Tarrier, N. (2010a). Defeat and entrapment in schizophrenia: The relationship with suicidal ideation and positive psychotic symptoms. *Psychiatry Research, 178,* 244–248.

Taylor, P. J., Gooding, P., Wood, A. M., & Tarrier, N. (2011a). The role of defeat and entrapment in depression, anxiety, and suicide. *Psychological Bulletin, 137*, 391-420.

Taylor, P. J., Wood, A. M., Gooding, P., Johnson, J., & Tarrier, N. (2009). Are defeat and entrapment best defined as a single construct? *Personality and Individual Differences, 47,* 795–797.

*Taylor, P. J., Wood, A. M., Gooding, P., & Tarrier, N. (2010b). Appraisals and suicidality: The mediating role of defeat and entrapment. *Archives of Suicide Research, 14,* 236–247.

*Taylor, P. J., Wood, A. M, Gooding, P. A., & Tarrier, N. (2011b). Prospective predictors of suicidality: Defeat and entrapment lead to changes in suicidal ideation over time. *Suicide and Life Threatening Behaviour, 41*, 297-306.

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion, 2,* 247–274.

Teicher, M.H., Andersen, S.L., Polcari, A., Anderson, C.M., Navalta, C.P., & Kim, D.M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neuroscience and Biobehavioral Reviews*, *27*, 33–44.

Terrin, N., Schmid, C. H., Lau, J., & Olkin, I. (2003). Adjusting for publication bias in the presence of heterogeneity. *Statistics in Medicine, 22,* 2113–2126.

Tolin, D. F., & Foa, E. B. (2006). Sex differences in trauma and posttraumatic stress disorder: A quantitative review of 25 years of research. *Psychological Bulletin, 132*, 959-992.

Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality*, *58*, 17-68.

*Trachsel, M., Krieger, T., Gilbert, P., & Holtforth, M. G. (2010). Testing a German adaptation of the entrapment scale and assessing the relation to depression. *Depression Research and Treatment*, 1-10.

*Troop, N. A. (forthcoming paper). Unpublished data.

*Troop, N. A., & Baker, A. H. (2008). The specificity of social rank in eating disorder versus depressive symptoms. *Eating Disorders, 16*, 331– 341.

*Troop, N. A., & Hiskey, S. (in press). Social defeat and PTSD symptoms following trauma. *Clinical Psychology & Psychotherapy?*

*Troop, N. A., Andrews, L., Hiskey, S., & Treasure, J. L. (2013). Social rank and symptom change in eating disorders: A 6-month longitudinal study. *Clinical Psychology & Psychotherapy*.

Uchino, B. N., Cacippo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488-531.

Vevea, J. L., & Woods, C. M. (2005). Publication bias in research synthesis: Sensitivity analysis using a priori weight functions. *Psychological Methods*, *10*, 428−443.

Wakefield, J. C. (1992). Disorder as harmful dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review*, *99*, 232-247.

Wakefield, J. C. (1999). Evolutionary versus prototype analyses of the concept of disorder. *Journal of Abnormal Psychology*, *108*, 374-399.

Wakefield, J. C. (2007). The concept of mental disorder: Diagnostic implications of the harmful dysfunction analysis. *World Psychiatry*, *6*, 149-156.

Waller, G., Cordery, H, Corstorphine, E., Hinrichsen, H., Lawson, R., Mountford, V., et al. (2007). *Cognitive-behavioral therapy for the eating disorders: A comprehensive treatment guide*. New York: Cambridge University Press.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, *134*, 163-206.

Watson, D. M. (2009). Differentiating the mood and anxiety disorders: A quadripartite model. *Annual Review of Psychology*, *5*, 221-247.

Weiss, E. L., Longhurst, J. G., & Mazure, C. M. (1999). Childhood sexual abuse as a risk factor for depression in women: Psychosocial and neurobiological correlates. *American Journal of Psychiatry*, *156*, 816–828.

Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H.-G., Joyce, P. R., Karam, E. G., Lee, C.-K., Lellouch, J., Lepine, J.-P., Newman, S. C., Rubio-Stipc, M., Wells, E., Wickramaratne, P. J., Wittchen, H.-U., & Yeh, E.-K. (1996). Cross-national epidemiology of major depression and bipolar disorder. *Journal of the American Medical Association*, *276*, 293–299.

Weissman, M.M., Sholomskas, D., Pottenger, M., Prusoff, B.A., & Locke, B.Z. (1977). Assessing depressive symptoms in five psychiatric populations: a validation study. *American Journal of Epidemiology*, *106*, 203–214.

Wells, A. (2008). *Metacognitive therapy for anxiety and depression*. New York, NY: The Guilford Press.

Wenzlaff, R. M., & Wegner, D. M. (2000). Thought suppression. *Annual Review of Psychology*, *51*, 59-91.

*White, R. G., McCleery, M., Gumley, A. I., & Mulholland, C. (2007). Hopelessness in schizophrenia: The impact of symptoms and beliefs about illness. *Journal of nervous and Mental Disease, 195,* 968–975.

Williams, J. M. G. (1997). *Cry of pain: Understanding suicide and self-harm.* Harmondsworth, England: Penguin.

