Self-esteem in depression and anxiety

van Tuijl, Lonneke

Self-Esteem in Depression and Anxiety: Low, Unstable, and Discrepant?

Lonneke Aniek van Tuijl

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by

Lonneke Aniek van Tuijl

born on 20 May 1987
in Stavanger, Norway

Supervisors
Prof. P. J. de Jong
Prof. C. L. H. Bockting

Co-supervisor
Dr. K. A. Glashouwer

Assessment Committee
Prof. R. De Raedt
Prof. R. A. Schoevers
Prof. P. Spinhoven

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1. Introduction

"Few variables studied by psychologists need less of an introduction than self-esteem"
pp. 392, Zeigler-Hill & Jordan (2010)

Self-Esteem, Depression, and Anxiety

A General Introduction to Self-esteem

Self-esteem - as Zeigler-Hill and Jordan point out, is ubiquitous in both research and lay conversation rendering an introduction seem unnecessary. The importance of self-esteem is evident to the authors of many self-help books and online articles on the topic. Many well-known fiction authors have also been chipping in with their insights, some of whom are quoted throughout this introduction. There is a general consensus in research that self-esteem refers to the extent that one evaluates oneself as positive or negative, either broadly speaking (global self-esteem) or in a specific context (e.g., work, relationships; domain-specific self-esteem). However, it seems more complex than this, as how (often) it is measured appears to tap into different aspects of self-esteem. Furthermore, the emergence of various theoretical dual-processing models (see Smith & DeCoster, 2000 for a review) that have subsequently been applied to self-esteem, has spurred theories that seek to dissect this once simple, and widely-accepted construct. However, the basic understanding that self-esteem refers to the extent of positivity with regard to the self remains consistent. Pivotal across facets is the highly subjective nature of self-esteem, and levels are not necessarily reflective of a person's true value or ability (Zeigler-Hill, 2013). As such, a grade-A student may have lower self-esteem than the student who failed the exam.

Self-esteem is thought to provide two basic functions "(1) it is involved in the transfer of information between the individual and the social environment; and (2) it offers a protective function that buffers individuals from negative experiences" (pp. 13, Zeigler-Hill, 2013). Self-esteem can therefore be thought of as a filter through which external information passes through and influences how we interpret our surroundings and react (e.g., mood, behaviour). While high self-esteem may offer protection against

negative experiences, it is feasible that low self-esteem may exacerbate slightly negative or even relatively neutral situations. Indeed, self-esteem was found to moderate response to rejection (Ford & Collins, 2010), and those with low self-esteem interpreted ambiguous phrases more negatively (Tafarodi, 1998). Further, individuals with high self-esteem are argued to partake more actively in their surroundings in order to pursue further self-enhancement, while individuals with low self-esteem partake considerably less in order to protect what little self-esteem they have (Baumeister, Tice, & Hutton, 1989). As such, low self-esteem can be a self-fulfilling prophecy, and its persistence poses an influence on many aspects of daily life. Low self-esteem also appears to be persistent, with levels of self-esteem being consistent from childhood, to adolescence, to adulthood, and older age (e.g., Orth, Maes, & Schmitt, 2015).

Self-esteem in Depression & Anxiety

Given that low self-esteem moderates behaviour and interpretation of information in the environment, it is unsurprising that low self-esteem is a prominent aspect in many models of psychopathology. The present thesis focusses on depression and anxiety as they are two of the most prevalent health disorders. For depression, the DSM recognises feelings of worthlessness as one of the possible criteria in identifying an episode of major depressive disorder (MDD; APA, 2013). Indeed, low self-esteem may even lead to depression given that periods of stress often precedes depression onset (Hammen, 2005), and high self-esteem is considered to provide individuals with protection against negative experiences. Further, cognitive models of depression highlight that negative biases in information processing give rise to negative self-attributions (Beck, 2002), both specific (e.g., self-depression associations) and general (e.g., low self-esteem). Therefore, the relationship between depression and low self-esteem appears to be reciprocal, making low self-esteem and depression persistent. Depression is noted for its high lifetime prevalence rates (e.g., 16.6% for MDD; Kessler et al., 2005), incidence rates, (e.g., 1 in 20 within a year; WHO, 2012), sometimes difficult to treat nature (e.g., 34% treatment nonresponse, Van, Dekker, Peen, van Aalst, & Schoevers, 2008; although spontaneous remission rates have been reported as high as 52% within three months,

Posternak et al., 2006), and, when remittance and recovery are achieved, relapse and recurrence rates of 42% (within 20 years; Hardeveld, Spijker, Graaf, Nolen, & Beekman, 2013), and even 85% have been reported (within 15 years; Mueller et al., 1999). A meta-analysis of longitudinal studies suggests that low self-esteem is not only an antecedent of depression, but can also be a consequence of depression (Sowislo & Orth, 2013). This has led to some suggesting that residual low levels of self-esteem following an episode of depression may partly explain the high relapse rates (i.e., the scar model for low self-esteem; Zeigler-Hill, 2011).

The lifetime prevalence rates for any anxiety disorder were reported to be even higher than the rates for any depressive disorder (28.8% vs. 20.8%; Kessler et al., 2005). Anxiety disorders (AD), too, have been reported to have a persistent aetiology (e.g., 35% remission rate in ten years for social anxiety disorder), with high rates of recurrence following recovery (e.g., 34% in ten years for social anxiety disorder; Keller, 2006). Some have argued that low self-esteem should be more relevant for depression than for anxiety, as the former is related to both a decrease in positive affect and an increase in negative affect, while anxiety is mostly characterised by an increase in negative affect alone. Given that both positive and negative affect is assumed to influence self-esteem, self-esteem should be lower in depression (see Sowislo & Orth, 2013, for a review). Despite this, theories linking self-esteem and anxiety abound. For example, high levels of self-esteem have been argued to act as an anxiety buffer (e.g., terror management theory; Solomon, Greenberg, & Pyszczynski, 1991). Indeed, increasing levels of self-esteem reduced anxiety amongst participants anticipating a shock (Greenberg et al., 1992), and self-esteem was found to be positively associated with resting vagal tone (associated with the downregulation of physiological threat; Martens et al., 2010). While low self-esteem is not a defining criterion in the DSM for any anxiety disorder (APA, 2013), distortions in how people view themselves, presumably fuelled by low self-esteem, is thought to play a causal role in maintaining social anxiety disorder (Hirsch, Clark, Mathews, & Williams, 2003). Furthermore, many models of anxiety disorders highlight the role of increased self-focused attention (e.g., Ingram, 1990). Low self-esteem, therefore, seems to play a role in anxiety as well as

depression. However, research would suggest that self-esteem is more complex than how it was once considered, and research would suggest that not all facets of self-esteem are related to depression and anxiety.

Before continuing onto a description of several different facets of self-esteem, it is important to acknowledge that low self-esteem has been argued to be a transdiagnostic factor common to both depression and anxiety, and that other factors may determine which disorder manifests (de Jong, Sportel, de Hullu, & Nauta, 2012). If low self-esteem does indeed represent a transdiagnostic factor, it may explain the high rates of comorbidity often observed between and within depression and anxiety disorders (e.g., 57% had more than one anxiety and/or mood disorder, Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Indeed, a cognitive behavioural therapy targeting self-esteem by focusing on, for example, enhancing self-acceptance was found to also have beneficial effects on both depressive and anxious symptomatology (Waite, McManus, & Shafran, 2012). It seems necessary to differentiate comorbid forms of depression and anxiety from relatively pure (singular) forms of the disorders, as comorbid depression and anxiety is related to more treatment resistance, characterised by a worse prognosis (Penninx et al., 2011), and an increased likeliness to report suicidal ideation (Goodwin et al., 2001). Furthermore, the presence of both cognitive vulnerabilities for depression and anxiety led to severer symptomatology than the additive effects of each vulnerability alone (Kleiman & Riskind, 2012), suggesting that comorbidity is more than the sum of its parts. As such, comorbid depression and anxiety may represent a disorder pathway with different causal and maintaining factors than depression or anxiety alone, and therefore self-esteem may manifest and have influence in different ways. If self-esteem is a transdiagnostic factor common across depression and anxiety, thus increasing the likeliness of comorbidity, self-esteem may be a feasible target for interventions and preventions for both disorders, and prevent the development of comorbidity.

"A man cannot be comfortable without his own approval"
(Mark Twain)

The Different Sides of Self-Esteem

Self-Reported Self-Esteem

Attempts to capture self-esteem have mostly been done using self-report questionnaires. One of the most utilised questionnaires in self-esteem research, and used throughout this thesis (chapters 2 – 6), is the Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1989). Questions are asked, non-specific to a particular context, to what extent participants like themselves and feel that they are capable, sometimes in comparison to others. One issue with self-report measures, like the RSES, are biases which may influence responses given in a systematic way and irrespective of the construct that is being targeted (e.g., demand, selection and response bias; Baumeister et al., 1989). As such, questionnaires are not "pure" measures of self-esteem, and self-report measures can be considered as measuring the self that is endorsed (Zeigler-Hill & Jordan, 2010), or a "self-presentation orientation" (Baumeister et al., 1989). Low self-reported self-esteem has consistently been found in both depression and anxiety (e.g., Izgiç, Akyüz, Doğan, & Kuğu, 2004). Further, a meta-analysis of longitudinal studies suggested that self-reported self-esteem predicted symptoms of depression and anxiety, and, to a lesser extent, vice versa (Sowislo & Orth, 2013). As such, the presence of low self-reported self-esteem in depression and anxiety is fairly indisputable. Self-report measures of self-esteem assume that all evaluations concerning the self are available for introspection. However, within the last two decades, an interest within research has emerged to capture self-esteem evaluations at an earlier stage preceding the influence of biases and other processes, and potentially eluding any introspection.

Implicit Self-Esteem

Dual-processing models of information evaluation highlight a distinction between two memory systems: one which is "slow learning" and effortless to retrieve, and the other can form unique representations but

requires more effort (Smith & DeCoster, 2000). The former is implicit in that it requires no effortful processes or motivation to trigger, and is mostly a case of pattern completion (Beevers, 2005). Through repeated experience (Beevers, 2005), and the simultaneous activation of concepts (Greenwald et al., 2002), associations between constructs form in the implicit system, meaning that certain constructs and attributions (e.g., "ice" and "cold") are more strongly associated than other certain combinations (e.g., "ice" and "oily") within memory. When associations are particularly strong, activated constructs (e.g., ice") may lead to the activation of other associated attributes and constructs (e.g., 'cold'; Fazio & Olson, 2003; Greenwald et al., 2002). The explicit system, on the other hand, does require motivation and cognitive resources (e.g., executive control; Beevers, 2005), and uses "symbolically represented and intentionally accessed knowledge to guide processing" (Smith & DeCoster, 2000, p. 111). Knowledge propositions are assessed sequentially, and information triggered from the implicit system may also be considered, until a conclusion is reached that the individual believes is most likely. Dual systems of processing have been argued to have differential influences on behaviour and mood (Strack & Deutsch, 2004), and therefore, information stored about the self at the implicit level may have a different influence on mood and behaviour (e.g., more spontaneous movements) than explicit self-related processing (e.g., more controlled movements).

Dual-processing models have also been applied to self-esteem. The self is assumed to be associated with a number of concepts and attributions, and the extent of positivity of associated concepts and attributions that are triggered when the self is activated refers to *implicit self-esteem*. For the author, for example, concepts of "footballer", "researcher", "funny", "friendly", and "stubborn" are more strongly associated with the "self" than "extrovert", "tidy" or "karate". When sufficiently strong, or the self is activated for long enough, the associated concepts may be triggered too, which may in turn trigger other concepts associated with it (e.g., "football" might trigger "strong", and "researcher" might trigger "patient"). Implicit self-esteem (ISE) is assumed to be devoid of any influences associated with self-report bias, and may therefore differ from self-esteem measured by self-report questionnaires (i.e., *explicit self-esteem*). Explicit self-esteem (ESE) refers to

self-related evaluations that are the result of considering knowledge propositions for the extent of their subjective truth value. Trait ESE refers to the overall tendency to derive at positive or negative self-evaluations. For example, upon seeing yourself in the mirror, mostly negative associated concepts may be activated (i.e., ISE). You might be motivated to overrule this (e.g., to correct for negative moods that have arisen as a consequence) and consider reappraising the situation by recalling relevant memories (e.g., remembering that you have looked a lot worse in similar situations) or create new rules (e.g., you are staying at home today, and there is no need to put a lot of effort into your appearance; i.e., ESE). Therefore, ISE and ESE do not have to be congruent (i.e., equally high or low), and indeed correlations between the two are often small (Bosson, Swann, & Pennebaker, 2000). ISE and ESE have also been shown to manifest in differential behaviours, with ESE being related to more purposeful and controlled behaviours (e.g., hand gestures) and ISE related to more spontaneous behaviours (e.g., nervous mouth movements; Rudolph, Schröder-Abé, Riketta, & Schütz, 2010).

As implicit self-esteem refers to the self-evaluation before additional processes and biases moderate the outcome, the measurement of ISE is not straightforward. Various attempts have been made at capturing ISE, and the most popular of these show low convergent validity (Bosson et al., 2000). The implicit association test (IAT; Greenwald, McGhee, & Schwartz, 1998) is arguably one of the most utilised measures of implicit attitudes and evaluations. IAT scores are derived from reaction times of a word-sorting task, based on the premise that concepts that are more strongly associated would result in shorter reaction times when sharing the same sorting key, than when words less strongly associated share the same key. The IAT has been found to predict implicit racial bias, gender/sexual orientation, consumer preferences, alcohol and drug use (Greenwald, Poehlman, Uhlmann, & Banaji, 2009). With regards to self-esteem, the IAT has been found to predict a buffering effect to a failure condition in terms of self-reported future task aspiration and task importance (Greenwald & Farnham, 2000), spontaneous self-confident behaviours (e.g., nervous mouth movements; Rudolph et al., 2010) and changes in mood following negative feedback (Meagher & Aidman, 2004; but see Buhrmester, Blanton, & Swann,

2011). The self-esteem IAT has consistently been found to be the most reliable implicit measure of self-esteem (Bosson et al., 2000; Krause, Back, Egloff, & Schmukle, 2011), and was therefore used throughout this thesis (chapters 2, 3, & 6).

Research concerning the presence of low self-esteem at the implicit level in depression and anxiety are a lot less consistent than research concerning ESE. Some studies have observed lower ISE in (symptoms of) a depressive disorder (e.g., Franck, De Raedt, & De Houwer, 2007; Phillips, Hine, & Thorsteinsson, 2010; Risch et al., 2010) or anxiety disorder (de Jong, 2002; de Jong et al., 2012; Glashouwer, Vroling, de Jong, Lange, & de Keijser, 2013; Tanner, Stopa, & De Houwer, 2006). Further Franck, De Raedt, Dereu, and Van den Abbeele (2007) found that low ISE was only present in MDD without suicidal ideation, not in MDD with suicidal ideation. However, many have failed to observe lower ISE in depression (e.g., Franck, De Raedt, & De Houwer, 2007; Lemmens et al., 2014; De Raedt, Schacht, Franck, & De Houwer, 2006). Conflicting findings may be the result of small sample sizes (e.g., n = 15, De Raedt et al., 2006), given that sampling from a clinical population is often difficult. This limits the power to detect smaller, but possible still relevant, effects. Poorly defined groups may also explain some of the findings, as the presence of a comorbid MDD and AD is often not controlled for (e.g., Risch et al., 2010). Further, differences in how ISE was measured may also help explain the inconsistent findings.

Implicit Self-esteem Scar

Given the high rates of relapse and recurrence in both depression and anxiety, much research has been conducted on identifying potential "scars" that remain following symptomatology that was not there before the onset of symptoms and which increases the chance for relapse (Burcusa & Iacono, 2007; Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Particularly low ISE may be a likely scar following depression, given its robust nature and the lack of possible controllability to alter dysfunctional associations. Based on the assumption that implicit associations arise following repetitive, consistent explicit associations that require less cognitive resources and become more automatic with time (Beevers, 2005), low ISE would

presumably only increase after prolonged periods of increased ESE. As such, while ESE may have improved following a period of depression or anxiety, low ISE may continue. If cognitive resources are limited (e.g., during periods of stress), it may not be possible to effectively override dysfunctional implicit appraisals concerning the self, thus leaving an individual vulnerable to develop other symptoms of depression and anxiety. Somewhat consistent with this, one study found that implicit public-speaking associations following exposure therapy predicted relapse in those with a phobia of public speaking (Vasey, Harbaugh, Buffington, Jones, & Fazio, 2012). However, both Risch et al., (2010; n = 33 remitted depressed) and Franck et al., (2008; n = 35 former depressed) did not observe lower ISE in remitted depressed individuals in comparison to never depressed comparison group, and further evidence for anxiety disorders could not be found. Potentially the small sample sizes limited the power to detect a smaller effect, but it may also be crucial to differentiate between those who have remitted (i.e., a recent reduction in symptoms) and those who have recovered (i.e., those with a longer period of reduced symptoms). If ESE continues to be improved, it is likely to slowly improve ISE. In both studies (Franck, De Raedt, et al., 2008; Risch et al., 2010), it is unclear how long the former depressed patients had been symptom free.

Self-Esteem Discrepancy

Discrepant self-esteem refers to the extent that implicit and explicit self-esteem differ. Briñol, Petty and Wheeler (2006) have suggested that the extent that they differ is related to increased implicit self-doubt (but not explicit self-doubt), which subsequently leads to negative and dysfunctional consequences, and may motivate behaviour to dissolve the discrepancy (Zeigler-Hill, 2011). Further, it was found that those with a large discrepancy between ISE and ESE where more persuaded by strong self-related messages that were provided supposedly about the person (Briñol et al., 2006). Therefore, it was argued that those with large self-esteem discrepancies may be more susceptible to external information as they were presumed to invest more cognitive resources into information that may resolve the discrepancy. The combination of high ESE and low ISE ("discrepant high self-esteem" or "fragile self-esteem") has been linked to narcissistic behaviour (Jordan,

Spencer, Zanna, Hoshino-Browne, & Correll, 2003). "Damaged self-esteem" (or "discrepant low") refers to the combination of high ISE and low ESE, and increased discrepancy in this specific combination has been linked to more depressive symptoms in adults (Creemers, Scholte, Engels, Prinstein, & Wiers, 2012) and social anxiety disorder in adolescents (Schreiber, Bohn, Aderka, Stangier, & Steil, 2012). Studies looking at self-esteem discrepancy in adult clinical populations are relatively few, and it may be that specific combinations of ESE and ISE are prevalent in anxiety and depression, particularly if discrepant self-esteem gives rise to self-doubt.

Self-Esteem Instability

The introduction till now has discussed self-esteem as a trait. However, evidence for both trait-levels and state-levels of self-esteem exist. While self-esteem appears to be relatively consistent across life (Orth et al., 2015), the fluctuations in self-esteem in response to, for example, daily stress or positive occurrences (Greenier et al., 1999), clearly supports a state-like dimension to self-esteem. Specifically, state self-esteem has been described as "the baseline self-view from which such fluctuations emerge" (Kernis, Grannemann, & Mathis, 1991, p. 1013). These state-like "fluctuations" brings us to the last self-esteem concept addressed in this thesis: self-esteem stability (SE-S). Based on the theory that depression and anxiety are characterised by highly reactive negative self-schemas which are overly sensitive to stress, sad mood, and other negative circumstances (e.g., diathesis-stress models, Zuckerman, 1999), it has been argued that the average level of ESE is not necessarily related to psychopathology, but rather the extent and frequency of fluctuations. Indeed, self-esteem was found to react to sad mood (Clasen, Fisher, & Beevers, 2015). Several studies have highlighted that depressive symptomatology is characterised by self-esteem instability (as quantified by the standard deviation across multiple measures of ESE) rather than a single measure of ESE (e.g., Roberts & Monroe, 1992). Others have found that depressive symptoms were related to the combination of low ESE and low SE-S (e.g., de Man, Gutiérrez, & Sterk, 2001), while others failed to find that SE-S outperformed a single measure of ESE (Roberts, Kassel, & Gotlib, 1995). Research focusing on clinical samples of anxiety and depression are considerably less. Low SE-S was related to social

anxiety disorder, but once accounting for ESE, the relationship disappeared (Farmer & Kashdan, 2014). Low SE-S was also observed in currently and formerly depressed samples, in comparison to a never-depressed sample (Franck & De Raedt, 2007). Further, ESE and SE-S are often reported to correlate, with low levels of ESE related to low SE-S (for a meta-analysis of studies see Okada, 2010). This is in keeping with the findings that those with high ESE are less affected by negative feedback (Ford & Collins, 2010). As such, although related, the combination of low ESE and low SE-S, particularly, may play a role in psychopathology. Based on the limited previous clinical studies, low SE-S might be a feature of depression, specifically, and not anxiety. Further, SE-S refers to fluctuations in ESE, but it remains unknown as to whether ISE is also susceptible to systematic fluctuations. Studies have looked at the influence of sad mood on ISE in remitted depression, compared to a never depressed comparison group with conflicting results (Franck, De Raedt, et al., 2008; Gemar, Segal, Sagrati, & Kennedy, 2001), but whether sad mood influences ISE independent of clinical status remains unknown. If ISE is indeed influenced by mood, it may highlight the possibility of an unstable ISE as a product of highly reactive mood.

Self-Depressed Associations

While ISE refers to the general degree of positivity and negativity of self-related associations, the self may also be related to other, more specific attributes. Disorder-specific implicit self-associated concepts have been observed both in anxiety and depression (Glashouwer & de Jong, 2010). Specifically, implicit self-depressed associations were found to be stronger in those with a depressive disorder than a never depressed or anxiety disorder comparison group, and individuals with an anxiety disorder. Likewise, implicit self-anxious associations were found to be stronger in those with an anxiety disorder than in individuals with a depressive disorder. Those who had comorbid anxiety and depressive disorders were found to have both strong implicit self-depressed associations and self-anxious associations, and these associations were found to be stronger than those with an anxiety or depressive disorder only. Further, stronger implicit self-depressed and self-anxious associations were related to decreased chance of remission in depression and anxiety, respectively (Glashouwer, de Jong, & Penninx, 2012).

Unlike with ISE (Franck, De Raedt, et al., 2008; Risch et al., 2010), there is some evidence that implicit self-associations remain strong in remission. Glashouwer, de Jong, and Penninx (2011) found that implicit self-anxious associations predicted relapse in those who were currently remitted from anxiety. In depression, Glashouwer and de Jong (2010) observed that while remitted depressed individuals had weaker self-depressed associations than those who were currently depressed, associations were stronger than in the never depressed group. Further, another study found that the number of previous MDD episodes, and the duration of depressive symptoms in the previous two years, were both related to stronger implicit self-depressed associations in remitted and recovered depressed individuals (Elgersma, Glashouwer, Bockting, Penninx, & de Jong, 2013). If implicit self-depressed associations represent a scar following a depressive episode, it should increase vulnerability for relapse and recurrence (Burcusa & Iacono, 2007). Till now, no research has been conducted to see if this is indeed the case.

"The man who does not value himself,
cannot value anything or anyone"
(Ayn Rand)

The Present Thesis

Overall Aims

Inarguably, much research has been conducted on self-esteem. This may be because low self-esteem is something that most have experienced at some point. Even within the context of depression and anxiety, much self-esteem research has been done. However, the understanding that self-esteem is more complex than once thought, and that self-report measures may only capture one side of the construct, call for research to further investigate the presence of these different self-esteem facets in depression and anxiety. Evident from the overview of several self-esteem facets, how (often) you measure self-esteem appears to be related to different outcomes. For example, while self-esteem at one given moment may be particularly

high, it may be highly reactive to changes in moods and external situations (i.e., high explicit self-esteem, low self-esteem stability). This may highlight that a person is not consistently vulnerable to symptoms of depression or anxiety, but when that person is going through a disruptive period (e.g., during a break-up), the risk for depression or anxiety becomes real. As such, it seems poignant to look at the different facets of self-esteem in the aetiology of depression and anxiety, as each facet may be uniquely relevant. Presumably each unique association would require a different, specific intervention. Explicit self-esteem, implicit self-esteem, self-esteem discrepancy, self-esteem instability, and self-depressed associations have all been looked at in previous depression and anxiety research. Despite this, several important questions remain, and the present thesis aims to address a number of them. Below, a chapter by chapter overview is provided, highlighting the main research question, brief justification/background, and how the question is addressed in the chapter. Addressing these questions will further existing knowledge of the role of self-esteem in depression and anxiety.

Chapter Overview

Does self-esteem in healthy adolescents relate to symptoms of depression and anxiety years later? Adolescence marks a period of decreasing self-esteem as identity confusion increases, the positivity bias present in childhood decreases, and stress starts to increase. First onset of anxiety disorders is often before adulthood and those with depressive disorders during adolescence and childhood often develop a highly recurrent pathology. The decrease in self-esteem may explain why symptoms of depression and anxiety manifest during this period. In chapter two, we look at whether ISE and ESE (and the discrepancy herein) in relatively healthy adolescents are related to symptoms of social anxiety and depression two years later. This chapter also looks at whether there is evidence of a self-esteem scarring model by testing whether symptoms of depression and social anxiety were related to subsequent levels of ISE and ESE.