Williams, J. M. G., Crane, C., Barnhofer, T., & Duggan, D. S. (2005). Psychology and suicidal behaviour: Elaborating the entrapment model. In K. Hawton (Ed.), *Prevention and treatment of suicidal behaviour: From science to practice* (pp. 71–89). Oxford, England: Oxford University Press.

*Willner, P., & Goldstein, R. C. (2001). Mediation of depression by perceptions of defeat and entrapment in high-stress mothers. *British Journal of Medical Psychology, 74,* 473–485.

World Health Organisation (2002). Deaths by age, sex and cause for the year 2002. Retrieved 16th October 2007, from http://www.who.int/healthinfo/global_ burden_disease/estimates_regional_2002_revised/en/index.html.

Worthington, R. L., & Whittaker, T. A. (2006). Scale development research - a content analysis and recommendations for best practices. *Counseling Psychologist, 34,* 806-838.

*Wyatt, R., & Gilbert, P. (1998). Dimensions of perfectionism: A study exploring their relationship with perceived social rank and status. *Personality and Individual Differences, 24,* 71–79.

*Yoon, H. (2003). Factors associated with family caregivers' burden and depression in Korea. *International Journal of Aging and Human Development, 57,* 291–311.

Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavia, 67,* 361-370.

APPENDIX 1

Assessment of Multiple Systematic Reviews (AMSTAR) is a measurement tool created to assess the methodological quality of systematic reviews (Shea, Grimshaw, Wells, Boers, *et al*., 2007; Shea, Hamel, Wells, Bouter, *et al*., 2009).

AMSTAR criteria:

1. Was an 'a priori' design provided?
The research question and inclusion criteria should be established before the conduct of the review.
_ Yes
_ No
_ Can't answer
_ Not applicable

2. Was there duplicate study selection and data extraction?
There should be at least two independent data extractors and a consensus procedure for disagreements should be in place.
_ Yes
_ No
_ Can't answer
_ Not applicable

3. Was a comprehensive literature search performed?
At least two electronic sources should be searched. The report must include years and databases used (e.g. Central, EMBASE, nd MEDLINE). Key words and/or MESH terms must be stated and where feasible the search strategy should be provided. All searches should be supplemented by consulting current contents, reviews, textbooks, specialized registers, or experts in the particular field of study, and by reviewing the references in the studies found.
_ Yes
_ No
_ Can't answer
_ Not applicable

4. Was the status of publication (i.e. grey literature) used as an inclusion criterion?
The authors should state that they searched for reports regardless of their publication type. The authors should state whether or not they excluded any reports (from the systematic review), based on their publication status, language etc.
_ Yes
_ No
_ Can't answer
_ Not applicable

5. Was a list of studies (included and excluded) provided?
A list of included and excluded studies should be provided.
_ Yes
_ No
_ Can't answer
_ Not applicable

6. Were the characteristics of the included studies provided?
In an aggregated form such as a table, data from the original studies should be provided on the participants, interventions and outcomes. The ranges of characteristics in all the studies analyzed e.g. age, race, sex, relevant socioeconomic data, disease status, duration, severity, or other diseases should be reported.
_ Yes
_ No
_ Can't answer
_ Not applicable

7. Was the scientific quality of the included studies assessed and documented?
'A priori' methods of assessment should be provided (e.g., for effectiveness studies if the author(s) chose to include only randomized, double-blind, placebo controlled studies, or allocation concealment as inclusion criteria); for other types of studies alternative items will be relevant.
_ Yes
_ No
_ Can't answer
_ Not applicable

8. Was the scientific quality of the included studies used appropriately in formulating conclusions?
The results of the methodological rigor and scientific quality should be considered in the analysis and the conclusions of the review, and explicitly stated in formulating recommendations.
_ Yes
_ No
_ Can't answer
_ Not applicable

9. Were the methods used to combine the findings of studies appropriate?
For the pooled results, a test should be done to ensure the studies were combinable, to assess their homogeneity (i.e. Chi-squared test for homogeneity, I2). If heterogeneity exists a random effects model should be used and/or the clinical appropriateness of combining should be taken into consideration (i.e. is it sensible to combine?).
_ Yes
_ No
_ Can't answer
_ Not applicable

10. Was the likelihood of publication bias assessed?
An assessment of publication bias should include a combination of graphical aids (e.g., funnel plot, other available tests) and/or statistical tests (e.g., Egger regression test).
_ Yes
_ No
_ Can't answer
_ Not applicable

11. Was the conflict of interest stated?
Potential sources of support should be clearly acknowledged in both the systematic review and the included studies.
_ Yes
_ No
_ Can't answer
_ Not applicable

APPENDIX 2

*P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses (PRISMA) (Moher, Liberati, Tetzlaff, Altman, *et al*., 2009) reporting criteria for systematic and meta-analytic reviews.