Is self-esteem lower in clinical adult groups with depression and/or anxiety than in a comparison group with no history of a depression or anxiety disorder? This question has been addressed in previous research using small clinical groups, and often, broadly defined inclusion criteria, which may explain the inconsistent findings. To address these methodological shortcomings, chapter three uses data collected in the Netherlands Study of Depression and Anxiety (NESDA), a large ongoing longitudinal study aiming to capture and understand predictors of depression and anxiety aetiology. Specifically, ISE and ESE will be compared between a comparison group with no history of a depression or anxiety disorder and clinical groups of anxiety, depression, and comorbid anxiety and depression differentiated by etiological phase (current, remitted, and recovered). By differentiating between these phases, it is also possible to give some indication as to whether low ESE or ISE persist into recovery. A novel approach to analysing self-esteem discrepancy is also employed in this chapter to tackle several shortcomings of previous approaches.

Is self-esteem stability lower in clinical adult groups of depression and anxiety than in a comparison group with no history of a depression or anxiety disorder? Previous research has indicated that, in analogue samples, heightened fluctuations in self-esteem increases vulnerability for symptoms of depression and anxiety. The few studies that have adopted clinical samples have used small sample sizes, and appear to derive at different conclusions. In chapter four, it is examined whether the clinical groups established in the previous chapter are characterised by low self-esteem stability (i.e., regular fluctuations in self-esteem). The inclusion of recovered depression and anxiety will reveal whether low levels of self-esteem stability persist into recovery.

Does the level of self-depressed associations predict likeliness of recurrence in remitted and recovered depressed individuals? Previous studies have highlighted that self-depressed associations are still relatively strong following an episode of depression. A scar in depression, by definition, should increase vulnerability for relapse and recurrence. Chapter five continues on previous research by predicting relapse and recurrence using level of self-depressed associations in recovered and remitted depressed

individuals. Specifically, a longitudinal analysis is conducted to see whether levels of implicit and explicit self-depressed associations predicts (time to) relapse up to six years later.

Does implicit self-esteem decrease in response to increased sad mood? The penultimate chapter aims to explore whether sad mood influences ISE. While there is some support for the construct of self-esteem stability (i.e., fluctuations in ESE), little research has looked at the possibility of ISE to fluctuate. Given that context can influence the strength of associations, it is assumed that during periods of sad mood, negative content may be more readily available, and as such, ISE would be lower. In this experimental study, ISE in university students is compared between those who received a sad-mood induction, and those who did not.

Final Chapter

This thesis ends with a general discussion. The findings for each research question are reiterated, thereby stipulating the findings of the thesis. Each facet of self-esteem is discussed in light of these findings and previous findings, with suggestions as to which facets may be most fruitful to pursue in the quest for effective anxiety and depression (relapse) prevention and intervention.

2. Implicit and Explicit Self-esteem and Symptoms of Depression and Social Anxiety: A Longitudinal Study in Adolescents

Abstract

A negative self-view is a prominent factor in most cognitive vulnerability models of depression and anxiety. Recently, there has been increased attention to differentiate between the implicit (automatic) and the explicit (reflective) processing of self-related evaluations. This longitudinal study aimed to test the association between implicit and explicit self-esteem and symptoms of adolescent depression and social anxiety disorder. Two complementary models were tested: the vulnerability model and the scarring effect model. Participants were 1641 first and second year pupils of secondary schools in the Netherlands. The Rosenberg Self-Esteem Scale, self-esteem Implicit Association Test and Revised Child Anxiety and Depression Scale were completed to measure explicit self-esteem, implicit self-esteem and symptoms of social anxiety disorder (SAD) and major depressive disorder (MDD), respectively, at baseline and two-year follow-up. Explicit self-esteem at baseline was associated with symptoms of MDD and SAD at follow-up. Symptomatology at baseline was not associated with explicit self-esteem at follow-up. Implicit self-esteem was not associated with symptoms of MDD or SAD in either direction. We relied on self-report measures of MDD and SAD symptomatology. Also, findings are based on a non-clinical sample. Our findings support the vulnerability model, and not the scarring effect model. The implications of these findings suggest support of an explicit self-esteem intervention to prevent increases in MDD and SAD symptomatology in non-clinical adolescents.

Cognitive vulnerability models of depression and anxiety attempt to identify risk factors that increase the likelihood of disorder onset and maintenance. Common across models is the salient role of a negative self-view construct (e.g., self-esteem, self-concept) that stems from the negative inferential style that is characteristic of both depression and anxiety (Sutton et al., 2011). Self-esteem is considered as the baseline self-view from which fluctuations may occur in a given context (Kernis, Grannemann, & Barclay, 1989). During early adolescence, self-views become more negative as the positivity bias that is present during childhood decreases (Baumeister & Tice, 1986), and identity confusion peaks (Erikson, 1968). Therefore, decreased levels of self-esteem during the identity confusion phase may increase vulnerability for depression and anxiety in adolescents. Two prominent mental health disorders during adolescence are social anxiety disorder (SAD) and major depressive disorder (MDD; Wittchen, Nelson, & Lachner, 1998), and lifetime prevalence rates by the age of 18 are 5.5% and 11.2%, respectively (Merikangas et al., 2010). Adolescent SAD and MDD increase the risk for more severe depression and anxiety symptoms in adulthood, as well as suicidal behaviour (Lim et al., 2012; Pine, Cohen, Gurley, Brook, & Ma, 1998; Wittchen, Stein, & Kessler, 1999; Zisook et al., 2007). Further knowledge into how symptoms of SAD and MDD develop during adolescence and particularly the role of self-esteem herein may highlight possible areas for prevention.

Previous cross-sectional studies have consistently observed lower self-esteem in those with relatively higher levels of depression and anxiety symptoms, both in adults (e.g., Ginsburg, Greca, & Silverman, 1998; Hammond & Romney, 1995) and adolescents (e.g., de Jong et al., 2012; Moksnes, Moljord, Espnes, & Byrne, 2010). Longitudinal studies are more apt for testing vulnerability models. A recent meta-analysis of 95 longitudinal studies (77 on depression, 18 on anxiety) suggests that low self-esteem was predictive of both symptoms of depression and anxiety (Sowislo & Orth, 2013). SAD symptoms, specifically, have also been found to be predicted by self-esteem in adulthood (Acarturk et al., 2009). Longitudinal studies looking at SAD and self-esteem in adolescents could not be found. Although Sowislo and Orth (2013) found age not to be a moderator of the effect size in the relationship between self-esteem and symptoms of depression, it is also

important to acknowledge that findings observed in adulthood and late adolescence might not be observed in a younger adolescent sample, particularly since several studies argue that adulthood depression and anxiety differ aetiologically and neurologically from adolescent and child depression and anxiety (e.g., Kaufman, Martin, King, & Charney, 2001).

While the vulnerability model in the current context suggests that relative decreases in self-esteem increase risk for later symptoms of psychopathology, a longitudinal relationship could also, theoretically, occur in the opposite direction. The "scar hypothesis" refers to residual negative cognitions following a depressive episode (Lewinsohn et al., 1981). In the current context, a model based on the scar hypothesis would suggest that self-esteem is lowered as a consequence of depression and anxiety (Zeigler-Hill, 2011). In the meta-analysis by Sowislo and Orth (2013), a significant reciprocal relationship was observed where prediction of self-esteem by depression was weaker than the prediction of depression by self-esteem. Also for anxiety there was a significant reciprocal relationship with self-esteem, yet in this case with both unidirectional relationships being equally strong. As such, there appears to be some support for both the vulnerability and the scar model. If symptoms of SAD and MDD in adolescence affect subsequent self-esteem, it may highlight the need for interventions that target residual negative self-related thoughts following increases in symptomatology.

While a vulnerability and scar model involving self-esteem has been extensively researched, it is important to note that the majority of studies utilise self-report measures of self-esteem like the Rosenberg's Self-esteem Scale (Rosenberg, 1989). Self-report measures of self-esteem rely on a person's explicit reflection of their self-worth. Based on current dual processing models (e.g., Beevers, 2005), when cognitive resources are limited and purposeful reflection is not possible, automatic heuristic based processes are adopted. Implicit self-esteem refers to the automatically elicited self-evaluation in a given context that guides and influences behaviour. Usually, one is aware of the output (e.g., a 'gut' feeling) while not being aware of the trigger. Explicit self-esteem is said to be adopted when there is motivation, time and cognitive resources to do so. Correlations between implicit and explicit self-esteem tend to be moderate at best

(Bosson et al., 2000), suggesting that for the most part, explicit and implicit self-esteem are two independent concepts. The distinction between explicit and implicit self-esteem suggests the possibility of differential roles in the aetiology of MDD and SAD. While a person with low explicit self-esteem may be able to actively counter negative self-related thoughts by considering positive propositions, implicit self-esteem suggests that the process itself is non-intentional and therefore harder to change. Therefore, differential roles of implicit and explicit self-esteem in the aetiology of MDD and SA would suggest the need for differential interventions.

As implicit self-esteem is a relatively new concept, only few longitudinal studies have been conducted to date. The available studies appear to support the inclusion of implicit self-esteem in the depression vulnerability model. Franck, De Raedt and De Houwer (2007) found that while at a cross-sectional design there were no significant differences between former, current, and never depressed adults, a six-month follow-up of depression scores in the former and never depressed adults indicated a significant prediction by implicit, and not explicit, self-esteem. Steinberg, Karpinski and Alloy (2007) conducted a four-month longitudinal study with undergraduate psychology students in which (marginally significant) results indicated that implicit self-esteem moderated the association between depressive symptoms and negative life events amongst those with high cognitive vulnerability (i.e. displaying negative cognitive style and high dysfunctional attitudes), while explicit self-esteem failed to contribute to the prediction. With regard to the inclusion of implicit self-esteem in a scar model, the available studies are not particularly conclusive. Franck, De Raedt, and De Houwer, (2007) found that former depressed and never depressed adults did not differ in implicit self-esteem, suggesting that the scar model does not hold true. However, Risch et al. (2010) found that previously depressed individuals with more than two depressive episodes had significantly lower implicit self-esteem than remitted individuals with less than three episodes. This also partially supports the notion of a reciprocal relationship between self-esteem and depression. If low self-esteem led to high levels of depression, which in turn lowered self-esteem, this would imply a downward spiral of deteriorating symptoms and self-esteem. It is therefore important to note that a vulnerability model and a scar model

could occur simultaneously. The main aim of the current study is to test the vulnerability model and the scarring effect of implicit and explicit self-esteem for adolescent MDD and SAD symptomatology. As the scar hypothesis refers to the effect of a depressive episode specifically, we use the term scarring effect to refer to the effect of symptomatology on self-esteem.

Although previous studies support that implicit and explicit self-esteem are two distinct constructs (e.g., Bosson et al., 2000), specific patterns of the two may also be predictors of SAD and MDD symptomatology. Discrepant self-esteem refers to a negative relationship between implicit and explicit self-esteem. Specifically, fragile (also known as "defensive" or "discrepant high") self-esteem refers to high explicit coupled with low implicit self-esteem and has been linked to narcissistic behaviour (Jordan et al., 2003). Damaged (or "discrepant low") self-esteem, on the other hand, refers to high implicit coupled with low explicit self-esteem and has been linked to more depressive symptoms in adults (Creemers et al., 2012). Following a social threat activation, damaged self-esteem has been linked to SAD in adolescents (aged 14 – 20; Schreiber et al., 2012). Discrepant self-esteem in adolescent MDD and SAD (before a social threat activation) has not been researched longitudinally. In order to look at whether discrepant self-esteem adds to the predictability of SAD and MDD symptoms in adolescents, the interaction between implicit and explicit self-esteem is included in the vulnerability model (as done previously in Schröder-Abé, Rudolph, & Schütz, 2007).

There are two broad research aims in the present study of adolescent SAD and MDD symptomatology. First, we aim to test the vulnerability model with implicit and explicit self-esteem as two distinct constructs being potential predictors of subsequent symptoms of MDD and SAD in a large longitudinal cohort study in adolescents. To test the potential added prediction of damaged and/or fragile self-esteem, the interaction between implicit and explicit self-esteem is also included in the vulnerability model. Second, we aim to test the scarring effect with both SAD and MDD symptomatology being predictive of explicit and implicit self-esteem. Further, as gender differences have not only been consistently observed in SAD and MDD (Merikangas et al., 2010), but also in self-esteem (Kling, Hyde, Showers, & Buswell, 1999), we will include gender in the analysis of the

vulnerability model and scarring effect in order to control for the possibility of gender differences in the aetiology of MDD and SAD.

Method

Participants

Adolescents in the first and second year of secondary school in the Northern part of the Netherlands (n = 5318) were invited to participate in a large longitudinal study: Prevention of Adolescent Social and Test Anxiety (PASTA; www.projectpasta.nl). Invitations were sent out to adolescents and their parents via the school. Consent from participants and one of their parents/guardians was obtained from 1811 (34%) of those invited. 97.1% were of Dutch nationality, and 68% came from a rural area as defined by Statistics Netherlands (Reijneveld et al., 2010). A number of participants (n = 170) were excluded from the analysis in the current study as they had received some form of intervention between baseline and the two-year follow-up as part of another study. The remaining 1641 participants had a baseline mean age of 13.14 (SD = .75, range 10-16), and 767 were male (46.7%). Over one third of the participants (n = 576; 35%) were not available at the two-year follow-up. PASTA received ethical approval by the Medical Ethics Committee of the University Medical Centre Groningen.

Measures

A number of measures were adopted in the PASTA study, however the ones described below were specific to the statistical analyses in the current study. It should be noted that a description of the measures used for the multiple imputation, but not in any further analyses, can be found in their respective articles (see subheading Multiple Imputation).

Implicit Association Test (IAT; Greenwald et al., 1998). A self-esteem version of the IAT was adopted as a measure of implicit self-esteem. There were two target concepts, 'self' (I, self, my, own, myself, personally) and 'other' (they, their, you, other, themselves, others), and two attribute concepts, 'positive' (good, smart, stable, beloved, active, valuable) and 'negative' (bad, stupid, unstable, failure, passive, worthless; translated from Dutch). The stimuli were used in a previous study involving adolescents (Bos, Huijding, Muris, Vogel, & Biesheuvel, 2010), though the exact meaning and content of the stimuli are not crucial as the IAT effect has been shown to be

driven mainly by the content of the labels (De Houwer, 2001). The category labels in the present study were not unlike the labels used in the original self-esteem IAT (i.e., me vs. not-me, pleasant vs. unpleasant; Greenwald & Farnham, 2000). Following three practise rounds, the first test round required that participants sorted positive- and self- related words on one key and, other- and negative- related words on the other key. Following further two practice rounds, the participants categorised other- and positive- related words with the same key and self- and negative- related words on another shared key (see Table 2.1 for overview). Reaction times and error rates were recorded. The premise for the IAT is that sorting is easier and thus faster for a person when the target and the attitude that are strongly associated share the same key (i.e., congruent) than when a relatively less associated target and attributed share the same key (i.e., incongruent). Therefore, slower reaction time in blocks 6 and 7, relative to blocks 3 and 4, is indicative of higher implicit self-esteem. Spearman-Brown corrected correlation between test halves was .74 and .77 at baseline and follow-up, respectively (test halves based on trials 1, 2, 5, 6, 9, 10, etc., and 3, 4, 7, 8, 11, 12, etc.; based on original scores as individual trial times were not imputed).

The Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1989). Explicit self-esteem was measured using a Dutch adaptation of the RSES, as used by Mayer, Muris, Meesters, and Zimmermann-van Beuningen (2009) in a study involving adolescents. Fifteen items based on the original RSES (Rosenberg, 1989) are rated on a five-point scale from 0 (completely not true) to 4 (completely true). Scores could range from zero to sixty. This version of RSES showed good reliability at both baseline (Cronbach's α = .91) and follow-up (Cronbach's α = .91; based on original data as individual scale items were not imputed). Increases in RSES scores are indicative of higher explicit self-esteem.

The Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000). The RCADS is a 47-item self-report measure that measures symptoms of social anxiety disorder (SAD), major depressive disorder (MDD), general anxiety disorder, obsessive compulsive disorder, separation anxiety and panic disorder. Answers are given on a four-point scale from 0 (Never) to 3 (Always). Only MDD (10 items) and SAD (9 items) scores were used in the present study. Reliability for

the SAD and MDD subscales was good at both baseline (Cronbach's α = .84 and .79, respectively) and follow-up (Cronbach's α = .84 and .80, respectively; based on original data as individual scale items were not imputed). Increases in SAD and MDD scores are indicative of increased SAD and MDD symptomatology.

Table 2.1
Arrangement of the Different IAT Blocks

Block	Left Label(s)	Right Label(s)	No. of Trials
1. Practice	Positive	Negative	10
2. Practice	Me	Other	10
3. Practice	Positive/Me	Negative/Other	20
4. Test	Positive/Me	Negative/Other	40
5. Practice	Other	Me	10
6. Practice	Positive/Other	Negative/Me	20
7. Test	Positive/Other	Negative/Me	40

Note. IAT = Implicit Association Test.

Procedure

The PASTA study took place in 25 schools in the Northern region of the Netherlands. There were two waves; baseline and a two-year follow-up. Participants took part at school, in groups no bigger than fifteen, with 2-3 researchers present. Participants always started with the IAT, as recommended by Bosson et al. (2000). All questionnaires were completed on a laptop, and as such missing information at an item level was very low (n = 4).

Data Analyses

Multiple Imputation. At the two-year follow-up there was a unit non-response of 576 (35%), one case that did not complete the RSES measure and three who were not able to complete the IAT measure at baseline due to a technical fault. To optimally correct for missing data, multiple imputation (40 iterations; Bodner, 2008) was conducted using the following baseline and follow-up scores: RCADS (all subscales and total), RSES (total), the Behavioural Inhibition/Behavioural Activation System Scales (Carver & White, 1994), the Adult Temperament Questionnaire (Rothbart, Ahadi, & Evans, 2000), Spielberger Test Anxiety Inventory (Spielberger et al., 1980), Brief Fear of Negative Evaluation-II (Carleton, McCreary, Norton, &

Asmundson, 2006), and a created scale that involved explicit ratings of the attribute concepts used in the IAT. Presented results are therefore based on averages across the 40 imputed datasets.

Data Reduction. IAT scores were computed according to the algorithm proposed by Greenwald, Nosek and Banaji (2003), which recently has shown to perform best in the current measurement setting (Glashouwer, Smulders, de Jong, Roefs, & Wiers, 2013). We report the D4-measure. First, reaction times that were above 10,000 ms were discarded, and error trials were replaced with the mean for that block plus an added penalty of 600 ms. Next, mean reaction times (RTs) of block 3 were subtracted from those of block 6 RTs, and RTs of block 4 were subtracted from RTs of block 7 (see Table 2.1). The mean of these two differences was then divided by the standard deviation of all responses (i.e., block 3, 4, 6 and 7) in order to control for individual variation. IAT scores were excluded (and subsequently replace with values from the multiple imputation) when there was a high percentage of errors (> 22%), a large number of trials with reaction times faster than 300ms (> 10%) or trials with reaction times longer than 10000ms (≥ 1%). One hundred and twenty-four cases (97 at baseline, 27 at follow-up) were excluded in total based on these criteria. Higher IAT scores are indicative of higher implicit self-esteem.

In order to increase the interpretability of the results, RSES, IAT and baseline psychopathology scores were mean centred before being entered into the regression models (Aiken & West, 1991). Interaction scores with gender were computed by multiplying the mean-centred raw score with 1 for females, and 0 for males. Interaction scores between RSES and IAT were computed by multiplying the two mean-centred raw scores.

Statistical Analyses

Following data screening and bivariate correlation analysis, two hierarchical regression analyses were conducted to test the vulnerability model (i.e., that self-esteem predicts subsequent SAD and/or MDD scores) with follow-up SAD score and MDD score as dependent variables. Baseline psychopathology score (i.e., SAD or MDD; square-rooted to correct positive skewness) was entered first, followed by gender at step two. IAT and RSES scores were simultaneously entered at the third step, and the interaction

between RSES and IAT was entered at the fourth. Gender interactions with the hereto included variables were entered at the fifth and final step.

Hierarchical regression analysis was also adopted to test the scarring effect with follow-up RSES score and IAT score as dependent variables. Baseline self-esteem score (i.e., IAT or RSES) was entered at step one, followed by gender at step two. Baseline SAD and MDD (square-rooted to correct positive skewness) were simultaneously entered at step three. At the fourth and final step, gender interactions with baseline self-esteem, SAD and MDD were entered.

Residual analyses were conducted after every regression analysis and standardised residuals falling outside of ± 3.29 (Field, 2005) were considered extreme and omitted before re-running the analysis. Including omitted extreme residuals changed some of the conclusions, and in order to present the best fit they were left out of the final analysis. Further, it should be noted that as there were 40 datasets with imputations, the presented results are based on the pooled or manually averaged results of all 40 datasets.

Results

Missing Data Analysis

Four t-tests were conducted in order to check for differences between those who were present at follow-up and those who dropped out of the study (n = 576). In order to limit the number of t-tests conducted, we checked for differences in variables relevant to the analysis (i.e., baseline IAT, RSES, MDD and SAD). A Bonferroni correction was adopted to account for conducting four t-tests, and therefore we used a significance level of .012. Results can be seen in Table 2.2. Although the drop-outs had lower explicit self-esteem and higher MDD symptomatology to a significant degree, the effect sizes were small and therefore it was deemed appropriate to use multiple imputation.

Table 2.2
Independent Samples T-tests Testing Randomness of Missing Data at Follow-Up

Baseline Measures	Completers Mean (SD)	Drop-outs Mean (SD)	t	df	SE of Diff	Cohen's d
RSES	49.60 (10.11)	47.24 (10.66)	4.23*	1639	.53	.23
IAT[a]	.69 (.32)	.66 (.36)	1.43	997.69	.02	.09
SAD[ab]	7.19 (4.16)	7.86 (4.80)	2.28	1089.02	.05	.15
MDD[b]	4.86 (3.50)	5.60 (3.77)	3.96*	1639	.04	.21

Note. SAD: Revised Child Anxiety and Depression Scale – social anxiety disorder; MDD: Revised Child Anxiety and Depression Scale – major depressive disorder; RSES: Rosenberg Self-esteem Scale; IAT: Implicit Association Test.
[a] Levene's Test significant, equal variances not assumed
[b] Square-rooted to correct significant skewness
* $p < .012$ (Bonferonni correction for four t-tests)

Descriptives

Means and standard deviations of the measures at baseline and follow-up are presented in Table 2.3. At baseline and follow-up, respectively, 20% and 5.8% scored above the cut-off for SAD (>10), and 5.8% and 2.9% scored above the cut-off for MDD (>11; cut-offs based on Chorpita et al., 2000). Relationships between baseline and follow-up measures were tested with Pearson's correlation coefficient and are presented in Table 2.4.

Table 2.3
Means and Standard Deviations of Baseline and Follow-Up Measures

	Baseline		Follow-Up	
	M	SD	Pooled M	Averaged SD
RCADS Total	23.94	15.28	20.27	10.98
SAD	7.42	4.41	6.80	3.59
MDD	5.12	3.61	4.47	3.08
RSES	48.77	10.36	51.12	8.81
IAT	.67	.34	.72	.35
RSES x IAT interaction	.05	1.01	.05	.99

Note. RCADS Total: Revised Child Anxiety and Depression Scale – total score; SAD: Revised Child Anxiety and Depression Scale – social anxiety disorder; MDD: Revised Child Anxiety and Depression Scale – major depressive disorder; RSES: Rosenberg Self-esteem Scale; IAT: Implicit Association Test

Testing the Vulnerability Model

Two hierarchical regression analyses were conducted in order to predict symptoms of MDD and SAD at follow-up (12-14 & 10-11 extreme cases excluded, respectively). Results from the final analyses are presented in Table 2.5.

Table 2.4
Correlations Between Baseline and Follow-Up Scores (Pooled Data)

		Baseline				Follow-up		
		SAD	MDD	IAT	RSES	SAD	MDD	IAT
Baseline	MDD	.62**	-					
	IAT	-.06*	-.05*	-				
	RSES	-.70**	-.62**	.05	-			
Follow-up	SAD	.37**	.23**	-.01	-.30**	-		
	MDD	.24**	.36**	-.03	-.28**	.51**	-	
	IAT	.01	.02	.19**	-.01	>-.01	-.02	-
	RSES	-.30**	-.28**	.03	.48**	-.57**	-.55**	.05

Note. SAD: Revised Child Anxiety and Depression Scale - social anxiety disorder; MDD: Revised Child Anxiety and Depression Scale - major depressive disorder; IAT: Implicit Association Test; RSES: Rosenberg Self-Esteem Scale.
* p <.05, ** p <.01.