PRISMA criteria:

Section/topic	Item No	Checklist item
Title		
Title	1	Identify the report as a systematic review, meta-analysis, or both
Abstract		
Structured summary	2	Provide a structured summary including, as applicable, background, objectives, data sources, study eligibility criteria, participants, interventions, study appraisal and synthesis methods, results, limitations, conclusions and implications of key findings, systematic review registration number
Introduction		
Rationale	3	Describe the rationale for the review in the context of what is already known
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS)
Methods		
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (such as web address), and, if available, provide registration information including registration number
Eligibility criteria	6	Specify study characteristics (such as PICOS, length of follow-up) and report characteristics (such as years considered, language, publication status) used as criteria for eligibility, giving rationale
Information sources	7	Describe all information sources (such as databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched
Search	8	Present full electronic search strategy for at least one database, including any limits used,

		such that it could be repeated
Study selection	9	State the process for selecting studies (that is, screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis)
Data collection process	10	Describe method of data extraction from reports (such as piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators
Data items	11	List and define all variables for which data were sought (such as PICOS, funding sources) and any assumptions and simplifications made
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis
Summary measures	13	State the principal summary measures (such as risk ratio, difference in means).
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (such as I^2 statistic) for each meta-analysis
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (such as publication bias, selective reporting within studies)
Additional analyses	16	Describe methods of additional analyses (such as sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified
Results		
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram
Study characteristics	18	For each study, present characteristics for which data were extracted (such as study size, PICOS, follow-up period) and provide the citations
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome-level assessment (see item 12).
Results of individual studies	20	For all outcomes considered (benefits or harms), present for each study (a) simple summary data for each intervention group and (b) effect estimates and confidence intervals,

		ideally with a forest plot
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see item 15)
Additional analysis	23	Give results of additional analyses, if done (such as sensitivity or subgroup analyses, meta-regression) (see item 16)
Discussion		
Summary of evidence	24	Summarise the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (such as health care providers, users, and policy makers)
Limitations	25	Discuss limitations at study and outcome level (such as risk of bias), and at review level (such as incomplete retrieval of identified research, reporting bias)
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research
Funding		
Funding	27	Describe sources of funding for the systematic review and other support (such as supply of data) and role of funders for the systematic review

APPENDIX 3

Email sent by Dr Nick Troop (n.a.troop@herts.ac.uk), Chair of the School of Psychology Ethics Committee on 29/10/2012:

Hi Andy
Sorry for the delay in replying
I can confirm that it is the case that you do not need ethics to carry out a meta-analysis. However, you should check with your project supervisor whether the course requires students to go through this process as part of the learning outcomes. I don't know about the DClinPsy course specifically but I know that it is enshrined in the learning outcomes of some of the taught courses that student complete an ethics application (but this is an education issue, not an ethical one).
Hope that helps
Cheers for now
Nick

APPENDIX 4

Sensitivity analysis to decide whether to report Pearson's *r* or Spearman's rho correlation coefficients when exploring the strength of continuous moderator variables.

Problem group	Continuous moderator variables	Correlation coefficients			
		Pearson's *r*	p	Spearman's rho	p
Depression	% female	.47	.001	.55	.000
	Year of publication	.15	.299	.18	.194
	Mean age	-.26	.096	-.17	.264
Anxiety problems	% female	.51	.044	.35	.182
	Year of publication	.65	.007	.78	.000
	Mean age	.37	.156	.53	.034
PTSD	% female	.31	.328	.15	.635
	Year of publication	.40	.195	.43	.168
	Mean age	-.59	.042	-.65	.022
Suicidality	% female	.16	.629	.24	.458
	Year of publication	.59	.043	.53	.078
	Mean age	.11	.733	.04	.912

Statistically significant results are displayed in bold.

APPENDIX 5

Full results of Vevea and Woods' (2005) weight function model of publication bias, calculating population effect size estimates (*r*) under moderate and severe one- and two-tailed selection bias scenarios.

		One-tailed		Two-tailed	
Analysis	Original estimate *r*	Moderate	Severe	Moderate	Severe
Grand mean	.64	.64	.64	.64	.64
Combined defeat and entrapment					
Depression	.68	.68	.68	.68	.68
Anxiety problems	.57	.57	.57	.57	.57
PTSD	.58	**.59**	**.59**	**.59**	**.59**
Suicidality	.61	.61	.61	.61	.61
Defeat					
Depression	.74	.74	.74	.74	.74
Anxiety problems	.58	.58	.58	.58	.58
PTSD	.58	.58	.58	.58	.58
Suicidality	.57	.57	.57	.57	.57
Internal Entrapment					
Depression	.65	65	**.64**	65	**.64**
Anxiety problems	.48	**.47**	**.47**	**.47**	**.47**
PTSD	.61	.61	.61	.61	.61
External Entrapment					
Depression	.64	.64	.64	.64	.64
Anxiety problems	.53	.53	.53	.53	.53
PTSD	.54	.54	.54	.54	.54
Total Internal and External Entrapment					
Depression	.63	.63	.63	.63	.63
Anxiety problems	.68	.68	.68	.68	.68
Suicidality	.62	.62	.62	.62	.62

Note. Discrepancies between the original population effect size estimate and the estimates obtained under different hypothetical publication bias scenarios are displayed in bold. As can be seen, there are no substantive discrepancies.