The significant predictors in the final step of the vulnerability model for MDD were baseline MDD, gender, and RSES. This suggests that lower explicit self-esteem and higher baseline MDD symptomatology were predictive of relatively higher scores in MDD symptoms two years later. Further, females accounted for higher MDD scores at follow-up compared to males. The final model explained 16% of variance in follow-up MDD symptomatology.

Similar findings were observed in the final step of the vulnerability model for SAD, in that baseline SAD, gender, and RSES were significant predictors and 16% of variance could be explained in follow-up SAD symptomatology. Further, there was also a significant interaction between gender and RSES scores. The regression analysis was conducted again, only split by gender. For females, in the final step of the model, RSES was not a significant predictor of follow-up SAD symptomatology (B = -.01, SE = .02, p = .47). For males, on the other hand, RSES was a significant predictor of follow-up SAD symptomatology (B = -.06, SE = .02, p < .001). Therefore, the difference between males and females in the prediction of SAD symptoms by explicit self-esteem was significantly different. Further analysis revealed that for males, only, relatively low explicit self-esteem was a significant predictor of relatively high SAD symptoms.

Table 2.5

Hierarchical Regression Analyses Predicting follow-up MDD and SAD Scores by Self-esteem (Pooled Data)

	MDD at Follow-up					SAD at Follow-up				
Baseline Measures	ΔR^2	B	SE (B)	Semi-Partial r	FMI	ΔR^2	B	SE (B)	Semi-Partial r	FMI
Step 1	.13**					.13**				
MDD/SAD		.29	.02	.36**	.16		1.47	.10	.37**	.15
Step 2	.02**					.02**				
MDD/SAD		.26	.02	.32**	.13		1.31	.10	.32**	.13
Gender		.87	.14	.15**	.14		1.01	.17	.14**	.11
Step 3	.003*					.005**				
MDD/SAD		.22	.03	.22**	.15		1.09	.13	.20**	.10
Gender		.84	.14	.14**	.15		.99	.17	.14**	.11
IAT		.03	.20	.003	.15		.25	.24	.03	.14
RSES		-.02	.01	-.06*	.18		-.03	.01	-.07**	.10
Step 4	<.001					<.01				
MDD/SAD		.22	.03	.22**	.15		1.08	.13	.20**	.10
Gender		.84	.14	.14**	.14		.99	.17	.14**	.11
IAT		.03	.21	.004	.14		.26	.24	.03	.13
RSES		-.02	.01	-.06*	.18		-.03	.01	-.07**	.11
IAT x RSES interaction		-.01	.02	-.02	.12		-.02	.02	-.02	.18
Step 5	.003					.004				
MDD/SAD		.18	.04	.11**	.17		.97	.18	.13**	.15
Gender		.84	.15	.14**	.14		.96	.17	.14**	.11
IAT		-.09	.30	-.01	.18		.40	.35	.03	.14
RSES		-.04	.01	-.07**	.18		-.06	.02	-.08**	.13
IAT x RSES interaction		>-.01	.03	>-.01	.19		.02	.04	.01	.16
Gender x MDD/SAD		.08	.05	.04	.16		.22	.28	.02	.20
Gender x IAT		-34	.44	.02	.22		-.16	.49	-.01	.11
Gender x RSES		.03	.02	.04	.17		.05	.02	.05*	.14
Gender x IAT x RSES		-.02	.04	-.02	.24		-.05	.05	-.02	.13

Note. FMI = Fraction Missing Info. SAD: Revised Child Anxiety and Depression Scale – social anxiety disorder; MDD: Revised Child Anxiety and Depression Scale - major depressive disorder; IAT: Self-Esteem Implicit Association Test; RSES: Rosenberg Self-Esteem Scale.

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

Testing the Scarring Effect

Two hierarchical regression analyses were conducted in order to test whether baseline SAD and MDD scores were predictive of follow-up RSES and IAT score (5-10 & 0-3 extreme cases omitted, respectively). Results are presented in Table 2.6.

Baseline MDD and SAD at no point added significantly to the model of prediction for follow-up RSES scores. In the final step of the model, baseline RSES and gender were significant predictors. This means there is a strong link in explicit self-esteem scores over two years, and females showed a smaller increase in explicit self-esteem compared to males. The final model explained 25% of variance in follow-up RSES scores.

At no step in the model of prediction for follow-up IAT scores was there a significant predictor other than baseline IAT scores. Baseline IAT score was only slightly related to follow-up IAT score, and the final model explained 4% of variance in follow-up IAT scores.

Discussion

The main findings of the present study can be summarized as follows: (i) There was partial support for the vulnerability model in that low explicit (but not implicit) self-esteem was predictive of relatively high MDD and SAD symptomatology at follow-up, even when controlling for baseline symptomatology; (ii) Discrepant self-esteem did not add to the prediction of follow-up MDD and SAD symptomatology as the interaction between explicit and implicit self-esteem showed no independent predictive value; (iii) There was no support of a scarring effect, neither in explicit, nor in implicit self-esteem.

Table 2.6

Hierarchical Regression Analyses Predicting follow-up RSES and IAT Scores with Baseline Symptoms of SAD and MDD (Pooled Data)

	RSES at Follow-up					IAT at Follow-up				
Baseline Measures	ΔR^2	B	SE (B)	Semi-Partial r	FMI	ΔR^2	B	SE (B)	Semi-Partial r	FMI
Step 1	.24**					.04				
RSES/IAT		.40	.02	.49**	.35		.19	.03	.19**	.31
Step 2	.006**					<.01				
RSES/IAT		.39	.02	.46**	.35		.19	.03	.19**	.31
Gender		-1.36	.44	-.08**	.24		-.01	.02	-.01	.38
Step 3	.002					.002				
RSES/IAT		.43	.03	.35**	.29		.20	.03	.19**	.31
Gender		-1.51	.44	-.08**	.24		-.01	.02	-.02	.39
SAD		.54	.37	.04	.30		.01	.02	.02	.31
MDD		.04	.08	.01	.29		.002	<.01	.02	.31
Step 4	.002					.002				
RSES/IAT		.46	.05	.25**	.26		.24	.04	.16**	.31
Gender		-1.49	.44	-.08**	.24		-.01	.02	-.02	.40
SAD		.25	.50	.01	.28		.01	.02	.01	.38
MDD		.12	.12	.02	.22		.003	.01	.02	.27
Gender x RSES/IAT interaction		.06	.06	.02	.25		.08	.06	.04	.32
Gender x SAD interaction		.62	.71	.02	.25		>-.01	.03	>-.01	.40
Gender x MDD interaction		-.14	.16	-.02	.26		>-.01	.01	-.01	.35

Note. FMI = Fraction Missing Info. SAD: Revised Child Anxiety and Depression Scale – social anxiety disorder; MDD: Revised Child Anxiety and Depression Scale - major depressive disorder; IAT: Implicit Association Test; RSES: Rosenberg Self-Esteem Scale.

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

The Vulnerability Model

We found partial support for a vulnerability model in that low explicit self-esteem at baseline was predictive of relatively high scores in both MDD and SAD symptomatology. This is in keeping with the results from the meta-analysis by Sowislo and Orth (2013) who found explicit self-esteem to be predictive of depression and anxiety symptoms, and Acarturk et al. (2009) who found explicit self-esteem to be predictive of SAD symptoms, specifically, in adults. The present study adds to these previous findings by including implicit self-esteem in the model. As such, we can conclude that implicit self-esteem does not account for any additional variance over the variance explained by explicit self-esteem. This is somewhat contradictory to previous studies that have found implicit self-esteem to be more predictive of symptoms of depression than explicit self-esteem (e.g., Steinberg et al., 2007).

One prominent difference between earlier studies and the current study concerns the participants' age. Previous longitudinal studies that have included implicit self-esteem in the prediction of depressive symptoms have all focused on samples over the age of 18. It is possible that while implicit self-esteem is more predictive than explicit self-esteem in an adult population, the reverse is true in an adolescent population. Previous studies have argued that depression differs qualitatively between children, adolescents and adults, and that it may not be one and the same disorder (e.g., Kaufman et al., 2001). If symptoms of adolescent depression differ from symptoms of adult depression, then it is feasible to suggest that they have different aetiological factors. Further, adolescent and adult implicit self-esteem may differ. The correlation between baseline and follow-up IAT scores was small, while the correlation between baseline and follow-up RSES scores was relatively high, suggesting that explicit self-esteem was more stable over the two years than implicit self-esteem. Research on dual processing models of cognition state that implicit processes are more robust as they build up over time based on repeated experiences (Beevers, 2005). It is possible that implicit self-esteem is more unstable during adolescence

than adulthood as adolescence marks a time for identity confusion and a decrease in the self-positivity bias that was previously present during childhood (Koole, Dijksterhuis, & Knippenberg, 2001). As such, adult and adolescent differences in depression or implicit self-esteem might explain why our results differ from previous similar studies. It should be noted that the small correlation between baseline and follow-up IAT scores may also reflect low reliability of the IAT. However, Bosson and colleagues (2000) concluded that based on the psychometric properties of several measures of implicit self-esteem, the IAT ranked amongst the highest in measures of validity and reliability. Also within the current sample, the reliability in terms of internal consistency was found to be good.

The second main difference between the present study and previous studies concerns the length of the follow-up period. While Orth, Robins, Trzesnieski, Maes and Schmitt (2009) found explicit self-esteem to be predictive of depressive symptoms up to three years later, implicit self-esteem has not been tested beyond six months (Franck, De Raedt, & De Houwer, 2007). If the relationship is causal, it is reasonable to suggest that changes in implicit self-esteem might have a more immediate effect on SAD and MDD symptoms relative to explicit self-esteem, and one would expect (at most) a weak association over a longer period. Future studies could adopt several assessment points, which would also provide further insight into the role of self-esteem stability in the prediction of SAD and MDD symptomatology (Zeigler-Hill, 2011).

There was a significant interaction between gender and baseline explicit self-esteem in the prediction of SAD symptoms. Further analysis revealed that for males, and not females, explicit self-esteem was significantly predictive of follow-up SAD symptoms. Therefore, gender acted as a moderator in the association between explicit self-esteem and symptoms of SAD. However, it should be noted that the effect size of the interaction in the present study was small (semi-partial $r = -.05$) and explicit self-esteem as a main effect still remained a significant predictor. Therefore, any future studies considering self-esteem interventions could still find benefits in both males and females. However, the present study highlights

the importance of considering gender differences in self-esteem research, particularly in an adolescent sample.

The Scarring Effect

Our findings do not support a scarring effect as baseline MDD and SAD symptomatology were not associated with explicit and/or implicit self-esteem at follow-up. This is in contrast to the findings from the meta-analysis by Sowislo and Orth (2013) who found that symptoms of depression and anxiety were predictive of subsequent explicit self-esteem. It is possible that a scarring effect is only present in adult depression and anxiety (see afore mentioned reasoning concerning sample age and time between measurements). Further, little is known about how long the scar takes to "heal", if it does "heal", and perhaps a scarring effect is present more immediately following rises in depression and anxiety symptoms. The lack of scarring effect in implicit self-esteem is in keeping with the study by Franck, De Raedt, and De Houwer (2007) who found that in a cross-sectional study, implicit self-esteem between never depressed and previously depressed individuals did not differ.

The scar model cannot be rejected on the grounds of the present findings, however. In the current study, we used the term scarring effect to refer to the potential linear relationship between psychopathology and subsequent self-esteem in a non-clinical sample. Therefore, it could still be that scarring occurs following remission from MDD or SAD (i.e., in a clinical sample), which is in keeping with the scar hypothesis (Lewinsohn et al., 1981). Further, it is possible that a lingering scar only occurs after a number of depressive episodes or prolonged SAD. Risch et al. (2010) found that remitted individuals with three or more depressive episodes showed lower implicit self-esteem than remitted individuals with less depressive episodes. Therefore, the low self-esteem scar may become more prominent with increased episodes, which would in turn make one more vulnerable for further relapse. This could potentially explain why increased number of episodes is linked to higher rates of relapse and decreased periods of remittance (Murray & Lopez, 1997). Further, Gayman, Lloyd and Ueno (2011) found an association between adolescent onset of depression and low

explicit self-esteem in young adulthood. This indicates support for a scar model in a clinical sample. Our findings therefore suggest that in a non-clinical (adolescent) sample, there was no scarring effect following symptoms of MDD and SAD.

Discrepant Self-esteem

We included the interaction between explicit and implicit self-esteem in the vulnerability model to account not only for differences between congruent high and congruent low self-esteem, but also for discrepant self-esteem. In the current study, we found no significant interaction, suggesting that patterns of congruent and incongruent self-esteem did not add to the prediction of symptoms in addition to the prediction of explicit self-esteem. This is in contradiction to Creemers et al. (2012) and Schreiber et al. (2012) who had found damaged self-esteem (i.e., high implicit and low explicit self-esteem) to be related to symptoms of depression and anxiety, respectively. Discrepant self-esteem may have a cross-sectional, and not a longitudinal, association with depression and anxiety. Further reasons might be related to age differences of the samples, which could influence the nature of psychopathology and self-esteem, as mentioned before. Another difference between previous studies and the present study is the statistical method in testing discrepant self-esteem.

Previous studies used a dummy variable to represent the direction of the discrepancy (fragile or damaged) based on median or mean splits within explicit and implicit self-esteem and a continuous variable highlighting the magnitude of this discrepancy (e.g., Creemers et al., 2012). By utilising this method, one can see whether any one direction is more predictive of symptomatology than the other direction. However, it does force people into either the damaged or fragile self-esteem group while their implicit and explicit self-esteem may actually be more congruent. The method used in the present study allows for comparison not only between damaged and fragile self-esteem, but also with congruent high or congruent low self-esteem. As this interaction is included in a model that also has implicit and explicit self-esteem as predictors, we were able to see whether combinations between implicit and explicit self-esteem accounted for additional or more

variance than the main effects. As such, in the present study, it would appear that explicit self-esteem, solely, accounts for increases in MDD and SAD symptomatology in a healthy adolescent sample.[1]

Implications

The partially significant vulnerability model suggests that explicit self-esteem interventions may prevent increases in SAD and MDD symptomatology. Current cognitive behavioural therapy (CBT) for depression and anxiety already includes components that may have a direct effect on self-esteem (e.g., correcting negative self-talk; Zeigler-Hill, 2011). Yet, providing comprehensive CBT seems premature for at-risk adolescents who do not actually fulfil the criteria of MDD or SAD. Preventative interventions focusing solely on self-esteem might be more appropriate and also more cost-effective, particularly if adolescents with low explicit self-esteem are able to partake in interventions by themselves at home. Dandeneau and Baldwin (2009) found that after partaking in a "find the smiling face" computer task (i.e., a rejection-inhibiting attentional training), adults reported higher self-esteem. Using such a task in future studies will provide more insight into the causal role of self-esteem in changes of MDD and SAD symptomatology and may in itself prove to decrease vulnerability for symptoms of MDD and SAD.

The present study also highlights that in a non-clinical adolescent sample, discrepant self-esteem does not add to the prediction of MDD and SAD symptoms. This only further emphasizes that focusing on increasing explicit self-esteem independent of implicit self-esteem could potentially be an effective prevention method against increases in symptomatology. As this is somewhat in contrast to previous studies who had found that implicit self-esteem contributed to the prediction either as a main effect or in

[1] In previous versions of the manuscript, we had tested a model that solely included discrepant self-esteem direction (i.e., fragile/damaged) and the magnitude of this discrepancy, as done in previous studies. We found that neither discrepancy direction, discrepancy magnitude, nor the interaction herein were predictive of SAD and MDD symptomatology. Following reviewer comments, we decided to omit this analysis as we agreed that in the absence of controlling for congruent self-esteem and implicit/explicit self-esteem as main effects the outcome of the analysis contributed little to our understanding of the role of self-esteem in the aetiology of MDD and SAD symptoms in a healthy adolescent sample.

combination with explicit self-esteem, it highlights the need for further research into the development of implicit self-esteem during adolescence. It is possible that as adolescence is a time of heightened identity confusion, explicit self-esteem is unstable and therefore automatic self-related associations are not yet formed. Indeed, some have argued that relatively large and frequent fluctuations in self-esteem from moment to moment (i.e., unstable [state] self-esteem) are more predictive of symptoms of psychopathology, than trait self-esteem as measured in a single moment (Roberts & Monroe, 1992). Once explicit self-esteem is more stable, with few fluctuations, then stable automatic behaviours may form on the basis of this. Explicit processes may change, but in theory, automatic behaviours change relatively slowly as associations are formed on repeated experiences with similar outcomes. If this is indeed the case, then increasing explicit self-esteem during adolescence may not only prevent increases in symptomatology, but may also decrease the chances of developing negative automatic self-associations (i.e., low implicit self-esteem).

The present study also highlights the importance of differentiating between a scarring effect and a scar. It is likely that sub-threshold SAD and MDD symptoms interfere very little with daily life, and therefore the relationship between SAD and MDD symptoms and subsequent self-esteem may not be entirely linear. Repeated exposure to higher SAD and MDD symptoms are more likely to affect, specifically, implicit self-esteem. This theory is consistent with the findings by Risch et al. (2010) that implicit self-esteem was lower in those who had experienced a number of depressive episodes.

Limitations

There are a number of limitations to consider in the present study. It should be noted that differentiating between the implicit and explicit is not the only way in which self-esteem has been characterised in research. Recent research showed that unstable self-esteem is related to a number of negative outcomes. For example, Franck and De Raedt (2007) found that unstable self-esteem (i.e., a relatively large variation in explicit self-esteem scores over a set period) increased vulnerability to depression, and

outperformed explicit self-esteem as a predictor. As self-esteem was measured only twice with a two-year gap in between, it was not possible to look at self-esteem stability. However, future studies may want to incorporate more frequent measurement points in order to get a broader insight into the role of self-esteem (instability).

The sample in the present study consisted of adolescents from a non-clinical population. In fact, after two years only 32 adolescents developed MDD (i.e., scored above the cut-off on the self-report questionnaire at follow-up and not at baseline). It could be that in a clinical sample, the association between self-esteem and symptoms is stronger. Further, no inferences can be made in the current study concerning the onset of MDD and SAD. In a clinical sample, one could look at number of depressive episodes or prolonged high levels of SAD which might find support for the scar model. Future studies using clinical samples will therefore be able to look at the association between self-esteem and specific aspects of MDD and SAD (e.g., number of episodes, relapse, onset).

We relied on self-report measures of SAD and MDD symptoms. Although the self-report measure used allowed SAD and MDD symptoms to be treated as a continuous variable (Hankin, Fraley, Lahey, & Waldman, 2005), it is more sensitive to response biases than, for example, clinical interviews. Such a response bias may be present in both the self-report on psychopathology symptoms, as well as the self-report on level of explicit self-esteem, which could, in theory, overestimate the association between explicit self-esteem and psychopathology. Future studies should seek to supplement these measures with behavioural measures.

Although we selected the measure that appears to have the most support for tapping into implicit self-esteem, relative to other well-known measures, the IAT is not without criticism (e.g., Fiedler, Messner, & Bluemke, 2006). It should be noted, however, that the psychometric properties of the IAT have been extensively tested (e.g., Bosson et al., 2000). Despite this, it should still be acknowledged that implicit self-esteem is a relatively new concept and research into the 'ideal measure' is still on-going.

Finally, it is important to acknowledge that in the present study the drop-out rate was 35%. The study by Orth, Robins and Roberts (2008) that used an adolescent sample and had a number of two-year follow-ups had an

overall missing data rate of 46% (both because of drop-outs and budgetary reasons). As such, the present drop-out rate was relatively low. Multiple imputation was considered to be an appropriate method for dealing with the present amount of missing data (Bodner, 2008) and all fraction missing information values in the present study were low. Although the effect sizes were small, t-tests revealed that participants missing at follow up had significantly lower self-esteem and higher MDD symptomatology at baseline. Therefore, some caution should be taken with the present results as they may not be representative of the complete sample.

Conclusion

In keeping with old and new cognitive models, the present large-scale longitudinal study in adolescents highlights the significance of self-views in the prognosis of MDD and SAD symptomatology. Although it remains to be seen if self-esteem is an aetiological factor, the significant association between explicit self-esteem and future symptoms of MDD and SAD highlights where preventative interventions may potentially be most effective. Persistent negative self-related thoughts would, logically, lead to an increase in negative mood and decrease in confidence. One would anticipate that a successful intervention that helps an individual to adopt techniques to raise self-esteem would decrease the likeliness of increased negative mood and decreased confidence, which could potentially decrease the vulnerability for MDD and SAD. The results from the current study suggest that interventions to increase explicit self-esteem, independent of the level of implicit self-esteem, should be the focus for future research.

3. Implicit and Explicit Self-Esteem in Current, Remitted, Recovered, and Comorbid Depression and Anxiety Disorders

Abstract

Dual processing models of psychopathology emphasize the relevance of differentiating between deliberative self-evaluative processes (explicit self-esteem; ESE) and automatically-elicited affective self-associations (implicit self-esteem; ISE). It has been proposed that both low ESE and ISE would be involved in major depressive disorder (MDD) and anxiety disorders (AD). Further, it has been hypothesized that MDD and AD may result in a low ISE "scar" that may contribute to recurrence after remission. However, the available evidence provides no straightforward support for the relevance of low ISE in MDD/AD, and studies testing the relevance of discrepant SE even showed that especially high ISE combined with low ESE is predictive of the development of internalizing symptoms. However, these earlier findings have been limited by small sample sizes, poorly defined groups in terms of comorbidity and phase of the disorders, and by using inadequate indices of discrepant SE. Therefore, this study tested further the proposed role of ISE and discrepant SE in a large-scale study allowing for stricter differentiation between groups and phase of disorder. In the context of the Netherlands Study of Depression and Anxiety (NESDA), we selected participants with current MDD (n = 60), AD (n = 111), and comorbid MDD/AD (n = 71), remitted MDD (n = 41), AD (n = 29), and comorbid MDD/AD (n = 14), recovered MDD (n = 136) and AD (n = 98), and never MDD or AD controls (n = 382). The Implicit Association Test was used to index ISE and the Rosenberg Self-Esteem Scale indexed ESE. Controls reported higher ESE than all other groups, and current comorbid MDD/AD had lower ESE than all other clinical groups. ISE was only lower than controls in current comorbid AD/MDD. Discrepant self-esteem (difference between ISE and ESE) was not associated with disorder status once controlling for ESE. Findings suggest a prominent role for ESE in MDD and AD, while in comorbid MDD/AD negative self-evaluations are also present at the implicit level. There was no evidence to support the view that AD and MDD would result in a low ISE "scar". One limitation is the correlational and cross-sectional design which limits causal inferences and conclusions about the direction of the relationships.

Keywords: self-esteem, depression, anxiety, scar, discrepancy

Self-reported low self-esteem has consistently been observed in episodes of major depressive disorder (MDD; e.g., Orvaschel, Beeferman, & Kabacoff, 1997) and in episodes of anxiety disorders (AD; e.g., Silverstone, 1991) like social anxiety disorder, panic disorder, and generalized anxiety disorder. It is unsurprising that low self-esteem is prevalent in MDD given that it is one of the possible symptoms as outlined in the most recent version of the Diagnostic and Statistical Manual of Mental Disorders ("feelings of worthlessness"; APA, 2013). In a meta-analysis (Sowislo & Orth, 2013), low self-esteem was shown to predict prospective symptoms of depression and anxiety, highlighting a potential causal role in MDD and AD etiology. The authors argued that low self-esteem might lead to social disruptions, increased self-focus, and rumination, which would in turn lead to the development (and maintenance) of depressive symptoms. For symptoms of anxiety, high self-esteem has been argued to act as an anxiety buffer (Greenberg et al., 1992), and therefore low levels of self-esteem would make an individual more vulnerable to anxious thoughts and feelings. Indeed, those who had been manipulated with a self-esteem boost reported less anxiety following a traumatic video than those who had received a neutral self-esteem manipulation (Greenberg et al., 1992). As such, the presence and relevance of low self-esteem in both AD and MDD is fairly indisputable. In the present article, we use the term "episode" also for the duration that a person meets the criteria for an AD without recovery.

Self-esteem has been predominantly indexed by self-report measures, which are limited by what the respondent is willing and able to disclose (Greenwald et al., 2002; Nisbett & Wilson, 1977). Meanwhile, relevant self-associations may not necessarily be accessible for conscious introspection, and individuals may dismiss certain initial associations as irrelevant when asked to verbalize a global affective self-evaluation. This points to the importance of complementing self-report measures reflecting the "self-endorsed", deliberate self-evaluations (explicit self-esteem; Zeigler-Hill & Jordan, 2010) with measures of automatic associations that require neither verbalization nor introspection (implicit self-esteem; cf. Fazio & Olson, 2003). The relevance of differentiating between explicit (ESE) and implicit self-esteem (ISE) is further emphasized by the view that both facets

of self-esteem are differentially involved in more controlled/strategic versus more automatic/spontaneous behaviors. ESE is considered to be especially relevant in the context of more deliberative/reflective behavior (e.g., choosing not to speak up in a group discussion), while ISE is argued to be critically involved in more reflexive, spontaneous behaviors (e.g., blushing during public speaking; Rudolph et al., 2010). In support of this, Spalding and Hardin (1999) found that those low in ISE, as indexed with an affective priming task, displayed more observer-rated anxious behaviors during an interview, specifically when the interview involved self-related questions. A more recent study found that ESE was related to self-reported measures of anxiety/nervousness, and controlled nonverbal behaviors during a speech (e.g., hand gestures to emphasize the point verbally made), while ISE, as indexed by both a cognitive load task and the implicit association test (IAT), was related to general observer-rated anxiety and spontaneous nonverbal behaviors during a speech (e.g., nervous mouth movements; Rudolph et al., 2010). Given the distinct roles of ISE and ESE in automatic and controlled dysfunctional (pathogenic) behaviors, it is feasible that these two components of self-esteem are (partly) differentially involved in AD and MDD.

Empirical evidence for the presence of low ISE in AD and MDD is mixed. Studies looking at ISE in AD samples are relatively few and seem to have focused almost exclusively on social anxiety disorder (symptoms). Two analogue studies using an IAT as a measure of ISE found relatively low ISE in females scoring high on symptoms of social anxiety (de Jong et al., 2012; Tanner et al., 2006). Consistent with this, a correlational study focusing on adolescents showed a negative relationship between symptoms of social anxiety and ISE measured with an IAT (de Jong et al., 2012). A more recent small-scale clinical study found that patients with social anxiety disorder (n = 45) had lower ISE, as indexed by an IAT, than healthy controls (Glashouwer, Vroling, et al., 2013). Patients with panic disorder (n = 24) showed a similar tendency that was not significant, possibly due to low statistical power. Concerning MDD, a meta-analysis of 25 studies (n = 2831, of which 77% non-clinical) indicated that there is an association between low ISE and symptoms of depression (Phillips et al., 2010). However, when focusing only

on studies using clinical, adult depressed samples, many have failed to find lower ISE in comparison to those who have never been depressed (De Raedt et al., 2006; Franck, De Raedt, & De Houwer, 2007; Franck, De Raedt, Dereu, et al., 2007; Lemmens et al., 2014). Risch et al. (2010) did, however, find lower ISE in current MDD patients (both first-onset and recurrent) compared to healthy controls, when using an IAT. Franck, De Raedt, Dereu and van den Abbeele (2007) also found evidence for lowered ISE in current MDD patients using an IAT but only in those without suicidal ideation, while MDD patients with suicidal ideation did not differ from never-depressed controls. As such, despite the strong theoretical grounds for anticipating low ISE in MDD and AD, the evidence thus far, particularly for MDD, is not very convincing, and suggests that dysfunctional self-related thoughts and behaviors may be most pronounced at the deliberate, explicit level.

Previous studies involving clinical samples may simply not have had enough power to detect an effect of ISE. Further, as little is known about ISE in comorbid MDD and AD, and many studies looking at MDD do not have anxiety disorder diagnosis as an exclusion criterion (e.g., De Raedt et al., 2006; Risch et al., 2010), it is important to look at ISE not only in comorbidity, but in (relatively) pure forms of MDD and AD as well. This may elucidate the apparently inconsistent earlier findings regarding ISE in MDD and AD. Therefore, the present study was designed in the context of a large-scale, national study: The Netherlands Study of Depression and Anxiety (NESDA; www.nesda.nl/). Specifically, ISE (and ESE) in (relatively) pure forms of MDD and AD, as well as in individuals with comorbid MDD/AD were analyzed. Given previous reports of relatively high reliability (Bosson et al., 2000) with strong evidence of validity (Rudolph et al., 2010), the IAT was used to measure ISE. In addition, we took suicidal ideation into account to test the robustness of the earlier finding that only in the absence of suicidal ideation is MDD associated with lowered ISE (Franck, De Raedt, Dereu, et al., 2007).

Given that low self-esteem is prevalent in both depression and anxiety, it seems feasible that self-esteem is a transdiagnostic factor which may explain the high rates of comorbidity between the two (e.g., Brown et al., 2001; Ohayon & Schatzberg, 2010). Indeed, low self-esteem may increase vulnerability for both depression and anxiety, and other factors may then

determine, specifically, which of the two develops. Consistent with this view, a relatively large scale study among non-referred adolescents found that the association between symptoms of depression and social anxiety could largely be explained by adolescents' explicit self-esteem (de Jong et al., 2012). This earlier study found that the interaction between symptoms of social anxiety and depression was not associated with self-esteem over and above the independent associations between self-esteem and symptoms of depression and social anxiety. This suggests that low self-esteem may increase the chance of concurrent symptoms of anxiety and depression by independently increasing the chance of developing both types of symptoms. Given that this earlier study was limited to a non-clinical sample of adolescents, it remains to be seen whether self-esteem can (partly) explain the high rates of comorbidity in an adult sample containing many who meet the clinical criteria for a depression and/or anxiety disorder. Therefore, the relationship between depressive symptoms and anxious symptoms are analyzed in the present study, once controlling for self-esteem.

It is important to see to what extent low self-esteem will be normalized when remitted/recovered from a depressive episode or anxiety disorder. Given the high rate of recurrence that is typical of both MDD and AD, identifying possible "scars" that remain following episodes (e.g., lowered ISE) may be crucial in identifying those who will relapse (Lewinsohn et al., 1981). Even when AD and MDD enters remission, or recovery, it is important to differentiate between ISE and ESE. It is feasible that those who are in remission/recovery are able to address negative self-related thoughts, and are motivated to reappraise a situation in order to derive at a more positive self-evaluation. Indeed, self-awareness and addressing negative thoughts effectively is the core aim of most therapies (e.g., cognitive behavioral therapy). However, given the lack of control over ISE, spontaneous (pathogenic) behaviors may continue that are not necessarily within the realm of awareness. Consistent with this, Vasey and colleagues (2012) found that implicit associations, as measured with an IAT, following an exposure therapy predicted return of fear in public-speaking phobia. Despite this, two studies reported that ISE and ESE in remitted MDD did not differ from never-depressed controls, both of which had used the IAT (Franck, De Raedt, & De

Houwer, 2007; Risch et al., 2010). Given the small sample sizes used, it is important to test the hypothesis in larger samples, and also take AD and comorbid MDD and AD into consideration as distinct disorders with distinct etiologies. As ISE may improve over time through consistent high ESE that becomes overlearned and the default reaction (Beevers, 2005), it is also important to differentiate between those whose symptoms have recently remitted and those who no longer meet the criteria for MDD and AD for some time (recovered). The present study therefore included remitted and recovered MDD, AD and comorbid MDD/AD groups.

For some individuals, ESE and ISE may differ considerably (i.e., *discrepant self-esteem*; Zeigler-Hill, 2011). While *fragile self-esteem* refers to the pattern of low ISE and high ESE, and has been linked to narcissistic tendencies (Zeigler-Hill, 2006), *damaged self-esteem* refers to the pattern of high ISE and low ESE, and has been linked to symptoms of depression (Creemers et al., 2012). While some studies looked at discrepancy by including the interaction between ISE and ESE into the model (e.g., Schröder-Abé et al., 2007; see also chapter two), others argued that this fails to acknowledge the potential influence of the direction of the discrepancy (e.g., Leeuwis, Koot, Creemers, & Lier, 2015). That is, the extent that ISE and ESE differ may only be related to symptoms of depression or anxiety when, for example, ISE is higher than ESE. Without distinguishing the direction of discrepancy, the interaction may appear statistically non-significant. Other studies looking at self-esteem discrepancies adopted analyses that allowed for differentiating between the direction of the discrepancy (i.e., fragile or damaged), but did not allow for the inclusion of the main effects (i.e., ESE and ISE; Briñol et al., 2006). The inclusion of ESE into the model resulted in an issue of multicollinearity. Given strong relationships between ESE and psychopathology are often reported, previously observed associations between damaged self-esteem and depression may have simply been an artefact of ESE, regardless of ISE. The final aim of the present study is therefore to explore an alternative method to analyse the role of discrepant self-esteem in MDD and AD which allows for differentiating between fragile and damaged self-esteem while statistically controlling for the potential main effect of ESE.

All in all, the present study tested the following hypotheses: i) those without a lifetime diagnosis of depression or anxiety (i.e., the comparison group) will show higher ESE and ISE than current MDD, AD, and comorbid MDD/AD; ii) ISE and ESE in the comparison group will also be higher than those who were recovered or remitted from AD, MDD, or comorbid MDD/AD; iii) those who have recovered or remitted from AD, MDD or comorbidity, will have higher ISE and ESE than those who currently meet the diagnosis; iv) self-esteem is a transdiagnostic factor, and as such, levels between MDD, AD and comorbid MDD/AD will not differ at the remitted level, the recovered level and at the current diagnosis level. Further, a novel way of testing the presence of self-esteem discrepancies in those with a current depression or current anxiety disorder is explored that allows for the inclusion of ESE as a main effect in the model.

Method

Participants

The Netherlands Study of Depression and Anxiety (NESDA; www.nesda.nl/) is an ongoing longitudinal cohort study that, at baseline (2004-2007), involved 2981 participants who have been followed-up biannually across a number of measures. In order to follow the long-term course of depression and anxiety, 1701 participants with a current depressive or anxiety disorder, and 907 participants with a life-time diagnosis or at-risk (e.g., subthreshold symptoms), were recruited from the community, primary care, and mental health organisations. A further 373 participants with no current or history of any depressive disorder or AD were recruited as controls. There were two exclusion criteria in the NESDA: 1) A primary, clinically overt diagnosis of a psychotic disorder, an obsessive-compulsive disorder, a bipolar disorder, or a severe addiction; 2) Non-fluent command of the Dutch language. A thorough overview of the recruitment process, design, and overarching aims of NESDA are published elsewhere (Penninx et al., 2008). All participants provided written consent, and ethical approval was granted by all ethical committees of participating universities (VU University Medical Center, Leiden University Medical Center and University Medical Center Groningen).

The present study makes use of data collected in the most recent wave at the time of writing, which is approximately 6 years since baseline, and the fourth biannual measurement. At this wave, 2256 participants were measured (24% attrition since baseline), where 1799 (80%) received the measures relevant for the present analysis. The remaining 457 did not receive all the measures for various technical reasons (e.g., completing measures at home or over the phone prohibiting computer-based measures). Participants who were given all relevant measures were aged between 23 and 72 (M = 48.05, SD = 13.18; 63.6% female). Participants were excluded from the present analysis if they had developed a bipolar disorder at some point during the study, or reported an alcohol dependence since the last interview (approximately 2 years ago; n = 83).

Clinical groups were formed for MDD and AD, and split by those currently in an episode (diagnosis in past month), those in remission (an episode that had ended in the last six - one month), and those recovered (an episode in the last seven years – six months). We used these cut-offs as these were more readily available within the study. It should be noted that what defines, for example, a depression in remission varies across studies. Frank et al. (1991) recommends that remission be considered as a depression-free period of 2-6 months, with longer than 6 months considered a recovery. Our cut-offs are not too far from this. Cut-offs for ADs are dependent on the type of AD, however we apply the same cut-offs as used for MDD for consistency when comparing the groups and creating comorbid groups. To create a more homogeneous group, participants were excluded from the current and remitted AD groups if they had also met the criteria for MDD (or dysthymia; n = 135 & 27, respectively) since the last interview. Likewise, those with an AD since the last interview were excluded from the current and remitted MDD groups (n = 78 & 45, respectively). Those in the recovered AD or MDD groups had no history of MDD (and dysthymia) or AD, respectively. Current and remitted comorbid AD and MDD (CM) groups were also formed based on the same criteria as the MDD and AD groups. Those who had comorbid dysthymia in the current CM and MDD groups were not excluded. A recovered CM group was not created given that the available information made it difficult to determine whether AD and MDD had occurred and ended

at the same time. The comparison (control) group consisted of individuals without a history of AD, MDD or dysthymia. The upper half of Table 3.1 provides an overview of the demographics and size of each group.

Measures

Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI is a self-report questionnaire measuring the severity of 21 anxiety symptoms in the past week (e.g., "Nervous", "Hot/cold sweats"). The degree of botheration is answered on a 4-point Likert scale from 1 (*Not at all*) to 4 (*Severely [I could barely stand it]*). Total scores were calculated (possible range: 21 – 84), with higher scores indicative of relatively more anxious symptoms in the preceding week. Missing answers were replaced with participant's mean response (n = 47). From the 1799 participants, 29 participants failed to return the questionnaire and four had more than nine missing answers; these were excluded from any analysis involving the BAI. The BAI showed excellent internal reliability across all those without missing answers (n = 2084; Cronbach's α = .92).

Inventory of Depressive Symptomatology – self-report (IDS; Rush et al., 1986). The self-report IDS was used to measure depressive symptomatology in the preceding seven days, based on the DSM-IV criteria for MDD. In the original version, at two points, participants can choose to answer one of two items (e.g., "Decreased appetite" or "Increased appetite"), and therefore answer 28 of the 30 items in total. The version in NESDA combines the paired items, and therefore contains 28 items, all of which are answered by the participant. For each of the 28 items (e.g., "Feeling sad") there are four corresponding answers from "0" that is indicative of no depression (e.g., "I do not feel sad") to "3" referring to a more severe depressive symptom (e.g., "I feel sad nearly all the time"). A total score is derived (possible range: 0 – 84), and higher scores are indicative of relatively severe depressive symptomatology. From the 1799 participants, 29 failed to return the questionnaire and three had too many missing answers (>6 items); these were excluded from any analysis involving the IDS. The IDS showed excellent internal reliability across all those without missing answers (n = 2150; Cronbach's α = .90).

Composite International Diagnostic Interview v2.1 (CIDI; Robins et al., 1988; Wittchen, 1994). Depressive and anxiety disorders were determined using the semi-structured CIDI (v2.1). The CIDI is used worldwide and WHO field research has found high inter-rater reliability (Wittchen et al., 1991), high test-retest reliability (Wacker, Battegay, Mullejans, & Schlosser, 1990), and high validity for depressive and anxiety disorders (Wittchen, 1994; Wittchen et al., 1989). Diagnosis of MDD, dysthymia, panic disorder (with and without agoraphobia), generalized anxiety disorder, social anxiety and agoraphobia were determined based on the criterion outlined in the DSM-IV. MDD episode severity was determined by the number of nine possible depressive criteria met (including the two core symptoms). Number of previous MDD episodes was asked at baseline when participants indicated a history of (or current) MDD. A total number of MDD episodes was derived by adding the number of waves where an MDD episode was reported to the number of previous MDD episodes reported at baseline. Age of onset at first MDD/AD episode was also asked when participants reported an MDD or AD disorder since the previous interview. Trained research staff conducted the interview.

Implicit Association Test (IAT; Greenwald et al., 1998). Implicit self-esteem was measured with a self-esteem version of the computer-based IAT. The IAT is a word-sorting task where words are presented from two target categories: *I* (I, myself, self, my, own) and *other* (other, you, they, them, themselves); and two attribute categories: *positive* (meaningful, successful, important, worthwhile, confident) and *negative* (worthless, unimportant, weak, failure, useless; translated from Dutch). Following two practice rounds of ten trials, participants sorted *positive*- and *I*- related words with the same key and *negative*- and *other*- related words with the other key (pairing 1). This was repeated for two blocks of 20 trials. Participants then completed another practice block of ten trials with only attribute words, although key allocation had been swapped. Participants ended the task with two blocks of 20 trials where *negative*- and *I*- related words (and *other*- and *positive*-related words) shared the same key (pairing 2). Reaction time of the initial response and accuracy were recorded. The premise of the IAT is that the attribute and target categories that are more strongly associated for the

participant are easier to sort when they share a key. A person with high implicit self-esteem is therefore expected to find it easier to sort words when *I* and *positive* share a key than when *I* and *negative* share a key.

The IAT was scored based on the D_4-measure (Glashouwer, Smulders, et al., 2013). First, trials with reaction times longer than 10,000 ms were discarded. Reaction times on error trials were replaced with the mean of the correct answers in that block with an added 600 ms error penalty. The mean reaction time for pairing 1 was then subtracted from the mean reaction time for pairing 2, and subsequently divided by the pooled standard deviation of both pairings to control for individual variation. Higher scores were therefore indicative of a relatively fast response when categories "I" and "Positive" share a key, thus indicating higher implicit self-esteem. Participants were excluded from any analysis involving IAT scores when more than 10% of trials were faster than 300 ms, an error rate of over 20%, or where more than 1% of trials were longer than 10,000 ms (n = 114; Greenwald & Farnham, 2000; Greenwald et al., 2003). Spearman-Brown corrected correlation between test halves was .85 (test halves based on trials 1, 2, 5, 6, etc., and 3, 4, 7, 8, etc.).

Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1989). Explicit self-esteem was measured with a self-report questionnaire containing 10 items answered on a 4-point Likert scale from 1 (*strongly agree*) to 4 (*strongly disagree*). Higher scores (possible range: 10 – 40) were indicative of more positive self-esteem. The measure showed good internal reliability in the present study (Cronbach's α = .92; based on all 1799 participants).

Scale for Suicide Ideation (SSI; Beck, Kovacs, & Weissman, 1979). The first five items from the original 19-item SSI were included in NESDA. These items were initially used as a screening instrument to identify those who had active or passive suicidal ideation before receiving the rest of the questions to gain further insight into the severity and attitudes of the suicidal ideation. The first five questions (e.g., "What feelings did you have last week about dying. Did you want to die and how strong was this wish") were asked in a semi-structured interview with answers given on a three-

point scale, where 0 indicated no suicidal ideation and 2 indicated moderate to strong suicidal ideation.

A dichotomous variable was created to identify those with suicidal ideation (1) and those without (0) in the current MDD group. Suicidal ideation was quantified by a score above zero on the SSI and on item 18 of the IDS ('Thoughts of death or suicide'; Glashouwer et al., 2010). Participants who scored 0 on both were identified as not having suicidal ideation. Those who scored a zero on one measure, and higher on the other, were excluded from the analysis.

Procedure

NESDA assessments take between three and five hours, and are completed in one sitting (see Penninx et al., 2008). Assessments contain computer tasks, self-report questionnaires, interviews, and biological measures carried out by trained staff. In all cases, participants completed the RSES after the IAT. Participants received travel expenses and a 15-euro gift certificate.

Statistical Analysis

In the first part of the analysis, a MANOVA was conducted with RSES and IAT scores as dependent variables, and group as the independent variable (current MDD, remitted MDD, current AD, remitted AD, current CM, remitted CM, recovered MDD, recovered AD or controls). Univariate extreme outliers in RSES and IAT scores, per group, were standardized values exceeding ±3.3 and omitted when present. In order to test for multivariate outliers, Mahalanobis distance was calculated by regressing participant ID number onto RSES and IAT scores for each group. With 2 degrees of freedom, and a critical alpha of .001, the critical chi-square value was 13.82. Mahalanobis values exceeding this were excluded from the MANOVA as they were considered multivariate outliers. Homogeneity of variance-covariance matrices was checked using Box's M. According to Tabachnick and Fidell (2007), robustness is not guaranteed when sample sizes are unequal and Box's M test is significant at p <.001. We reported Pillai's Trace for the multivariate tests, and conducted Sidak comparisons to adjust for multiple

testing. Cohen's d, unadjusted for the multiple testing, is reported for each significant comparison to give an indication of the effect size and as such, reported p-value (adjusted for multiple testing) and CI for the Cohen's d (not adjusted for multiple testing) may differ (e.g., the latter may contain a zero while the give p-value is significant). Comparisons between explicit self-esteem scores were conducted on means slightly different than those reported in Table 3.1 given that a number of participants had been excluded from the analysis based on invalid IAT scores.

In the final part of the analysis, looking at discrepant self-esteem (i.e., the extent that ISE and ESE differ), two logistic regressions were conducted: current MDD group (vs. controls), and current AD group (vs. controls). The absolute difference between standardized scores of the IAT and RSES were computed for all participants. Two discrepant self-esteem variables were created: damaged self-esteem and fragile self-esteem. The damaged self-esteem variable was computed by taking the absolute difference when IAT was higher than RSES; a 0 was assigned for participants where RSES is higher than IAT. Likewise, the fragile self-esteem variable was computed by taking the absolute difference when RSES was higher than IAT; a 0 was assigned for participants where the reverse was true. As such, for each participant, an absolute difference score appeared in either the damaged self-esteem variable or the fragile self-esteem variable, and had a score of 0 in the other discrepant self-esteem variable. The two discrepant self-esteem variables were entered at step one, with raw score on the RSES entered at step two.

Results

Missing Data Analysis & Descriptives

In order to check whether those who had received the self-esteem measures (i.e., completers, n = 1799) were not systematically different from those who had not (i.e., non-completers, n = 457), comparisons were made on BAI, IDS, and age. There was no evidence that the mean age of non-completers (M = 46.81, SD = 12.80) differed from completers (M = 48.05, SD = 13.18), $t(2254) = 1.81$, $p = .07$, $d = -0.09$, 95% CI [-0.20, 0.01]. Scores on the BAI and IDS were significantly positively skewed for both groups, and thus square-rooted (mean raw scores reported for interpretability). Results based

on equal variances not assumed suggested that non-completers had higher BAI scores than completers (M = 8.40, SD =9.64 versus M = 7.43, SD = 7.95), $t(518.02) = 4.57$, $p < .001$, $d = 0.28$, 95% CI [0.17, 0.39], and higher IDS scores (M = 17.95, SD = 13.85 versus M =14.52, SD = 11.31), $t(516.93) = 4.04$, $p < .001$, $d = 0.25$, 95% CI [0.13, 0.36].

Means and standard deviations of the various outcome and predictor variables are given in the lower half of Table 3.1. Transformations did not correct data skew for IAT, RSES, BAI and IDS scores (Shapiro Wilk's > .97, p's <.001) in the complete sample, and therefore Spearman's Rho is reported. There was a small but statistically significant positive correlation between ISE and ESE, $\rho(1600) = .18$, $p<.001$. Further, there were small but statistically significant negative associations between ISE and symptoms of anxiety, $\rho(1570) = -.09$, $p = .001$, and depression, $\rho(1572) = -.12$, $p<.001$, which disappeared once statistically controlling for ESE, $\rho(1569) = .02$, $p = .55$ and $\rho(1571) = .01$, $p = .78$, respectively. ESE was strongly correlated with symptoms of anxiety, $\rho(1684) = -.55$, $p<.001$, and symptoms of depression, $\rho(1685) = -.67$, $p<.001$. Anxiety and depression symptomatology were also highly correlated, $\rho(1682) = .78$, $p<.001$. This correlation decreased once controlling for ESE, $r(1681) = .68$, $p < .001$, and barely changed once controlling for ISE, $r(1568) = .77$, $p<.001$.

Within the current MDD group, 29 participants indicated no suicidal ideation and 14 did. Seventeen were excluded based on inconsistent answers on the SSI and the suicide-related item on the IDS. An independent samples t-test indicated that those with suicidal ideation reported lower ESE (M = 23.86, SD = 5.01) than those without (M = 28.17, SD = 5.13), $t(41) = 2.60$, $p = .01$, $d = 0.85$, 95% CI [1.53, 0.16]. Excluding 5 with invalid IAT scores, there was no evidence of a difference in ISE between those with suicide ideation (M = .68, SD = .61) and those without (M = .54, SD = .40), $t(36) = .81$, $p = 0.42$, $d = -0.29$, 95% CI [-1.02, 0.44]. Those with suicidal ideation (M = 35.71, SD = 10.78) did report substantially more depressive symptoms than those without (M = 23.00, SD = 7.20), $t(41) = 4.59$, $p<.001$, d = -1.50, 95% CI [-2.23, -0.76].

Table 3.1

Means (& standard deviations; unless stated otherwise) of demographics and variables per group

	Major Depressive Disorder (MDD)			Anxiety Disorder(s) (AD)			Comorbid MDD & AD		Comparison
	Current (n = 60)	Remitted (n = 41)	Recovered (n = 136)	Current (n =111)	Remitted (n = 29)	Recovered (n = 98)	Current (n =71)	Remitted (n = 14)	Non-Clinical (n = 383)
Age	49.05 (12.65)	49.02 (12.84)	46.95 (13.29)	48.85 (12.23)	45.45 (12.12)	47.56 (13.83)	46.90 (11.17)	44.93 (12.39)	48.23 (14.53)
Female (%)	68.3	70.7	61.8	70.3	75.9	57.1	69	71.4	57.1
BAI	12.85 (8.04)	9.38 (6.09)	5.47 (5.15)	14.03 (9.63)	11.17 (8.24)	6.16 (4.79)	20.32 (10.17)	9.08 (6.65)	2.74 (3.48)
IDS	28.05 (9.82)	19.83 (7.51)	12.26 (8.99)	20.74 (10.59)	16.14 (8.45)	11.42 (7.01)	33.86 (10.85)	18.17 (8.16)	5.46 (4.74)
Invalid IAT (n)	5	3	8	5	2	8	11	1	25
RSES	26.13^{e} (5.24)	27.71^{ed} (4.53)	32.45^{c} (4.24)	28.44^{ed} (5.11)	30.31^{cd} (5.23)	31.65^{c} (4.57)	23.07^{b} (4.98)	27.50^{ed} (3.88)	35.18^{a} (3.98)
IAT	.62^{ab} (.47)	.63^{ab} (.50)	.65^{a} (.45)	.61^{ab} (.44)	.48^{ab} (.56)	.64^{a} (.47)	.41^{b} (.47)	.54^{ab} (.41)	.74^{a} (.43)

Note. BAI = Beck Anxiety Inventory; IDS = Inventory of Depressive Symptomatology; RSES = Rosenberg Self-Esteem Scale; IAT = Implicit Association Test; Current = episode in the past month; Remitted = episode ended one – six months ago; Recovered = episode ended 6 months – 7 years ago.

For rows RSES and IAT: means with the same superscripts did not differ significantly (p > .05).

To explore differences in self-esteem between types of AD, two one-way ANOVAs were conducted. Differentiating between social anxiety disorder (n = 35), panic disorder (with or without agoraphobia; n = 21), agoraphobia (n = 26), and general anxiety disorder (n = 9), participants from the current AD groups were excluded if another AD was present in the previous six months (i.e., comorbidity within AD). Results indicated that while there was no evidence of a difference in ISE, $F(3, 82) = 0.05$, $p = .98$, partial $\eta^2 = 002$, there was a difference in ESE, $F(3, 87) = 5.00$, $p < .01$, partial $\eta^2 = .15$. Post-hoc t-tests (applying a Bonferroni corrected $\alpha = .008$) indicated that those with a social anxiety disorder (M = 26.80, SD = 4.56) had lower ESE than those with panic disorder (M = 30.90, SD = 4.29), $t(54) = 3.34$, $p = .002$, $d = -0.92$, 95% CI [-1.50, -0.34], and those with agoraphobia (M = 30.31, SD = 4.23), $t(59) = 3.06$, $p = .003$, $d = -0.79$, 95% CI [-1.33, -0.26]). There was no evidence of further differences between AD types.

Differences in Self-Esteem between Clinical Groups and Controls

A MANOVA following the exclusion of two extreme univariate outliers revealed a Box's M that did not exceed the critical cut-off point of .001 ($p = .03$). The multivariate test was significant suggesting an overall difference in self-esteem across groups, $F(16,1726) = 30.36$, $p < .001$, partial $\eta^2 = .22$. This was true for both ISE, $F(8,863) = 4.95$, $p < .001$, partial $\eta^2 = .04$, and ESE, $F(8, 863) = 80.91$, $p < .001$, partial $\eta^2 = .43$. Sidak comparisons suggested that those in the control group had significantly higher ISE than those in the current CM group ($p < .001$, $d = 0.78$, 95% CI [0.50, 1.06]), but with no evidence that it was higher than those in the remitted CM group, $p = .98$, $d = 0.48$, 95% CI [-0.07, 1.04]. Those who had recovered from MDD or AD also reported higher ISE than those in the current CM group (p's = .02, d's = 0.54, 95% CI [0.22, 0.85] & 0.58, 95% CI [0.25, 0.92], respectively). There was no evidence that those in the control group differed in ISE from those in the current AD group, $p = .15$, $d = 0.32$, 95% CI [0.11, 0.55], nor from those in the remitted AD group, $p = .12$, $d = 0.59$, 95% CI [0.20, 0.99]. There was no evidence of further differences in ISE between the control group and the other clinical groups, nor between the clinical groups (p's > .21). For ESE, the control group had significantly higher levels than all the current, remitted and recovered groups (p's <.01, d's = 0.66 – 2.95). Further, those who had

recovered from MDD and those who had recovered from AD had higher ESE than all remitted and current groups (*p*'s <.05, d's = 0.70 – 2.12) with the exception of remitted AD (*p*'s = .46 & .99, respectively). There was no evidence of a difference in ESE between those who had recovered from MDD and those who had recovered from AD, *p* = .99, *d* = 0.19, 95% CIs [-0.08, 0.46]. Further, those with a current CM had lower ESE than all MDD and AD groups (i.e., remitted & current; *p*'s <.01, d's = 0.70 – 1.43), and lower than those remitted from CM (*p* = .02, d = 0.92, 95% CI [-1.55, -0.29]). Finally, those with a current MDD reported lower ESE than those with a remitted AD (*p* = .01, d = 0.72, 95% CI [-1.20, -0.24]). There was no evidence of further differences between the clinical groups (*p*'s >.19).

Discrepant Self-Esteem

A two-step logistic regression was conducted to see whether discrepant self-esteem (step one) would predict current MDD from controls, and whether this would remain once including ESE (step two). In the control condition, 238 participants had fragile self-esteem (e.g., standardized RSES>IAT) and 119 had damaged self-esteem (e.g., standardized IAT>RSES). In participants with a current MDD, 15 had fragile self-esteem and 40 had damaged self-esteem. Correlations between ESE and fragile self-esteem, *r*(410) = .51, *p* <.001, and ESE and damaged self-esteem, *r*(410) = .-66, *p* <.001, were high but not multicollinear. Following the exclusion of eight extreme outliers, 88.4% of the participants would have been predicted accurately based on chance alone (i.e., a model without predictors). This accuracy increased to 90.3% (Nagelkerke's R_N^2 = .29) with the inclusion of absolute discrepancy for damaged self-esteem, Wald = 21.11, *p* < .001, OR = 3.03, and the absolute discrepancy for fragile self-esteem, Wald = 5.33, *p* = .02, OR = .32). With the inclusion of RSES scores, the accuracy increased to 93.1% (Nagelkerke's R_N^2 = .63). There was no evidence that damaged self-esteem, Wald = .09, *p* = .77, OR = .91, or fragile self-esteem, Wald = .09, *p* = .76, OR = 1.17, contributed significantly to this model. ESE was a significant coefficient, suggesting relatively higher levels decreased the odds of current MDD, Wald = 44.51, *p*<.001, OR = .60.

A similar two-step logistic regression was conducted to differentiate current AD from controls. In participants with a current AD, 40 participants had fragile self-esteem and 66 had damaged self-esteem. Correlations between ESE and fragile self-esteem, $r(461) = .52$, $p < .001$, and ESE and damaged self-esteem, $r(461) = -.65$, $p < .001$, were high but not multicollinear. Following the exclusion of eight extreme outliers, a model without predictors had an accuracy of 78.5%. This accuracy increased to 79.8% with the inclusion of the discrepancy self-esteem variables (Nagelkerke's $R_N^2 = .18$). Both damaged self-esteem, Wald = 18.20, $p < .001$, OR = 2.37, and fragile self-esteem, Wald = 6.69, $p = .01$, OR = .55, were significant predictors. With the inclusion of RSES scores in the model, the accuracy increased to 85.9% (Nagelkerke's $R_N^2 = .53$). Neither damaged self-esteem, Wald = 1.51, $p = .22$, OR = .73, nor fragile self-esteem, Wald = .72, $p = .40$, OR = 1.26, showed evidence of being significant determinants in the model. RSES scores was a significant determinant, Wald = 78.26, $p < .001$, OR = .64, suggesting that a relatively high ESE considerably reduced the odds of being in the current AD group.

Discussion

The key findings of the present study were: i) Implicit self-esteem was lower in current comorbid individuals compared to the non-clinical comparison group; ii) Explicit self-esteem was lower in all current, remitted, and recovered clinical groups than the non-clinical comparison group, and lowest in the current comorbid participants; iii) Explicit self-esteem in current MDD and AD was not lower than in remitted MDD and AD, respectively. However, self-esteem was lower than in recovered MDD and AD; iv) There was no support for discrepant self-esteem in current MDD and current AD, once statistically controlling for explicit self-esteem.

Previous studies have consistently found lowered ESE in individuals with MDD and AD (e.g., Orvaschel et al., 1997; Silverstone, 1991). It is not particularly surprising that current MDD had lower ESE in both previous studies and the present study given that low ESE is a possible symptom of MDD. Despite this, ESE was not lower in MDD than in AD, where low self-esteem is not a symptom per se. This may highlight low self-esteem as a

transdiagnostic factor explaining why comorbidity between depression and anxiety is so high (e.g., Ohayon & Schatzberg, 2010). Some argue that depression and self-esteem have overlapping causal factors, which may explain some of the shared variance. For example, Neiss and colleagues (2009) concluded that there was a common genetic and environmental influence on self-esteem, negative emotionality and depression, based on stronger associations in monozygotic twins, compared to dizygotic twins. In the present study, although self-esteem in current MDD and current AD was similarly low, the correlation between symptoms of depression and anxiety remained high when partialling out ESE. Although this partial-correlation is consistent with the findings from a previous cross-sectional study (de Jong et al., 2012), it need not mean that self-esteem does not increase the risk of developing comorbid depression and anxiety. A longitudinal design is required to see which aspects of a current depression or anxiety, like low self-esteem, is related to increased risk for comorbid anxiety and depression, respectively. It may be particularly important to identify which symptoms to target first in order to prevent comorbid disorders from developing given that comorbid AD/MDD is more persistent and treatment-resistant than purer forms of MDD and AD (Penninx et al., 2011).

Despite the indisputable presence of low ESE in AD and MDD, how it relates to anxiety and depression remains an important question. Low self-esteem has also been argued to have a causal role in the development of AD and MDD, as lower levels are often observed to precede symptomatology (Sowislo & Orth, 2013). Through social disruptions, increased self-focus, rumination, and lack of ability to negate or minimize the effects of anxious or threatening thoughts and feelings, low self-esteem could make a person more vulnerable to the development of AD and MDD symptomatology (Greenberg et al., 1992; Sowislo & Orth, 2013). This may explain why those with social anxiety disorder had lower ESE than both panic disorder and agoraphobia, given both the social-related aspects and feelings of anxiety that may arise in low self-esteem. Interventions that focus specifically on increasing self-esteem might prevent both MDD and AD from developing. For example, in competitive memory training, positive self-images are made more salient and therefore increases the likeliness that the positive self-

image is activated when the self is brought to attention. This intervention was found to improve self-esteem and depressive symptomatology in addition to treatment as usual in patients with MDD (Korrelboom, Maarsingh, & Huijbrechts, 2012), but it remains to be seen whether it can also be used in prevention. Given the nature of self-report measures (e.g., the awareness that answers will be seen by someone else), negative self-evaluations are purposefully self-endorsed in MDD and AD, self-esteem interventions should not only aim to change the content of self-related evaluations but also aim to learn how to oppose negative self-evaluations that arise.

ISE in relatively pure MDD and AD was found not to differ from controls. In other words, self-related negative thoughts occur when purposefully thought about in individuals with AD/MDD, whereas negative self-associations do not seem to arise at the automatic and reflexive level in this group. For MDD, this does not appear to be related to the presence (or absence) of suicidal ideation as was found in a previous study (Franck, De Raedt, Dereu, et al., 2007). The lack of low ISE in MDD is consistent with many prior findings (e.g., Lemmens et al., 2014) but conflicts with the findings of Risch and colleagues (2010). Given that the same measure of ISE was used, explaining the conflicting findings is not straightforward. As we found lower ISE in the current CM group, the potential presence of AD in the MDD group may have accounted for the lower ISE observed in their study. Indeed the presence of comorbidity was also reported in another previous study where low ISE was observed in individuals with social anxiety disorder (n = 33; 40% also reported a comorbid depressive disorder; Glashouwer, Vroling, et al., 2013). Although in this study a comparison of those with and without a depressive disorder suggested no difference in ISE, the small sample sizes may have limited the power to detect an effect. Previously reported presence of low ISE in AD as well as MDD might be explained by the unaccounted presence of comorbidity. Then together with the numerous previous cross-sectional studies using clinical populations where no difference in ISE was found (e.g., Lemmens et al., 2014), one would be tempted to conclude that low ISE is not a feature of pure AD and MDD. However, if ISE is formed following prolonged periods of ESE that becomes

overlearned, as argued by many (e.g., Zeigler-Hill & Jordan, 2010; although see DeHart, Peña, & Tennen, 2013), the low ESE prevalent in MDD and AD should eventually manifest in lower ISE. Further research is necessary to explore how ESE and ISE are related. Another assumption of this dual processing model is that the level of ESE is stable, before it becomes overlearned and automatic (Beevers, 2005). However, highly unstable ESE has been argued to be a larger vulnerability factor for MDD and AD than consistently low ESE (e.g., Farmer & Kashdan, 2014; Franck & De Raedt, 2007). As such, if ESE is particularly unstable in MDD and AD, then this may also explain why the findings involving ISE are relatively inconsistent.

The role of prolonged low ESE in the development of low ISE may explain why low ISE was observed in comorbid AD and MDD. There is some indication that the duration of symptomatology in CM would be longer given that remission rates for CM between baseline and two-year follow-up in NESDA were a lot lower, and more months with symptomatology were reported, than those with either MDD or AD alone. This is despite those with CM were more likely to seek treatment (Penninx et al., 2011). Prolonged periods of MDD and AD might give rise to longer periods of (stable) low ESE, and this in turn might explain why lower ISE was observed in CM only. Future studies looking at the duration, severity, and stability of low self-esteem, should look at whether the duration of symptoms in current clinical groups is associated with low ISE. Alternatively, more frequent measures of ESE over a period of time might be a more precise way of looking at the role of ESE in ISE, and could also be used to look at the role of self-esteem stability in clinical disorders (e.g., de Ruiter, 2015). Untargeted negative self-evaluations at the implicit level may also explain why those with CM are more likely to show a poorer response to treatment (e.g., Fava et al., 2015). Concerning self-esteem scars in those with a previous MDD and/or AD, ISE was not lower in remitted and recovered AD/MDD in comparison to controls. However, this is perhaps unsurprising given that current AD and MDD also did not display lower ISE. ESE in participants with a previous MDD or AD was significantly lower than those who had never had a MDD or AD. This might partially explain the highly recurrent nature of both disorders given that low ESE is argued to increase the vulnerability for symptomatology (Sowislo & Orth,

2013), and may suggest the need for a self-esteem intervention in those who have recently recovered or remitted from AD or MDD. Interventions for remitted MDD, in general, have been shown to be effective in reducing relapse risk (Bockting, Hollon, Jarrett, Kuyken, & Dobson, 2015), and often include aspects of targeting negative self-beliefs.

Damaged self-esteem increased, while fragile self-esteem decreased, both the likelihood of current MDD and current AD, compared to controls. This is consistent with previous findings (Creemers et al., 2012), although we extend this by highlighting that discrepant self-esteem variables were no longer significant once controlling for the main effect of ESE. Previous methodology either did not allow for the inclusion of ESE in the model (Creemers et al., 2012; Leeuwis et al., 2015), or did not allow for the specification of the direction of discrepancy (i.e., using the interaction between ISE and ESE to represent discrepancy). Although the current methodology allows for the inclusion of one main effect, it does not allow the direction of the discrepancy to be taken into account (i.e., damaged vs. fragile) regardless of the extent of discrepancy which has been argued to be an important factor in discrepant self-esteem (Leeuwis et al., 2015). However, the relevance of such a variable is debatable. Even if one were to select only those with discrepant self-esteem (i.e., excluding those with fairly congruent self-esteem), it is theoretically still anticipated that a relatively large discrepancy in one specific direction would have a stronger association with symptomatology compared to a smaller discrepancy within the same direction. Therefore, the method used in the present study not only allows for the inclusion of one main effect, but also eradicates variables that are difficult to interpret.

Although we feel that the method used in the current study is an improvement, there are still a number of limitations. In using the IAT and RSES to derive at measures of discrepancy there is an assumption that the two measures only differ on the construct they tap into. However, there are also important methodological differences. The RSES refers to explicit self-esteem in the past seven days, while in the IAT self-esteem is measured at that specific moment. Further, although ISE is anticipated to be a trait rather than a state, it is also argued to be context-dependent. As such, the lab

settings may have an influence. For ESE, participants might be able to reflect to more natural, daily situations. Consequently, not just a difference in timing, but also a difference in context might be measured. Ideally, a measure would be developed that directly taps into self-esteem discrepancy. We do not reject the notion of discrepant self-esteem, necessarily, but current methods (present study including) of combining ISE and ESE measures may introduce too much noise for quantifying discrepancies.

Limitations

Little can be said about the causal relationship of self-esteem in MDD and AD given the correlational and cross-sectional design of the present study. As low ESE was prominent across most clinical groups, the next logical step would be to study whether manipulating self-esteem influences symptomatology. There is some support that this may be the case given that self-esteem interventions also showed beneficial effects on symptoms of depression and anxiety (e.g., Korrelboom et al., 2012). However, it is also theoretically feasible that low self-esteem is the result rather than the cause of symptoms. Experience sampling might be a more elegant way of looking at whether self-esteem precede symptoms or symptoms precedes self-esteem, particularly as many studies argue that self-esteem fluctuates on a daily basis in response to negative and positive events (Greenier et al., 1999).

Even within the large scale NESDA study, both remitted AD (n = 29) and remitted comorbidity (n = 14) had comparatively small groups, and therefore the possibility to detect an effect may have been limited. Small clinical samples are relatively common in research (e.g., Franck et al., 2008; Risch et al., 2010) because, for example, recruitment can be difficult (especially if one needs relatively "pure" groups), and drop-out rates are high. The power to detect a difference between remitted comorbidity (i.e., the smallest group) and the comparison group was 83% for a large effect (.80), 45% for a medium effect (.50) and 11% for a small effect (.20; Faul, Erdfelder, Lang, & Buchner, 2007). As such, there was limited power to detect medium and small effects, which may have resulted in type II errors. However, the limited sample size is unlikely to have inflated the Type I error.

Future studies need to focus on recruiting larger samples of remitted AD and remitted comorbidity, as these, particularly, were underpowered in the present study.

There are a number of critics of the IAT, and other measures of ISE. Many criticisms concern the lack of applicability with regards to using cut-off scores (e.g., above a specific score is indicative of an implicit racial bias; Fiedler, Messner, & Bluemke, 2006). However, even when comparing scores on the IAT between groups, as in the present study, a number of criticisms remain relevant. One of the more recent criticisms concerns the inability for the IAT to highlight how attributes and targets are related (e.g., differentiating between implicit ideal self and actual self, Remue, De Houwer, Barnes-Holmes, Vanderhasselt, & De Raedt, 2013). Despite promising findings in differentiating between ideal self and actual self in dysphoric students (Remue et al., 2013), research is needed to justify the notion of implicit goal-oriented constructs (i.e., construct validity of implicit ideal self). Also, given the relatively more obvious nature in the way it was measured (i.e., "I am" and "I want to be" remained onscreen during the word-sorting task), it is unclear in how far this may have triggered explicit processing thereby influencing the speed with which words were sorted. Further criticism comes from the low validity of ISE measures, which have led some to doubt whether ISE actually exists. One common argument stems from comparing the validity of ISE measures to the validity of ESE measures (e.g., Buhrmester et al., 2011; Falk, Heine, Takemura, Zhang, & Hsu, 2015). Validity of ESE measures is likely to be overinflated given that biases and measurement error is likely to apply to all measures of ESE (i.e., inflated convergent validity), and is likely to apply to self-report measures of other constructs (i.e., inflated predictive validity). For example, self-enhancement bias is not only, presumably, going to affect all self-report measures of ESE, but also self-report measures of depressive symptomatology. As such, it is unsurprising that self-report measures of ESE often trump implicit measures of ISE. Another line of criticism regarding IAT and ISE seems to stem from the conceptual misunderstanding that a single self-esteem exists which can be accessed implicitly or explicitly. Doubts are then voiced because of the lack of correlation between ISE and ESE measures which is often lower than other

implicit and explicit constructs (Hofmann, Gawronski, Gschwendner, Le, & Schmitt, 2005). However, theories postulate that ISE is best considered as the most primitive self-evaluation. With increasing time, cognitive resources and motivation, other processes are activated that may alter, overrule, or support the initial reaction. Indeed, when encouraged to rely on their gut-feeling (i.e., intuition), correlations between ESE and ISE increase (Jordan, Whitfield, & Zeigler-Hill, 2007). There are many possible reasons as to why ESE does not correlate with ISE (e.g., self-enhancement bias, self-protection bias, narcissism), and not all apply to other implicit/explicit constructs. As a result, correlations between the two could be very small or non-existent, and may explain why correlations in other implicit/explicit constructs are higher (Hofmann et al., 2005). ISE is likely to be considered to contain more truth value in a person who values their "intuition" or "gut feeling" highly, or does not have the cognitive resources to effectively counter or further process ISE evaluations. Undeniably, the IAT is no perfect measure of ISE, and our understanding of ESE and ISE certainly requires further refinement. However, previous research has demonstrated the IAT to have high validity (Asendorpf, Banse, & Mücke, 2002; Rudolph et al., 2010; Spalding & Hardin, 1999) and reliability (Bosson et al., 2000; Glashouwer, Smulders, et al., 2013), and differences have been observed previously using this measure (e.g., Risch et al., 2010). Therefore, despite the shortcomings of the IAT, it would seem justified to use this measure, particularly as a highly valid and reliable measure still remains absent.

Conclusions

The present study was the first to compare levels of both ESE and ISE across various phases of MDD and AD groups and with careful consideration of comorbidity. We found that consistent across all clinical groups, at all phases, a more negative, self-evaluation was endorsed in comparison to the non-clinical control group. This may suggest that conscious behaviour consistent with low ESE is more likely to manifest in MDD and AD, and remain during remittance and recovery. However, negative self-evaluations at the implicit level were only evident in individuals with current CM, while mean levels of ISE in individuals with either MDD or AD were equivalent to mean levels of the non-clinical controls. This may not only highlight why CM

is more persistent and treatment-resistant, but also emphasizes the need for future research to investigate whether the aetiology of CM differs from MDD or AD. As such, it is also important to control for the potential presence of a comorbid disorder when further examining the relevance of ISE (and ESE) in AD or MDD.

4. Self-Esteem Instability in Current, Remitted, Recovered, and Comorbid Depression and Anxiety

Abstract

Self-esteem (i.e., global self-esteem, ESE) has not only been observed as being generally low in major depressive disorder (MDD) and anxiety disorders (AD), but also being relatively unstable (i.e., low self-esteem stability; SE-S). Low SE-S, potentially regardless of ESE, may be a crucial maintaining factor of MDD and AD due to increased vulnerability to daily stress and minor forms of perceived rejection. However, few studies have looked at SE-S in clinical samples, and none have differentiated between remittance and recovery to see whether low SE-S is persistent shortly and long after an episode. Therefore, the present study compared self-reported SE-S across current MDD (n = 60), AD (n = 111), and comorbid MDD/AD (n = 71), remitted MDD (n = 41), AD (n = 29), and comorbid MDD/AD (n = 14), recovered MDD (n = 136) and AD (n = 98), and never MDD or AD comparison group (n = 382). SE-S and ESE were measured by means of a self-report questionnaire. The comparison group had higher SE-S than all other clinical groups. Once controlling for ESE, differences with current MDD/AD, remitted MDD, and recovered MDD/AD remained, but disappeared for the comorbid groups. The current findings are consistent with the view that not only enduring low self-esteem per se, but also high self-esteem reactivity to external events may contribute to the development or maintenance of affective disorders. Further, such reactivity appears to persist into remittance and recovery which may contribute to the increased risk of relapse.

Keywords: self-esteem, instability, anxiety, depression, comorbidity

Low global self-esteem (ESE[2]; i.e., the degree that one values oneself irrespective of specific context) is a prominent aspect in most explanatory and causal models of major depressive disorder (MDD) and anxiety disorders (AD; Beck, 2002). Indeed, research has consistently found low levels of ESE in clinical samples (e.g., Orvaschel et al., 1997; Silverstone, 1991). Low ESE appears to precede increases in symptomatology suggesting a potential causal role (Sowislo & Orth, 2013). Many studies show that ESE is mostly consistent over the life span, with slight increases observed from adolescence to young adulthood, and middle age, before starting to decrease in old age (Orth & Robins, 2014). However, the extent of change in levels of self-esteem from moment-to-moment appears to vary between persons. Self-esteem stability (SE-S) refers to the extent and frequency of short-term self-esteem fluctuations usually in response to mood states (Clasen et al., 2015; Roberts & Monroe, 1994) or positive and negative daily situations (Kernis et al., 1991). This is somewhat in keeping with diathesis-stress models of depression and anxiety that highlight that those who are more vulnerable will react stronger to external factors, or require less intensity to gain a reaction (Zuckerman, 1999). With regards to MDD and AD, some have argued that low levels of baseline self-esteem (i.e., low ESE) are not a prominent aspect per se (e.g., Franck & De Raedt, 2007), but rather the degree and frequency of fluctuation from this baseline level (i.e., low SE-S; also known as unstable self-esteem or self-esteem instability). While ESE might be considered to reflect trait self-esteem, given the relevant consistent level which provides a self-esteem baseline across situations, SE-S is relatively state-like, with possible reactions to external situations and stimuli, and the potential for it to swing.

Prior studies looking at SE-S have been conducted using student samples with a focus on depressive symptomatology, and the results are inconsistent. Some found that SE-S was a better predictor of depressive symptoms than ESE (Roberts & Monroe, 1992), some found an interaction

[2] For consistency in this thesis, ESE is used to denote global (trait) self-esteem as the two constructs are measured with the same questionnaires. However, in the absence of implicit measures, previous research normally does not specifically state the explicit aspect of global/trait self-esteem. This is not to say that ISE only occurs at the trait level per sé (see Chapter 6).

between SE-S and ESE in predicting symptoms (de Man et al., 2001; Kernis et al., 1991; study 1, Roberts et al., 1995), while others failed to find the predictive validity of SE-S over and above ESE (study 2 & 3, Roberts et al., 1995). Given that the mean level of depressive symptoms in student samples are often very low, it is unclear how these findings relate to clinical samples of MDD or AD based on the few clinical studies conducted. Low SE-S was observed in social anxiety disorder, but this disappeared when ESE was taken into account suggesting that ESE was key to differentiating between those with and without a social anxiety disorder (Farmer & Kashdan, 2014). Individuals with a current MDD reported lower SE-S than a never-depressed comparison group, and similar levels of SE-S as those who previously met the criteria for an MDD (Franck & De Raedt, 2007). However, ESE was not controlled for in this study. Longitudinal analysis revealed that SE-S was related to symptoms 6 months later in the never-depressed comparison group and former MDD, while ESE and an interaction between the two were not (Franck & De Raedt, 2007). Research conducted till now seem to support the hypothesis that SE-S plays a role in MDD, either in addition to, or in an interaction with, ESE. Given the lack of relevant studies, the case for AD is weak, and it is unclear as to whether MDD and AD may differ in level of SE-S. As comorbid MDD and AD have previously shown to have lower levels of ESE than those with either an MDD or AD only (van Tuijl et al., 2016), it is feasible that SE-S may also be lower in the comorbid group which may explain the poorer rate of remittance (Penninx et al., 2011). Therefore, the first aim of the present study was to compare levels of SE-S between MDD, AD, co-morbid MDD and AD, and a never depressed or anxious comparison group (i.e., the comparison group).

Many studies report a significant positive relationship between ESE and SE-S (Okada, 2010), which is in keeping with assumptions concerning the relationship between trait and state levels of self-esteem in the sociometer theory (Leary & Baumeister, 2000). This theory postulates that individuals with high (trait) self-esteem are less likely to lower their self-esteem in response to rejection (state self-esteem), due to high expectations of being accepted. Indeed, this is mirrored somewhat by the finding that those with higher ESE are more likely to report more stable levels of self-esteem (Okada,

2010). The second aim of the present study, therefore, was to compare SE-S across clinical groups and a comparison group while correcting for ESE. Further, to test the possibility that SE-S is relevant only when ESE is low, the interaction between ESE and SE-S is explored in predicting symptoms of depression and anxiety.

High relapse and recurrence rates are often reported in MDD and AD. This has fuelled several scarring hypotheses that argue that following periods of symptomatology, residual cognitions like low self-esteem remain that increase vulnerability for relapse (Lewinsohn et al., 1981). Indeed, lower levels of ESE were observed in remitted and recovered AD and MDD when compared to those who had never been diagnosed with a depressive or anxiety disorder (van Tuijl et al., 2016). The notion that remaining scars lie dormant and can be activated by mild sad moods (Gemar et al., 2001; Segal, Gemar, & Williams, 1999), should mean that recovered and remitted MDD and AD are likely to report lower SE-S than the comparison group, even when controlling for ESE. Former MDD showed lower SE-S than a never-depressed comparison group, and similar levels as current MDD (Franck & De Raedt, 2007). However, the former MDD group did not differentiate between those who were in remittance (i.e., recently experienced an episode) and those who were recovered. It is feasible that scars continue to heal after an episode of MDD. Further, no studies have included clinical groups of remitted and recovered AD. Therefore, the present study differentiated within the clinical groups (MDD, AD and comorbid) between those who currently met the criteria for the disorder, those who were current in remission, and those who had recovered (for MDD and AD only).

Method

Participants

Participants were recruited from community, primary care and mental health organisations into the Netherlands Study of Depression and Anxiety (NESDA; www.nesda.nl/) if they currently had a depressive disorder or AD (n = 1701), were at risk of developing a disorder (e.g., family member with a depression) or had a life-time diagnosis (n = 907). A further 373 participants with no history of a depressive or anxiety disorder were recruited

as a comparison group. Baseline measures took place in 2004-2007 (N = 2981), and have been followed up biannually on several measurements. At baseline, exclusion criteria were: a) Primary diagnosis of other psychiatric disorders such as psychotic disorder, an obsessive-compulsive disorder, a bipolar disorder, or a severe addiction; b) Non-fluent command of the Dutch language (Penninx et al., 2008). The present study makes use of data collected at the six-year follow-up. There was a 24% attrition rate at this wave since baseline (N = 2256 remaining), and 1799 received the self-esteem measures (age range 23 – 72, M = 48.05, SD = 13.18; 63.6% female). Incomplete participation (i.e., no self-esteem measures given; n = 457) occurred for various technical and practical reasons (e.g., participation via telephone). A further 83 participants were excluded as they met the criteria for a bipolar disorder during the study, or reported an alcohol dependence since the last interview. All participants provided written consent, and ethical approval was granted by all participating universities.

The same clinical groups were used as in van Tuijl et al., 2016 (and Chapter three). To recap, clinical groups were formed based on answers given on the Composite International Diagnostic Interview (v2.1; CIDI; Robins et al., 1988; Wittchen, 1994). The CIDI is a semi-structured interview conducted by trained staff to determine depressive and anxiety disorders. Diagnosis of MDD, dysthymia, panic disorder (with and without agoraphobia), generalized anxiety disorder, social anxiety and agoraphobia were determined based on the criterion outlined in the DSM-IV. Information concerning disorder diagnosis and recency (when symptoms ceased) was used to form the different clinical groups (for more detail see van Tuijl et al., 2016). In brief, MDD and AD clinical groups were split by those currently in an episode (diagnosis in past month), those in remission (an episode that had ended in the last six - one month), and those recovered (an episode in

the last seven years – six months)[3]. In order to establish relatively pure MDD, those who had also met the criteria for AD since the last interview were excluded (n = 162). Likewise, participants who had a current AD and also met the criteria for any depressive disorder (e.g., MDD, dysthymia) since the last interview were excluded (n = 123). Those in the recovered AD or MDD groups had no history of MDD (and dysthymia) or AD, respectively. Current and remitted comorbid AD and MDD groups were also formed based on the same criteria as the MDD and AD groups. Participants who have no history of AD, MDD or dysthymia formed the comparison group. The upper half of Table 4.1 provides an overview of the demographics and size of each group.

Measures

Beck Anxiety Inventory (BAI; Beck et al., 1988). The BAI is a self-report questionnaire containing 21 anxiety symptoms. The degree of disturbance in the past week was answered on a 4-point Likert scale from 1 (*Not at all*) to 4 (*Severely [I could barely stand it]*). Higher total scores were indicative of more anxious symptoms. Missing answers were replaced with participant's mean response (n = 47). From the 1799 participants, 33 participants were excluded from any analysis involving the BAI (29 failed to return the questionnaire and four had more than nine missing answers). The BAI showed excellent internal reliability across all those without missing answers (n = 2084; Cronbach's α = .92).

Inventory of Depressive Symptomatology – self-report (IDS: Rush et al., 1986). A self-report IDS was used to measure the severity of depressive symptoms in the last week, based on the DSM-IV criteria for MDD. Twenty eight items (e.g., "Feeling sad") were answered with four options where "0" indicated no depression (e.g., "I do not feel sad") and "3" referred to a severe depressive symptom (e.g., "I feel sad nearly all the time").

[3] We used these cut-offs as these were more readily available within the study. It should be noted that what defines, for example, a depression in remission varies across studies. Frank et al. (1991) recommends that remission be considered as a depression-free period of 2-6 months, with longer than 6 months considered a recovery. Our cut-offs are not too far from this. Cut-offs for ADs are dependent on the type of AD, however we apply the same cut-offs as used for MDD for consistency when comparing the groups and creating comorbid groups.

Higher total scores were indicative of relatively severe depressive symptomatology. From the 1799 participants, 32 were excluded from any analysis involving the IDS (29 failed to return the questionnaire and three had too many missing answers [>6 items]). The IDS showed excellent internal reliability across all those without missing answers (n = 2150; Cronbach's α = .90).

Rosenberg Self-Esteem Scale (Rosenberg, 1989). A self-report questionnaire containing 10 items was used to measure global self-esteem. Answers were given on a 4-point Likert scale from 1 (*strongly agree*) to 4 (*strongly disagree*). Higher scores were indicative of higher explicit self-esteem. Excellent internal reliability was observed in the present study (Cronbach's α = .92).

Self-Esteem Stability. Two items from a five-item Self-Esteem Instability Scale were administered to measure self-esteem stability: "How much I value myself is subject to changes" and "How much I value myself is stable across several situations at various times"[4]. A four-item version was previously validated (Raes & Gucht, 2009). Answers to both questions were answered on a 5-point Likert scale from 1 ("completely does not apply to me") to 5 ("completely applies to me"). Higher scores were indicative of more stable self-esteem, based on total scores following the reversal of the answer to the first question. The two questions were significantly correlated, r(1797) = .38, p <.001. The relatively low correlation indicated that both questions were also partly complementary.

[4] In the interest of keeping NESDA measurements as concise as possible, two items were selected based on face validity that they related to the conceptual understanding of self-esteem stability, and were not completely overlapping. As such, a positively phrased item and a negatively phrase item were selected. Excluded items were "The extent to which I value myself may vary at different times", "A certain event can make me value myself more, or less than how much I valued myself before the event." and "I often switch between 'feeling extremely positive about myself' and 'seeing only the bad things about myself, and feeling like a failure".

Procedure

NESDA assessments take between three and five hours, and are completed in one sitting (see Penninx et al., 2008). Assessments contain computer tasks, self-report questionnaires, interviews, and biological measures carried out by trained staff. Participants received travel expenses and a 15-euro gift certificate.

Statistical Analysis

Bivariate correlations between SE-S and ESE, IDS, and BAI were calculated. Other possible correlations have been reported previously (chapter 3; van Tuijl et al., 2016). In the first part of the analysis, an ANOVA was conducted to compare SE-S across groups (i.e., current/ remitted/recovered MDD, current/remitted/recovered AD, current/remitted comorbidity and the comparison group). This analysis was then repeated with ESE as a covariate. In the second part of the analysis, two multiple regression analysis were conducted to predict variance in IDS scores (n = 1574) and variance in BAI scores (n = 1572). In both models, ESE scores and SE-S scores (both standardized) were entered at step 1. At step 2, the interaction between standardized ESE and SE-S scores was entered. Following a residual analysis, extreme residuals (±3.3) were removed before re-running the analysis to improve the fit of the model. Two-way interactions were probed using a method outlined by Dawson (2014), and Aiken and West (1991). Slopes were tested at ± 1 SD of ESE.

Results

Descriptives

Mean age, BAI, IDS, ESE, and SE-S scores, and the percentage females, per group, are presented in Table 4.1. Based on Spearmans Rho, SE-S scores were significantly related to ESE, $\rho(1714) = .67$, $p<.001$, IDS, $\rho(1685) = -.51$, $p <.001$, and BAI, $\rho(1684) = -.44$, $p<.001$. In other words, relatively high SE-S was associated with higher ESE, and less depression and anxiety symptomatology. Previous missing data analysis highlighted that those who did not receive self-esteem measures (n = 457) did not differ in age, but did

have higher BAI (*d* = 0.28) and IDS (*d* = 0.25) scores than completers (n = 1799; van Tuijl et al., 2016).

To explore differences in SE-S between types of AD, a one-way ANOVA was conducted. Participants were excluded from this analysis if another AD was present in the previous six months (i.e., comorbidity within AD). Groups were formed based on the current presence of a social anxiety disorder (n = 35), panic disorder (with or without agoraphobia; n = 21), agoraphobia (n = 26), and general anxiety disorder (n = 9). Results indicated that there was no difference between AD types in SE-S, F(3, 87) = 1.31, p = .28, partial η^2 = .04, thus supporting one current AD group incorporating all AD types. Conclusions were the same both when BAI scores and ESE scores were statistically controlled for.

Self-Esteem Stability between Groups

A one-way ANOVA comparing scores on the SE-S across groups was significant, F(8.941) = 45.82, *p* <.001. Levene's test was significant (*p* = .03), and group sizes were unequal, thus Games-Howell post-hoc ANOVA comparisons were conducted. The comparison group had higher SE-S than all current and remitted clinical groups (*d*'s 1.04 – 1.60), and those who had recovered from MDD (*d* = 0.66, 95% CI [0.48, 0.99]) and AD (*d* = 0.78, 95% CI [0.60, 1.11]). Those who had recovered from MDD, and those who had recovered from AD, had higher SE-S than all other clinical groups (*d*'s 0.44 - 0.90) apart from remitted AD (*p* = .64 & *p* = .91, respectively). Recovered MDD and AD did not differ from one another in SE-S (*p* = .999). There were no further differences (*p*'s > .14).

The one-way ANOVA was repeated with ESE as a covariate, to see whether earlier differences between SE-S remained when correcting for ESE, and was significant, F(9,932) = 97.35, *p* <.001, partial η^2 = .49. With ESE as a significant covariate, F(1,932) = 366.12, *p*<.001, partial η^2 = .28, there was a significant effect of group, F(8,932) = 6.42, *p*<.001, partial η^2 = .05. As Levene's test was significant, F(8, 933) = 2.36, *p* = .02, and group sizes unequal, more conservative Bonferroni post-hoc ANCOVA comparisons were conducted (estimated marginal means reported in Table 4.1). In correcting for differences in ESE, the comparison group still had higher SE-S than

current MDD (p=.01), remitted MDD (p<.001), current AD (p=.002), recovered MDD (p=.001) and recovered AD (p = .01). There were no further differences (p's >.08).

Interaction between SE-S and ESE, and Symptomatology

In predicting symptoms of depression, seven extreme residuals were removed before running the analysis. With the inclusion of ESE and SE-S scores at step one, the model was significant, F(2, 1677) = 755.96, p<.001 and predicted 47% of variance in IDS scores (adjusted R^2 = .47). At this step, both ESE (B = -6.93, SE = .25, p<.001, semi-partial r = -.49) and SE-S (B = -0.62, SE = .25, p = .01, semi-partial r = -.04) were significant coefficients in the model. With the inclusion of the interaction between SE-S and ESE, the model improved, F-change (1, 1676) = 17.08, p<.001, and now predicted 48% of variance in scores (adjusted R^2 = .48; final model - F (3, 1676) = 514.50, p <.001). Both ESE (B = -6.79, SE = .25, p<.001, semi-partial r = -.48) and SE-S scores (B = -0.78, SE = .25, p = .002, semi-partial r = -.06) remained significant coefficients. Also the interaction between ESE and SE-S was a significant factor in the model, B = 0.77, SE = .19, p<.001, semi-partial r = .07. The interaction is plotted in Figure 4.1, and simple slopes revealed that when ESE was high (+1 SD), there was no difference in IDS score across low/high SE-S, gradient of slope = -0.01, t = -.04, p = .97. However, when ESE was low (-1 SD), the slope was significant, gradient of slope = -1.55, t = -4.63, p <.001, suggesting that those with lower SE-S reported higher IDS scores than those with higher SE-S.

In predicting symptoms of anxiety, 21 extreme residuals were removed before rerunning the analysis. At step one, the model was significant, F(2, 1662) = 355.27, p<.001, and predicted 30% of variance in BAI scores (adjusted R^2 = .30). Both ESE (B = -3.42, SE = .19, p<.001, semi-partial r = -.37) and SE-S scores (B = -0.58, SE = .19, p = .002, semi-partial r = -.06) were significant coefficients in this model. With the inclusion of the interaction between SE-S and ESE, improved the model slightly, F-change (1, 1661) = 4.59, p = .03, and still accounted for 30% of variance in BAI scores (adjusted R^2 = .30; final model - F(3, 1661) = 238.89, p<.001). Both ESE, B = -3.67, SE = .19, p<.001, semi-partial r = -.36, and SE-S scores, B = -0.64, SE =

.19, p = .001, semi-partial r = -.07, remained significant coefficients in the model. The interaction between ESE and SE-S was also significant, B = 0.30, SE = .14, p = .03, semi-partial r = .04, and is plotted in Figure 4.2. Simple slopes revealed that when ESE was high (+1 SD), there was no difference in BAI score across low/high SE-S (gradient of slope = -0.34, t = -1.53, p = .13). However, when ESE was low (-1 SD), the slope was significant (gradient of slope = -0.95, t = -3.71, p <.001), suggesting that those with lower SE-S reported higher BAI scores than those with higher SE-S.

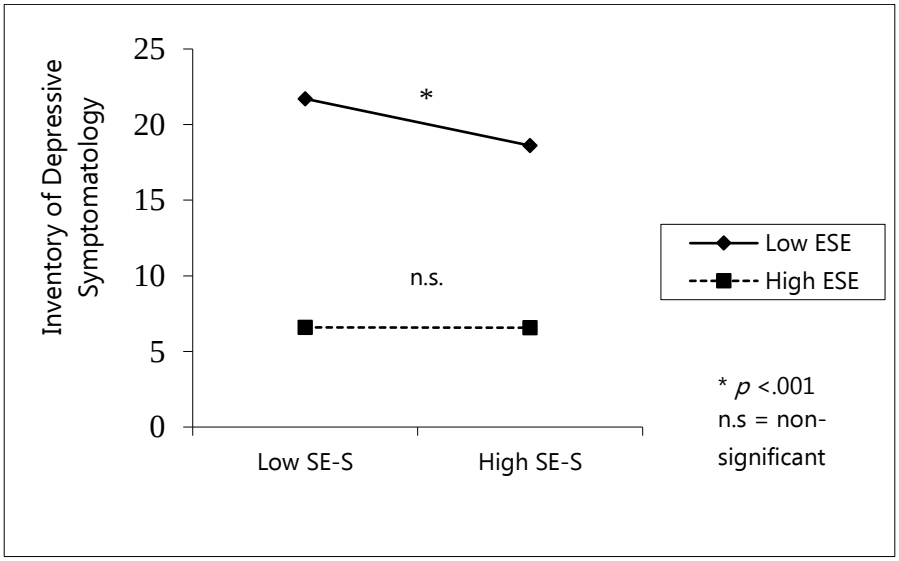

Figure 4.1. The two-way interaction between high and low (± 1 SD) ESE (global self-esteem as measured by Rosenberg Self-Esteem Scale) and SE-S (self-esteem stability) scores in the prediction of depression symptoms (N = 1680).

Table 4.1

Means (& standard deviations; unless stated otherwise) of demographics and variables per group

	Major Depressive Disorder (MDD)			Anxiety Disorder(s) (AD)			Comorbid MDD & AD		Comparison
	Current (n = 60)	Remitted (n = 41)	Recovered (n = 136)	Current (n =111)	Remitted (n = 29)	Recovered (n = 98)	Current (n =71)	Remitted (n = 14)	Non-Clinical (n = 383)
Age	49.05 (12.65)	49.02 (12.84)	46.95 (13.29)	48.85 (12.23)	45.45 (12.12)	47.56 (13.83)	46.90 (11.17)	44.93 (12.39)	48.23 (14.53)
Female (%)	68.3	70.7	61.8	70.3	75.9	57.1	69	71.4	57.1
BAI	12.85 (8.04)	9.38 (6.09)	5.47 (5.15)	14.03 (9.63)	11.17 (8.24)	6.16 (4.79)	20.32 (10.17)	9.08 (6.65)	2.74 (3.48)
IDS	28.05 (9.82)	19.83 (7.51)	12.26 (8.99)	20.74 (10.59)	16.14 (8.45)	11.42 (7.01)	33.86 (10.85)	18.17 (8.16)	5.46 (4.74)
ESE	26.13^{e} (5.24)	27.71^{ed} (4.53)	32.45^{c} (4.24)	28.44^{ed} (5.11)	30.31^{cd} (5.23)	31.65^{c} (4.57)	23.07^{b} (4.98)	27.50^{ed} (3.88)	35.18^{a} (3.98)
SE-S	5.25 (1.60)	5.22 (1.57)	6.77 (1.94)	5.86 (1.70)	6.14 (1.60)	6.60 (1.67)	5.15 (1.65)	5.14 (1.23)	7.97 (1.78)
SE-S EMM	6.37 (.20)	6.02 (.23)	6.58 (.13)	6.50 (.14)	6.39 (.28)	6.58 (.15)	6.92 (.20)	5.98 (.40)	7.21 (.09)

Note. BAI = Beck Anxiety Inventory; IDS = Inventory of Depressive Symptomatology; ESE = Global self-esteem as measured by the Rosenberg Self-Esteem Scale; SE-S = Self-Esteem Stability; SE-S EMM = SE-S estimated marginal means adjusted for group differences in ESE; Current = episode in the past month; Remitted = episode ended one – six months ago; Recovered = episode ended 6 months – 7 years ago. Adapted from "Implicit and Explicit Self-Esteem in Current, Remitted, Recovered, and Comorbid Depression and Anxiety", by L. A. van Tuijl, K. A. Glashouwer, C. L. H. Bockting, J. N. Tendeiro, B. W. J. H. Penninx, and P. J. de Jong (see Chapter 3).

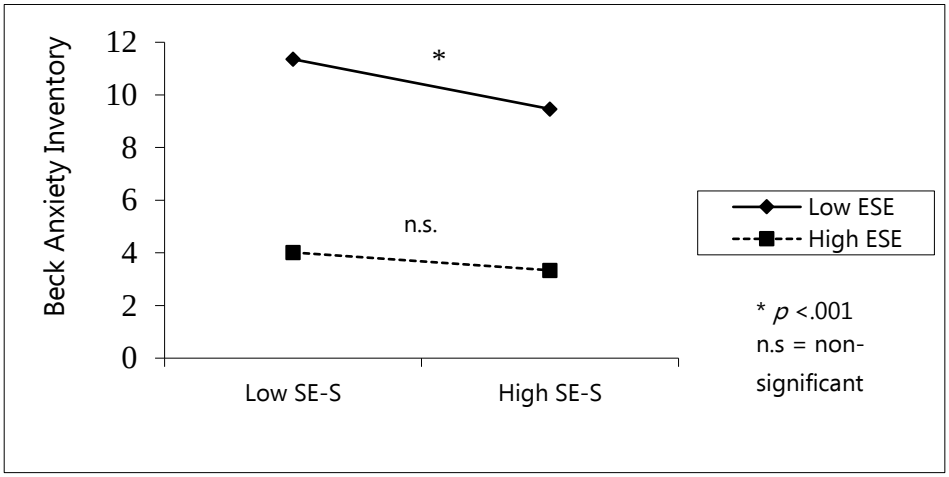

Figure 4.2. The two-way interaction between high and low (± 1 SD) E-SE (global self-esteem as measured by Rosenberg Self-Esteem Scale) and SE-S (self-esteem stability) scores in the prediction of anxiety symptoms (N = 1665).

Discussion

The main findings of the present study can be summed as follows: i) The comparison group showed higher SE-S than all current, remitted, and recovered clinical groups, whereas recovered AD and MDD showed higher SE-S than all other clinical groups; ii) For current MDD, current AD, remitted MDD, recovered MDD, and recovered AD, these differences in SE-S with the comparison group remained when correcting for ESE; ii) Particularly when ESE was low, symptoms of both depression and anxiety were related to low SE-S.

Self-esteem stability was lower in all clinical groups in contrast to the comparison group. This is in keeping with the previous studies who have found low SE-S in current MDD and AD (Farmer & Kashdan, 2014; Franck & De Raedt, 2007), and is consistent with previous studies focusing on analogue student samples (e.g., de Man et al., 2001). = The current findings extend those of Franck and De Raedt (2007) by highlighting that even when correcting for ESE, SE-S was still lowered in current MDD, remitted MDD and

recovered MDD. Moreover, low SE-S was also observed in the current AD group in the present sample, even when correcting for ESE. The latter is in contradiction with Farmer and Kashdan (2014) who found that the relevance of SE-S in social anxiety disorder disappeared when taking ESE into account. It seems unlikely that the conflicting findings are explained by broader inclusion criteria for AD of the present study since individuals with social anxiety disorder did not differ in SE-S from the other ADs. It is possible that Famer and Kashdan had less power to detect an effect given the smaller sample size, as in the present study the effect sizes were relatively small. Also, in Farmer and Kashdan's sample, 17.5% of the socially anxious individuals had a comorbid depression. Differences in comorbidity on SE-S were not compared. As such, the presence of a comorbid MDD may (also) account for the difference in findings, particularly as SE-S between the comparison group and comorbid group did not differ when controlling for ESE.

Differences in SE-S between comorbid MDD/AD and the comparison group disappeared once controlling for ESE, but remained for those with relatively pure MDD or AD. It is not entirely clear why lower SE-S was observed in MDD or AD, but not in comorbid MDD or AD. One explanation may lie in differences in ESE. In a previous study, comorbid MDD and AD was found to have lower ESE than both those with MDD and those with AD, potentially as a result of more persistent and severe symptomatology (van Tuijl et al., 2016). It is feasible that when ESE is already extremely low, there is little room for fluctuations. In other words, self-esteem cannot drop any lower. Likewise, those with relatively high ESE, like those in the comparison group, also have little room to fluctuate. As a consequence, the extent of instability might be similar between those with very high ESE (i.e., comparison group) and those with very low ESE (i.e., comorbid group).

The combination of low ESE and high SE-S may also explain the treatment-resistant nature of comorbidity (Penninx et al., 2011). Some self-esteem flexibility was argued to be vital for a psychoeducational group treatment to be effective, as those with lower SE-S pre-treatment showed a larger reduction in depressive symptoms (Roberts, Shapiro, & Gamble, 1999). As such, findings suggest that a self-esteem intervention is especially

necessary in comorbidity to not only increase ESE that is especially low, but also to introduce some flexibility into self-evaluations which may make other treatments more effective. Such an intervention may not be necessary for those with purer forms of MDD or AD, as common treatments such as cognitive behavioural therapy already appear to increase ESE in singular forms of these disorders (e.g., Richardson, Stallard, & Velleman, 2010), although it is unclear whether SE-S also increases. As such, it seems to be vital to differentiate between comorbid MDD/AD and relatively pure disorders as comorbidity may be more than simply the sum of MDD and AD symptoms.

In the present study, differences in SE-S were observed between the comparison group and remitted MDD, remitted AD, remitted comorbid, recovered MDD and recovered AD. These findings are in keeping with the lower SE-S observed in the former MDD group by Franck and De Raedt (2007). However, as we did not exclude recovered and remitted MDD with residual symptoms, Franck and De Raedt's findings are extended to highlight that low SE-S is present in both remitted and recovered MDD, more broadly. The presence of low SE-S in remittance and recovery could be explained in terms of a remaining SE-S scar that was a consequence of the episode. However, given the cross-sectional nature of the current study, it is just as feasible that this "scar" is a remaining prodromal factor that was present before the episode in question, or a preceding symptom of the next episode. Future longitudinal research should look at whether the extent of low SE-S following MDD and AD is predictive of (time to) relapse. Furthermore, scars have been hypothesised to lie dormant till activated by life events or stressors (Segal et al., 1999). Such stressors need not necessarily be major in order to (re-)activate the scars as self-esteem may fluctuate in response to subtle changes in mood and daily (minor) life events (Clasen et al., 2015; Kernis et al., 1991; Roberts & Monroe, 1994). As such, future longitudinal research may want to include a measure of (minor) stressors to see whether low SE-S specifically in the presence of stressors predicts relapse.

Further support for the differential role of ESE and SE-S in MDD and AD comes from the analysis of explaining variance in symptoms across both the clinical groups and the comparison group. For both, depressive and

anxiety symptomatology, SE-S explained variance over and above ESE, although ESE did explain more variance than SE-S. Consistent with the findings by De Man, Gutiérrez and Sterk (2001), particularly when ESE was low, SE-S was negatively related to depressive and anxiety symptoms. Previously, this has been taken to suggest that high SE-S is to some extent a protective factor when ESE is low. Indeed, those with more stable levels of low ESE may have short-term coping mechanisms when there are threats against self-esteem, while those who are reactive to threats may find it more difficult to deal with the resulting changes in self-esteem. It is also feasible to argue that fluctuations when self-esteem is high are not problematic because this may all occur within a positive range. Sociometer theory of self-esteem suggests that those possessing high self-esteem are less likely to react to instances of rejection given that acceptance is anticipated (Leary & Baumeister, 2000), as such, fluctuations may occur a lot less when ESE is high. Indeed, several studies have highlighted that self-esteem moderated responses to rejection (Ford & Collins, 2010). Further, many have reported a positive correlation between ESE and SE-S, suggesting that those with higher self-esteem are less likely to report instability (Okada, 2010), and this was also the case in the present study. Therefore, although there is support for distinction between ESE and SE-S, the two also appear to be related.

Symptom severity is often found to be a lot higher in comorbid depression and anxiety (Penninx et al., 2011). As such, there is some contradiction between the observed association between symptoms and SE-S, and the lack of support that the comorbid group and the comparison group differ on the latter. As the analysis dealt with depression and anxiety symptoms separately, it is plausible that in the presence of both symptoms, SE-S explains no additional variance over and above ESE. This is in keeping with theories that comorbid depression and anxiety is more than a sum of the parts (Kleiman & Riskind, 2012), and with previous observations of differences in another facet of self-esteem (implicit self-esteem) between comorbidity and the comparison group, but not with those with more singular forms of depression or anxiety (van Tuijl et al., 2016; chapter 3). These findings only further justify accounting for the presence of comorbidity within clinical groups. Future studies should adopt more

complex models as there are several ways in which comorbidity may occur (e.g., depression occurring first with anxiety symptoms developing second, and vice versa). Further it might be pivotal to acknowledge more complex associations between symptoms, ESE and SE-S, which may not be entirely linear. It is feasible that SE-S is particularly relevant in distinguishing individuals at risk of developing depression or an anxiety disorder when ESE levels are mid to low range, and not extremely low or high.

Another key direction for future studies would be to address the assumption that fluctuations in self-esteem are likely to be equal across negative and positive events. That is, whether self-esteem that is sensitive to negative events like rejection is equally as sensitive to positive events like acceptance. SE-S quantified by the standard deviation of multiple measurements or self-reported questionnaires like the one used in the present study assume this. However, in most contexts "bad is stronger than good" (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001), in that negative aspects (e.g., bad feedback) have a larger psychological impact than positive aspects (e.g., good feedback). While a previous study has highlighted that the extent that self-esteem decreases in reaction to sad mood is related to increases in depressive symptomatology (Clasen et al., 2015), it remains to be seen whether happy mood is as equally effective in raising self-esteem, and thus, reducing depressive symptoms. Given the potential presence of anhedonia in depression, it is feasible that the lack of positive-mood reactive self-esteem also plays a role in depression. This may also partially explain why ESE is low.

Limitations

Most previous studies looking at SE-S have quantified this construct based on the standard deviation of multiple self-report measures of ESE. This method may be less affected by self-report biases which assumedly would influence each measurement moment to a similar extent thus having a reduced influence on the standard deviation derived. The method employed in the current study would more likely be subject to self-report biases, and such bias would also affect measures of ESE to a similar degree (e.g., social desirability bias would presumably affect two measures concerning the self

to an equal extent). However, even when controlling for differences in ESE, differences in SE-S were still observed (albeit with small effect sizes), suggesting the measure of SE-S tapped into something else. Further, quantifying SE-S as is done in the present study also eases comparability. Given that previous studies have varied in how often they provide multiple measures of ESE (e.g., from weekly to daily), it is unclear what influence this may have on scores. Further, it is not clear how skewed scores should be dealt with (e.g., participants who often score high, or low), or extreme outliers (e.g., a rare good or bad day), both of which influence the mean, and subsequently the standard deviation (Baird, Le, & Lucas, 2006).

The present study adopted a cross-sectional design, and as such, the direction of the relationship between SE-S and depression and anxiety cannot be established. An important next step would be to test this association longitudinally to see whether low SE-S might be a vulnerability factor preceding increases in symptoms. In employing a longitudinal design, negative life events can also be recorded as many studies using student populations suggest that life stressors, particularly of an interpersonal nature, decrease SE-S which may increase subsequent depressive symptomatology (Hayes, Harris, & Carver, 2004). Such a design may also help differentiate between random fluctuations in self-esteem, and fluctuations in response to daily events.

In conclusion, the present study underlines the presence of self-esteem instability in clinical groups of MDD and AD. The current findings are consistent with the view that not only enduring low self-esteem per se, but also high self-esteem reactivity to external events may contribute to the development or maintenance of affective disorders. Further, such reactivity appears to persist into remittance and recovery which may contribute to the increased risk of relapse. From a clinical perspective, these findings highlight that interventions should not only aim to increase self-esteem, but also ensure that a stable level of self-esteem is achieved. If this is not the case, then a minor perceived rejection may undo any intervention effects.

5. Predicting Depression Relapse and Recurrence with Self-Depressed Associations

Abstract

Previous studies have highlighted that implicit and explicit self-depressed associations (SDA) were stronger in remitted depressed than in never depressed persons, and that more months with depressive symptoms and more previous episodes were related to stronger SDA associations at follow-up. In the present study, it was hypothesized that SDA, and the extent to which SDA weakens following recovery, represents a scar from depression increasing the risk for recurrence. Longitudinal analyses were conducted on data from the on-going Netherlands Study of Depression and Anxiety (NESDA) to test the value of implicit SDA (measured with the Implicit Association Test) and explicit SDA (self-report questionnaire) in predicting (time to) depression recurrence. Two main analyses were conducted: (i) predictive validity for recurrence based on SDA in individuals with a history of depression (at least six months depression-free) at the NESDA baseline assessment (n = 387, with six-year follow-up); (ii) predictive validity for recurrence based on the extent of change in SDA following recovery in individuals who had a current depression at baseline but were remitted at the two-year follow-up (n = 279, with four-year follow-up). Relapse/recurrence rate was 42.4% within the timeframe of the first analysis (six years), and 54.5% for the second main analysis (four years). The first analysis indicated that SDA were not related to (time to) recurrence, neither at the explicit, nor implicit level. In the second analysis, explicit, and not implicit, SDA predicted (time to) recurrence over and above residual depressive symptomatology. In conclusion, there was no support that implicit SDA represents an active scar after a depressive episode. However, explicit SDA might represent a scar, particularly when depressive symptoms have recently deceased.

Major depressive disorder (MDD), as defined by the DSM-IV, is one of the most prevalent lifetime disorders, with prevalence rates reported as high as 16.6% (Kessler et al., 2005). MDD is persistent with high rates of recurrence (i.e., return of symptoms following at least six months of no symptoms) and relapse (i.e., return of symptoms following a symptom-free period of less than six-months; Frank et al., 1991). Indeed, high rates of recurrence have been reported (42% within 20 years, Hardeveld et al., 2013). Time between episodes have been observed to decrease with each episode (Hardeveld et al., 2013), while the risk for recurrence increases with each episode (e.g., within 15 years, 71.1% with one prior episode vs. 82.8% with history of ≥3 prior episodes recurred, Mueller et al., 1999). As such, MDD can very much be considered a slippery slope given the persistent and sometimes treatment-resistant course. Given that the chance of developing MDD is much higher when there have been episodes in the past, many researchers have tried to identify hidden "scars" that remain following a period of depression (i.e., the scar hypothesis; Lewinsohn et al., 1981). These presumed "relatively permanent" scars are argued to highlight increased vulnerability, and make the path to depressive symptoms easier and quicker, hence the high rates of recurrence (Rohde, Lewinsohn, & Seeley, 1990).

Dysfunctional thoughts and attitudes about the self ("negative self-attributions") are a main element in many cognitive models of depression symptomatology (e.g., Beck, 2002). Given that low self-worth is thought to precede increases in depressive symptoms, even in healthy samples (see Sowislo & Orth, 2013, for meta-analysis), negative self-related attitudes that remain following an episode may add to the increased risk of recurrence. Often, self-related appraisals in those with a previous depressive episode are less negative in comparison to those currently in a depressive episode (Franck, De Raedt, & De Houwer, 2007; van Tuijl et al., 2016). Further, many depression interventions highlight an improvement in attitudes towards the self as an (additional) outcome (e.g., Richardson et al., 2010). However, self-associations are often still more negative compared to self-associations in never-depressed participants (van Tuijl et al., 2016), and as such, remaining negative self-associations may still represent a scar increasing the risk of recurrence. Indeed, Bockting and colleagues (2006) found that dysfunctional

attitudes (including attitudes about the self) predicted recurrence in recovered depressed individuals.

Most studies rely on self-report measures of self-associations, while self-associations at a more implicit level are not necessarily available for introspection, and as such, may be overlooked in questionnaires and diagnostic interviews (Gawronski & Bodenhausen, 2014). Further, given that negative implicit self-associations are thought to be overlearned explicit associations that become increasingly automatic (Beevers, 2005), they might be more persistent and more reluctant to change than explicit self-associations. As such, more negative self-associations at the implicit level, developed through prolonged periods of negative explicit self-associations prevalent in depression, may still remain following relative improvements in depression and explicit self-associations. Such negative implicit self-associations following an episode of depression may increase the risk for recurrence.

Self-depressed associations (SDA) refer to self-related evaluations that are depressive in nature. SDA at the implicit level, refers to the extent that the self is associated with attributes that are strongly linked to depression (e.g., hopeless, worthless) within memory structures. If these depressive attributes are strongly associated with the self, it may suggest that when the self is activated, concepts of depression are unintentionally, uncontrollably and potentially, non-consciously activated too. Pointing to the possible relevance of implicit SDA as a risk factor for recurrence, it was found that although implicit SDA were weaker in individuals who were remitted from a depression than in people with a current depression, they still were stronger than in a never-depressed comparison group (Glashouwer & de Jong, 2010). Further, in a single-predictor model, implicit self-depressed associations predicted the onset of the first episode of depression (Kruijt et al., 2013), and also predicted time to remittance in those with a current depression (Glashouwer et al., 2012). In depression recovery, more previous MDD episodes were related to stronger implicit self-depressed associations (Elgersma et al., 2013). Furthermore, in those with a current or history of depression, those who reported more months with depression in the preceding two years (particularly in women) reported stronger implicit SDA

at follow-up (Elgersma et al., 2013). Thus there is converging evidence suggesting that implicit SDA may play a role in the recurrent nature of depression. For implicit SDA to be considered a scar following depression, however, an increased vulnerability for recurrence should be present in those with relatively strong implicit SDA (Burcusa & Iacono, 2007). As of yet, no previous study has tested this hypothesis.

Explicit SDA refer to the potential depressive content of more deliberate self-related processing. When there is sufficient motivation, cognitive resources, and time, explicit processing related to self-evaluations may occur which involves retrieval of relevant memories and purposeful interpretation of propositions that either counters or supports the initial implicit association (Smith & DeCoster, 2000). Explicit processing is thought to manifest in deliberative behaviours, while implicit appraisals have been argued to be related to more spontaneous, impulsive actions (Strack & Deutsch, 2004). For example, a recently depressed individual may automatically feel targeted (implicit processing) when a mail about the importance of mental health circulates at work which may trigger nervous spontaneous behaviours like mouth movements. However, with motivation and the ability to do so (explicit processing), the individual may consider that he/she is currently feeling much better in comparison to a month ago, or might remember a colleague who recently endured a burn-out which more likely would have fuelled the e-mail.

Explicit SDA were found to be relatively strong in a remitted MDD group compared to a never-depressed comparison group, although weaker than those currently meeting the criteria for MDD (Glashouwer & de Jong, 2010). As with implicit SDA, recovered depressed individuals with more previous episodes and more months with depressive symptoms in the previous two years reported stronger explicit SDA. However, current levels of neuroticism appeared to account more for the relationship between explicit SDA and previous episodes, than for the relationship between implicit SDA and previous episodes (Elgersma et al., 2013). This may highlight that dysfunctional explicit SDA are more likely to be influenced (i.e., exacerbated or overruled) by current levels of depression-related characteristics, unlike implicit SDA. As such, while it may be easier to overrule and correct

remaining explicit SDA, implicit SDA is harder to change as it requires awareness of the output (e.g., sad mood) and trigger (e.g., the self was activated which subsequently activated depressive attributes). Implicit SDA may therefore be more likely to represent a scar than explicit SDA, given that it is harder to overrule and correct SDA. However, given that residual depressive symptoms are often observed in remitted depressed individuals, the ability to overrule and correct might be limited. The critical next step is to test whether the presence of a SDA scar, at both the implicit and explicit level, can predict (time to) recurrence in recovered depression (i.e., more than six months symptom free) and relapse in remitted depression (i.e., less than six months symptom free). Further, while a scar might highlight vulnerability for recurrence, those who show relatively more improvement in SDA following recovery from depression may have a weaker scar, thereby being less likely to recur than those who showed relatively little improvement in SDA.

The current study was designed to test the hypothesis that implicit and explicit self-depressed associations would predict (time to) recurrence in individuals who were remitted or recovered from MDD and/or dysthymia. Number of MDD episodes in the past and sex were previously shown to both be related to explicit and implicit SDA (Elgersma et al., 2013), and as such, these factors were included as potential predictors. Further, current depressive symptomatology was included in the analysis to control for any relapse or recurrence risk that might simply be explained by residual symptoms. Specifically, the first hypothesis is that stronger implicit SDA and explicit SDA will predict recurrence from non-recurrence in those with a history of depression, and also that time to recurrence will be shorter. For a more specific analysis of remaining SDA following remission, the second hypothesis was that relatively less weakening of SDA from current depression to recurrence will be related to (a shorter time to) recurrence. In other words, the second analysis tests whether there is a relationship between the persistent nature of SDA and (time to) recurrence. To test the specificity of the SDA, analysis was repeated using self-anxious associations when SDA showed predictive validity. We use the term recurrence throughout the method and results sections, although some in the sample

used to test the second hypothesis would have relapsed (i.e., return of symptoms within six months, Frank et al., 1991).

Method

Participants

The Netherlands Study of Depression and Anxiety (NESDA; www.nesda.nl) is an ongoing longitudinal cohort study. At baseline (2004-2007), participants were included in the study based on meeting the age criterion (18 – 65) and the presence of a depression or anxiety disorder (n = 1701), or if they were at-risk for or had a history of depression or anxiety (n = 907). A further 373 participants were included as the comparison group who reported no depression or anxiety currently or in the past, bringing the total sample to 2981. Participants who met the criteria for other psychiatric disorders (e.g., psychotic disorder, severe addiction) or did not have a fluent command of the Dutch language were excluded from the study. A thorough overview of NESDA has been described elsewhere (Penninx et al., 2008). All participants provided written consent, and all participating universities granted ethical approval.

The present study makes use of data collected at baseline, the two-year follow-up (T2), the four-year follow-up (T4), and the six-year follow-up (T6), and participants were selected to form two groups: i) Recovered at Baseline; and ii) Recently Depressed. To form the Recovered at Baseline group, 789 participants were selected who reported a history of either MDD and/or dysthymia at baseline and had not met the criteria for a depression for at least six months as determined by the Composite International Diagnostic Interview (CIDI; Robins et al., 1989; see measures section). Of these, 61 participants were excluded because they had not completed measures of implicit and explicit SDA at baseline (e.g., technical faults; remaining n =728). Participants were excluded if they met the criteria for a current anxiety disorder (social anxiety disorder, agoraphobia, panic disorder, general anxiety disorder, in the last six months, as determined by the CIDI; n

= 271)[5]. From the remaining 457, 70 were excluded as (non-) recurrence could not reliably be determined due to absence at follow-up. From the final sample (n = 387), and based on the CIDI, 223 had not recurred within the six years following baseline (57.6%), 97 had recurred by T2 (25.1%), 39 had recurred for the first time between T2 and T4 (10.1%), and 28 had recurred for the first time between T4 and T6 (7.2%).

To form the Recently Depressed group, participants were selected who reported MDD and/or dysthymia in the last month at baseline and were remitted at least a month (and no longer than two years) without dysthymia or MDD at the two-year follow-up (n = 426). Participants were excluded if they had not completed self-depressed associations measurements at both waves (e.g., technical reasons; n = 103), and a further 44 were excluded as (non-) recurrence could not be reliably determined due to absence at follow-up. Of the final 279, 127 had not recurred at all by the four- and six- year follow-up (45.5%), 101 had recurred by the four-year follow-up (36.2%), and 51 had recurred for the first time by the six-year follow-up (18.3%). Of these, only 105 reported no anxiety disorder in the previous six months at baseline (and 74 without an anxiety disorder at some point in the lifetime; based on the CIDI). These were therefore not excluded, but analyses were rerun excluding those with a recent anxiety disorder at baseline (i.e., within the last six months) to see whether conclusions held.

Measures

Implicit Association Test (IAT; Greenwald et al., 1998). A thorough overview of the depression IAT given at baseline and T2 in NESDA has been described previously (Glashouwer & de Jong, 2010). In brief, the depression IAT is a computer-based word-sorting task where words are presented from two target categories: *I* (I, myself, self, my, own) and *other* (other, you, they, them, themselves); and two attribute categories: *depressed*

[5] Although this reduced the sample by 37% it was felt necessary as means reported by Glashouwer and de Jong (2010) would suggest that those with an anxiety disorder also had strong self-depressed associations compared to a healthy comparison group. As such, it is unclear as to whether self-depressed associations in recovered depressed individuals with a current anxiety disorder represents a scar from a previous depression or a symptom from the current anxiety disorder.

(useless, pessimistic, inadequate, negative, meaningless) and *elated* (positive, optimistic, active, valuable, cheerful; translated from Dutch). Participants sorted *depressed*- and *I*- related words with the same key and *elated*- and *other*- related words with the other key (pairing 1). This was repeated for two blocks of 20 trials. In the next test block, *elated*- and *I*- related words (and *depressed*- and *other*- related words) were sorted with the same key (pairing 2). Response and reaction time are recorded for each trial. The premise of the IAT is that the attribute and target categories that are more strongly associated for the participant are easier to sort when they share a key. A person with strong self-depressed associations is therefore expected to find it easier to sort words when *I* and *depressed* share a key than when *I* and *elated* share a key. For all participants, an anxiety IAT was given before the depression IAT (see Glashouwer & de Jong, 2010, for description of anxiety IAT), and the IATs given at baseline and T2 were identical.

The IAT was scored based on the D_4-measure (Glashouwer, Smulders, et al., 2013). First, trials with reaction times longer than 10,000 ms were discarded. Reaction times on error trials were replaced with the mean of the correct answers for that participant in that block, with an added 600 ms error penalty. The mean reaction time for pairing one was then subtracted from the mean reaction time for pairing two, and subsequently divided by the pooled standard deviation of both pairings to control for individual variation. This was done for the practice blocks first, then the test blocks, before calculating the average between the two. Higher scores were therefore indicative of a relatively fast response for pairing one, thus indicating stronger implicit self-depressed associations[6]. Participants were excluded from any analysis involving IAT scores when more than 10% of trials were faster than 300 ms, an error rate of over 20%, or where more than 1% of trials were longer than 10,000 ms (Recovered at Baseline: 8 & 3 excluded, Recently Depressed: 6 & 9 from the depression IAT at baseline & T2, respectively; Greenwald & Farnham, 2000; Greenwald et al., 2003).

[6] Note that this is different from the other chapters in this thesis where the IAT was used to measure implicit self-esteem, and higher scores were indicative of a more positive construct (i.e., higher implicit self-esteem). Here, higher scores are indicative of a more negative construct (i.e., stronger self-depressed associations).

Spearman-Brown corrected correlations between test halves were previously calculated to be .92 and .91 (depression IAT), and .86 and .84 (anxiety IAT) for the complete sample at baseline and T2, respectively (test halves based on trials 1, 2, 5, 6, etc., and 3, 4, 7, 8, etc.; Glashouwer, Smulders, et al., 2013).

Explicit self-associations. Two measures of explicit self-associations were created for the purpose of NESDA at baseline and T2, one for depressed (vs. elated) and one for anxious (vs. calm). Participants scored from 1 "Hardly/not at all" to 5 "very much" how much each word from the depression IAT and anxiety IAT attribute categories described themselves. Scores for elated (calm) attributes were subtracted from depressed (anxious) attributes. Higher scores indicated stronger explicit self-depressed (self-anxious) associations. These measures have not been previously validated, but showed good internal consistency across the complete NESDA sample in a previous study (Cronbach's α = .94 & .95 for self-anxious and self-depressed, respectively at baseline, Glashouwer & de Jong, 2010).

Composite International Diagnostic Interview v2.1 (CIDI; Robins et al., 1988; Wittchen, 1994). Depressive disorders were determined using a semi-structured CIDI. Diagnosis of MDD and dysthymia were determined based on the criterion outlined in the DSM-IV. Recurrence was defined at T2 (for Recovered at Baseline only), T4, and T6 as meeting the criteria for MDD or dysthymia on the CIDI since the last interview. Number of previous MDD episodes was asked at baseline when participants indicated a history of MDD. Interviews were conducted by trained research staff.

Life Chart Interview (Lyketsos, Nestadt, Cwi, Heithoff, & Eaton, 1994). The number of months to recurrence was calculated using the Life Chart Interview. In the version given at T2, T4, and T6, primary autobiographical memories for each year in the preceding two years were asked for, and these were then used as a memory aid in recalling months with psychopathology and the amount of burden for each of those months (from 1 "no burden at all" to 5 "severe burden"). Based on similar previous study, months to recurrence was defined as the number of months depression-free till the first month with depressive symptoms that posed at least a small burden (Penninx et al., 2011). This was calculated either from

baseline (for Recovered at Baseline), or from T2 (for Recently Depressed). For those who did not recur based on the CIDI measure, the number of months refers to the number of months they were followed-up. Participants who had months with missing answers before recurrence were excluded from any analysis involving months to recurrence.

Inventory of Depressive Symptomatology – self-report (IDS; Rush et al., 1986). The IDS was used to measure depressive symptomatology in the preceding seven days, based on the DSM-IV criteria for MDD. The version used required a response to 28 items. For each of the 28 items (e.g., "Feeling sad") there were four corresponding answers from "0" which indicated no depressive symptom (e.g., "I do not feel sad") to "3" referring to a more severe depressive symptom (e.g., "I feel sad nearly all the time"). Answers were summed (possible range: 0 – 84), with higher scores indicating relatively severe depressive symptomatology. For Recovered at Baseline, one had too many missing answers (>6 items) at baseline and 5 at T2 (0 and 5 from Recently Depressed at baseline and T2, respectively), and were excluded from any relevant analysis. Previous studies have shown the IDS to have excellent internal consistency (e.g., Cronbach's α = .94, Rush, Gullion, Basco, Jarrett, & Trivedi, 1996).

Procedure

Participants completed computer tasks, self-reported questionnaires, interviews and biological assessments in one sitting lasting three to five hours (see Penninx et al., 2008). In return for participation, travel expenses and a 15-euro gift voucher was given to each participant.

Statistical Analysis

Recovered at Baseline. First, a binary logistic regression to highlight those who had recurred within the six follow-up years (1) and those who had not (0) was conducted. Baseline implicit SDA were entered at step one, and baseline explicit SDA, baseline IDS scores, sex, and number of MDD episodes in lifetime reported at baseline were entered at step two. Second, a Cox Regression was run to predict survival (i.e., the number of depression-free months till recurrence). Predictors were entered in the same order as the

logistic regression (i.e., first: baseline implicit SDA; second: sex, baseline IDs, baseline explicit SDA, number of MDD episodes in lifetime).

Recently Depressed. First, a binary logistic regression was conducted to predict who had recurred (1) by the four- and six- year follow-up from those who had not (0). Remitted depression IAT scores (i.e., IAT scores at the two-year follow-up) were entered at step one, with changes in depression IAT scores (T2 – baseline) entered at step two (i.e., changes in implicit self-depressed associations from current to remitted depression). At step three, sex, number of MDD episodes in lifetime reported at baseline, IDS score at T2, and changes in IDS score (T2 – baseline), explicit self-depressed associations at T2 and changes in explicit self-depressed associations (T2 – baseline) were entered. Second, a Cox regression was conducted to predict depression-free months till recurrence. The same variables from the binary logistic regression were entered in the same order.

There were a substantial few who had not provided an answer to the number of MDD episodes in lifetime or had not experienced any MDD episodes (e.g., history of dysthymia only; 13.4% for the Recovered at Baseline group). As such, when the number of MDD episodes was found not to predict (time to) recurrence, the analysis was rerun without this variable in order to maximize sample size. Analysis was rerun with self-anxious associations when SDA showed significant prediction, either at step one (single predictor) or step two (multi-predictor model), to test the specificity of self-related associations.

Results

Recovered at Baseline

Descriptives. Means and standard deviations of the demographics and relevant variables are presented in the left side of Table 5.1. Spearman's rank correlations were calculated between relevant baseline variables and months to recurrence, and are displayed in the upper half of Table 5.2. In those who had recurred, recurrence was quicker when baseline depressive symptoms were higher and explicit self-depressed associations were stronger.

Table 5.1
Means (SD) of Demographics, Depressive symptoms, and Self-Depressed Associations per Group

	Recovered at Baseline			Recently Depressed		
	Complete (n = 387)	Recurred (n = 164)	Non-recurred (n = 223)	Complete (n = 279)	Recurred (n = 152)	Non-Recurred (n = 127)
Age	43.51 (13.00)	41.88 (12.77)	44.71 (13.07)	41.47 (13.18)	42.08 (12.73)	40.75 (13.72)
Female (%)	72.2	73.8	71.3	64.9	65.8	63.8
# MDD episodes pre-NESDA	2.94 (6.49)	3.25 (4.48)	2.71 (7.65)	6.10 (13.07)	7.50 (16.57)	4.36 (6.12)
T0 IDS	14.05 (8.53)	17.69 (8.87)	11.36 (7.17)	32.32 (10.93)	34.18 (11.08)	30.10 (10.35)
T2 IDS	13.08 (9.65)	17.64 (10.60)	9.77 (7.31)	18.92 (9.76)	21.25 (10.27)	16.15 (8.34)
T0 EA depression	-2.12 (1.12)	-1.87 (1.17)	-2.31 (1.04)	-.26 (1.52)	.06 (1.54)	-.65 (1.39)
T2 EA depression	-2.09 (1.20)	-1.66 (1.36)	-2.41 (.94)	-1.37 (1.31)	-1.01 (1.31)	-1.79 (1.17)
T0 IAT depression	-.31 (.40)	-.29 (.39)	-.33 (.40)	-.15 (.40)	-.11 (.39)	-.18 (.41)
T2 IAT depression	-.33 (.38)	-.28 (.38)	-.36 (.38)	-.25 (.37)	-.21 (.38)	-.30 (.35)

Note. Recovered at Baseline = depression free for at least six months at T0; Recently Depressed = depressed in the last month at T0, depression free for at least two years at T2; T0 & T2 – baseline & two-year follow-up, respectively; IDS = Inventory of Depressive Symptomatology; IAT = implicit association test (higher scores = stronger self-depressed associations); EA – explicit associations (self-depressed associations – self-elated associations); MDD = major depressive disorder.

Recurrence vs. non-recurrence. A two-step logistic regression was conducted to identify participants who had not recurred in the six years (n = 218; 0) and those who had (n = 160; 1). Number of MDD episodes in lifetime was a non-significant predictor, thus the model without number of MDD episodes is reported in Table 5.3. At step 1, implicit SDA were not a significant predictor of relapse or recurrence, and 57.1% were correctly identified in this model; 0.6% less than at chance level (i.e., an empty model). With the inclusion of baseline IDS scores, explicit SDA, and gender, the

model improved to correctly classify 66.9%, X^2 (4) = 52.42, p<.001. Only baseline symptomatology was of independent predictive value, with higher scores relating to increased chance of recurrence. Conclusions did not change when excluding participants who had developed an AD before or at the same time as the depression recurrence (n = 103).

Table 5.2
Spearman's Rank Correlations (unless stated otherwise) per group

Recovered at Baseline (n = 387)						
	2.	3.	4.	5.	6.	
1. Age [a]	.04	.04	-.01	-.01	.03	
2. # MDD episodes	-	13*	.12*	.04	-.11	
3. T0 IDS	-	-	.51**	.09	-.22**	
4. T0 EA depression	-	-	-	.20**	-.19**	
5. T0 IAT depression	-	-	-	-	.06	
6. Months till recurrence	-	-	-	-	-	
Recently Depressed (n = 279)						
	2.	3.	4.	5.	6.	7.
1. Age [a]	.08.	.12*	.03	-.05	.06	-.07
2. # MDD episodes	-	-.03	.05	-.10	.002	-.11
3. T2 IDS	-	-	.55**	.09	.17**	-.23**
4. T2 EA depression	-	-	-	.15*	.25**	-.14
5. T0 IAT depression [a]	-	-	-	-	.40**	-.03
6. T2 IAT depression [a]	-	-	-	-	-	.02
7. Months till recurrence	-	-	-	-	-	-

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

Note. Recovered at Baseline = depression free for at least six months at T0; Recently Depressed = depressed in the last month at T0, depression free for at least two years at T2; T0 & T2 – baseline & two-year follow-up, respectively; IDS = Inventory of Depressive Symptomatology; IAT = implicit association test (higher scores = stronger self-depressed associations); EA – explicit associations (self-depressed associations – self-elated associations); MDD = major depressive disorder. Months to till recurrence = from baseline for recovered depressed, from the two-year follow-up for remitted depressed, in those who recurred.

[a] Pearson correlation

Time to recurrence. A Cox regression was conducted to predict months to recurrence. Number of MDD episodes in lifetime was a non-significant predictor, and was therefore excluded. Number of months till recurrence was calculated for 155 recurred participants, and the number of months follow-up was calculated for 218 non-recurred participants. At step

one, with baseline implicit SDA as the only predictor, the model was not significant, X^2 (1) = 1.01, p = .31. Including baseline explicit SDA, baseline IDS scores, and sex improved the model significantly, final model: X^2 (4) = 56.38, p <.001. IDS score was the only significant predictor in this model (see right side of Table 5.3), suggesting that relatively severe symptoms predicted quicker recurrence. Conclusions did not change when excluding those who had developed an AD before or at the same time as the depression recurrence (n = 90).

Table 5.3
Baseline Coefficients in Predicting Recurrence (Logistic) and Months to Recurrence (Cox) in those with a History of Depression at Baseline

	Logistic (n = 378)				Cox (n = 373)			
Baseline Coefficients	B (SE)	Wald	Odds ratio	*p*	B (SE)	Wald	Hazard ratio	*p*
Step 1								
Constant	-.23 (.13)	3.00	.80	.08	-	-	-	-
IAT depression	.26 (.26)	.99	1.30	.32	.20 (.20)	1.01	1.22	.31
Step 2								
Constant	-1.51 (.44)	11.73	.22	<.01	-	-	-	-
IAT depression	.07 (.29)	.05	.1.07	.82	<.001 (.21)	<.001	1.00	.99
EA depression	.05 (.12)	.15	1.05	.70	.06 (.09)	.51	1.06	.48
Sex	-.04 (.26)	.02	.96	.88	-.02 (.19)	.01	.98	.92
IDS	.09 (.02)	32.37	1.10	<.001	.06 (.01)	33.55	1.06	<.001

Note. IDS = Inventory of Depressive Symptomatology; IAT = implicit association test; EA = explicit associations (self-depressed associations – self-elated associations)

Recently Depressed

Descriptives. Means and standard deviations of the demographics and relevant variables are presented in the right side of Table 5.1.

Spearman's rank correlations were calculated between relevant T2 variables, baseline IAT scores, and months to recurrence, and are displayed in Table 5.2 (lower half). In those who had recurred, recurrence was quicker when baseline depressive symptoms were higher. Two paired-samples t-tests were conducted to see whether implicit and explicit SDA decreased from baseline to T2 (i.e., from current depression to remission). For explicit SDA, there was a significant decrease in strength, t(278) = 12.90, p <.001, Cohen's d = 0.78. Implicit SDA also became weaker, t(264) = 4.16, p<.001, Cohen's d = 0.28.

Recurrence vs. non-recurrence. A three-step logistic regression was conducted to predict recurrence at T4 and T6 (1; n = 140) vs. non-relapse/recurrence (0; n = 120; left side of Table 5.4). Number of previous MDD episodes was non-significant, and thus the model without this variable is reported here. An empty model predicted 53.8% accurately by chance alone. With the inclusion of post-depression IAT scores (T2), the model remained non-significant X(1) = 2.36, p = .12. Including changes in IAT scores from current to remitted depression (baseline – T2), did not significantly improve the model, X(2) = 2.65, p = .27. The final model with all variables included significantly predicted 65.4% of recurrence from non-recurrence, X(7) = 28.06, p < .001. There was only support for T2 explicit SDA, regardless of SDA levels during depression, as a significant predictor in the model, with stronger explicit SDA after depression increasing the likeliness of recurrence. When re-doing the analysis to check the specificity of explicit self-related associations, neither explicit self-anxious associations, nor the change herein were significant predictors of time to recurrence. The analysis was also rerun excluding those who met the criteria for an anxiety in the past six months at baseline (i.e., currently comorbid; 52 non-relapse vs. 45 relapse remaining). The resulting model for this reduced subsample suggested that although it was significant, not one predictor showed independent predictive value.

Time to recurrence. A Cox regression was conducted to predict months to recurrence, and results are presented in the right side of Table 5.4. Information about months to recurrence was available for 129 participants who had recurred, and months without recurrence for 111 participants who had not recurred. IAT scores at T2 (i.e., during remission) were not a

significant predictor at step one, and the model was not significant, X(1) = 1.84, p = .17. Entering changes in IAT scores did not improve the model, X(2) = 1.97, p = .37. Including sex, IDS scores at T2, change in IDS scores, explicit self-depressed associations at T2 and change scores from baseline to T2, improved the model significantly (final model: X(8) = 41.08, p <.001). More MDD episodes previously, stronger explicit SDA at T2 (i.e., during remittance) and more depressive symptoms at T2 were independently related to a relatively bad prognosis (i.e., quicker recurrence). In re-doing the analysis to check the specificity of explicit self-related associations, neither explicit self-anxious associations, nor the change herein, were significant predictors of time to recurrence. Repeating the analysis after excluding those with an anxiety disorder in the previous six months at baseline (leaving 39 relapsed and 49 non-relapsed with information concerning months of survival) produced similar results. The only difference was that in the final step of the model, explicit self-depressed associations at T2 was no longer a significant predictor.

Discussion

The present study sought to test the hypothesis that implicit self-depressed associations in those recovered and remitted from MDD and/or dysthymia would represent a vulnerability for recurrence and relapse. The findings suggest: i) Neither implicit nor explicit SDA predicted (time to) recurrence in those who were depression free for at least six months at baseline (Recovered at Baseline); ii) While implicit SDA provided no predictive validity, explicit SDA shortly following an episode of depression predicted (time to) recurrence in those who were depressed at baseline, and were symptom-free for at least one month at the two-year follow-up (Recently Depressed); iii) Findings involving explicit SDA were disorder specific and not replicated when using explicit self-anxious associations.

Table 5.4

Final Model of Coefficients Predicting Recurrence (Logistic) and Months to Recurrence (Cox) in those with Current Depression at Baseline, and Remittance at Follow-up

	Logistic (n = 260)				Cox (n = 240)			
Coefficients	B (SE)	Wald	Odds ratio	*p*	B (SE)	Wald	Hazard ratio	*p*
Step 1								
Constant	.29 (.16)	3.58	1.34	.06	-	-	-	-
T2 IAT depression	.54 (.35)	2.36	1.71	.13	.34 (.25)	1.84	1.41	.18
Step 2								
Constant	.30 (.16)	3.72	1.35	.05	-	-	-	-
T2 IAT depression	.64 (.40)	2.52	1.89	.11	.39 (.29)	1.89	1.48	.17
Change IAT depression	-.17 (.34)	0.26	0.84	.61	-.09 (.24)	0.15	0.91	.70
Step 3								
Constant	-.09 (.60)	0.02	0.91	.88	-	-	-	-
T2 IAT depression	.09 (.45)	0.04	1.09	.84	.06 (.31)	0.04	1.06	.84
Change IAT depression	.01 (.37)	0.001	1.01	.98	-.03 (.26)	0.02	0.97	.89
Sex	-.002 (.29)	<0.001	1.00	>.99	.17 (.20)	0.72	0.40	.18
T2 EA depression	.41 (.14)	8.21	1.50	.004	.25 (.09)	7.73	1.28	.01
Change EA depression	-.17 (.12)	2.08	0.84	.15	-.14 (.08)	3.34	0.87	.07
T2 IDS	.04 (.02)	3.43	1.04	.06	.03 (.01)	6.10	1.03	.01
Change IDS	-.001 (.02)	0.01	0.99	.94	-.001 (.01)	0.01	>0.99	.94
# of MDD Episodes	-	-	-	-	.01 (.01)	5.55	1.01	.02

Note. T2 = two-year follow-up; IDS = Inventory of Depressive Symptomatology; IAT = implicit association test; EA – explicit associations (self-depressed associations – self-elated associations); Change variables = T2 – baseline.

Previous studies made a strong case for the possibility of an implicit SDA scar in those with a previous depression. Those who were remitted from depression showed stronger implicit and explicit SDA than a never-

depressed comparison group, although SDA were stronger in those who currently met the criteria for a depression (Glashouwer & de Jong, 2010). Indeed, this was supported in the present study as improvements were observed when comparing SDA in the current and remitted phase at both the explicit and implicit level. Further, a potential SDA scar was also supported by studies who showed that those with worse depression prognosis in terms of months with depression symptomatology and number of MDD episodes in the past was related to both a higher recurrence risk (e.g., Hardeveld et al., 2013) and stronger implicit SDA (Elgersma et al., 2013). However, the findings in the present study suggest that remaining implicit SDA does not increase vulnerability for recurrence. Burcasa and Iacono (2007) argue that among other criteria, a depression scar must increase vulnerability for a new depression episode. It may be important to differentiate between scars that increase the risk for recurrence (i.e., active scars) from scars that remain but do not increase the risk for recurrence (i.e., passive scars). In the absence of including daily hassles or negative life events, passive scars that increase vulnerability only in the presence of activating factors (e.g., the diathesis-stress hypothesis, Zuckerman, 1999), were not tested in this study.

While implicit SDA did not have predictive value for recurrence, explicit SDA did, in those who entered the NESDA study with a depression and remitted by the two-year follow-up. The extent that participants considered themselves "useless", "pessimistic", "inadequate", "negative", and "meaningless" compared to "positive", "optimistic", "active", "valuable", "cheerful" shortly after a depressive episode, was related to quicker recurrence, even when controlling for depressive symptomatology. Many previous studies have shown that residual depressive symptoms following an episode predicts relapse (e.g., Hardeveld et al., 2013), thus it is somewhat surprising that a relatively simple explicit SDA measure may outperform a well-known validated measure of depressive symptomatology. Although this still needs to be formerly tested, it is feasible that measures of depressive symptomatology like the IDS tap into too many aspects of depression that are not all related to increased relapse risk. Indeed, the IDS includes various questions about somatic symptoms like sleep disturbances, changes in

appetite, diarrhoea/constipation, aches and pains, restlessness, and other bodily sensations while the presence of more affective symptoms might be a better predictor. This is supported by a study who found that asking remitted recurrent depressed individuals to rate their current mood on a single scale predicted time to relapse within 5.5 years better than 17-item depression scale that covered affective, behavioural and biological symptoms (van Rijsbergen et al., 2012). The IDS may therefore have included more noise than the self-depressed association questionnaire, as the latter may have tapped into more relapse-relevant symptoms. Given that explicit SDA, and number of previous episodes did not predict recurrence in the analysis involving those who were depression free for at least six months at baseline, while the IDS did, it is feasible that explicit SDA and number of previous episodes are predictive of relapse (i.e., failure to recover from the depressive episode) rather than recurrence (i.e., the onset of a new depressive episode). Further research is needed to test this latter point, as while those in the Recovered at Baseline group were all recovered (i.e., at least six months depression-free), those in the Recently Depressed group would have contained both remitted (less than six month depression free; Frank et al., 1991) and recovered individuals. As such, it is not possible to adequately compare recovery and remittance within the current approach.

A relatively severe course of depression previously was found to be related to the strength of implicit SDA (Elgersma et al., 2013). Yet, the present study would suggest that this does not increase vulnerability for recurrence. However, although the current findings do not support the hypothesis that implicit SDA are related to recurrence risk, it should be acknowledged that there are some methodological differences between the studies that may explain the findings. The present study used data derived from the same study, although the inclusion criteria were somewhat stricter than those imposed by Elgersma et al. (2013). Importantly, we excluded those with the presence of an anxiety disorder in the previous six months in the Recovered at Baseline group, which reduced the sample size by 37.2%. As such, it may simply have been a matter of power which did not allow for the detection of the effect. We therefore post-hoc repeated the logistic and Cox regression looking at implicit SDA in predicting (time) to recurrence in

the recovered depressed group without excluding those with a current anxiety. Implicit SDA was still a non-significant predictor for recurrence, both in a single-predictor and a multiple-predictor model, and therefore the lack of evidence for implicit SDA to predict recurrence seems not attributable to the absence of those with a current or recent anxiety disorder.

For Recently Depressed, excluding current anxiety resulted in a dramatic decrease in sample size, and explicit SDA were no longer related to (faster) recurrence. This may have been a matter of reduced power, or that more comorbid forms of depression and anxiety have a different aetiology than more singular forms. Indeed, previous studies have suggested that negative self-associations in more comorbid forms of depression and anxiety are a lot stronger than those with either depression or anxiety, even at the implicit level (Glashouwer & de Jong, 2010; van Tuijl et al., 2016). It is feasible that an interaction between symptoms of anxiety and SDA increases the likeliness of relapse as it signifies a post-episode state that is further from recovery than those with no symptoms of anxiety and weaker SDA. Future studies should aim to test a more complex model of recurrence, allowing for interaction between variables, and distinguishing between different post-episode states (e.g., remitted depressed with no anxiety disorder vs. remitted depressed with anxiety disorder).

The absence of a sad mood induction before measuring implicit SDA may also help explain the lack of predictive validity of implicit SDA for recurrence. Mild sad-mood states have been argued to trigger depressive-like processing in remitted depressed individuals (Gemar et al., 2001). Although sad mood did not appear to influence implicit self-esteem in healthy individuals (chapter 6), it is feasible that the influence of sad mood is limited to those with a history of depression as especially in these individuals depressive-like processing may lie dormant. Indeed, while implicit self-esteem following a sad-mood induction did not differ between remitted and never depressed individuals, the extent of change from before to after the sad mood induction did (Franck, De Raedt, et al., 2008). The extent of SDA reactivity in response to sad mood might therefore highlight a potential scar in remitted and recovered depressed individuals and, as such, it may be necessary to include a sad-mood induction to detect the implicit SDA scar.

This is consistent with the notion to differentiate between passive and active scars. A sad mood might be required to activate passive scars, and the sensitivity of passive scars to be activated and the extent of activation, might highlight predictive validity for recurrence (e.g., the diathesis-stress hypothesis, Zuckerman, 1999). Further research is required to test whether passive scars can indeed be triggered to become active scars, thereby increasing the risk of recurrence.

There are a number of limitations that need to be addressed. First, the present study relied solely on the IAT to quantify implicit SDA. A medium correlation was observed between scores as baseline and two-year follow-up, attesting to the reliability of the SDA IAT in NESDA. However, while the validity of an implicit SDA IAT has not been formerly tested, the validity and reliability of implicit self-esteem IAT has been reported. While implicit SDA refer to the extent that depressive content is related to the self in comparison to elated content, implicit self-esteem refers more broadly to the extent that negative content is related to the self, compared to positive content. The implicit self-esteem IAT ranked highly among other often-used implicit self-esteem measures in terms of predictive validity, test-retest reliability, and discriminant validity, convergent validity among measures was low (Bosson et al., 2000). Further, implicit SDA as measured by the IAT was previously found to predict remission amongst currently depressed individuals (Glashouwer et al., 2012), and depression onset amongst never-depressed individuals (Kruijt et al., 2013) further supporting the discriminant validity of the IAT. However, criticism remains concerning what exactly the IAT measures (Remue et al., 2013), and given the low convergent validity amongst measures, it is feasible that the IAT only partially captures implicit self-related associations.

Although participants with a current anxiety were excluded from Recovered at Baseline, this is not to say that participants did not have an anxiety disorder more than six months ago. Indeed, some participants may have recovered from a comorbid depression and anxiety. More thorough background information would be required to differentiate between previous comorbidity, and previous depression and anxiety that did not co-occur. Further, information given at baseline about depression and anxiety

present previously in life is subject to hindsight bias and forgetting. As the NESDA is an ongoing study, future analyses will be able to select those who have anxiety and depression onset during the study and be able to differentiate more reliably between depression, anxiety, and comorbid depression and anxiety. Although comorbidity is high and the disorders have been argued to have overlapping factors (Brown et al., 2001; Löwe et al., 2008), it may still be pivotal to differentiate between disorder (combinations), as comorbid anxiety and depression may have a different aetiology than simply a combination of the aetiologies of depression and anxiety.

In conclusion, (residual) implicit self-depressed associations as indexed by the IAT do not represent a scar that increases the likeliness of relapse and recurrence in formerly depressed individuals. Future studies should make sure to differentiate between those who may also have a history of comorbid depression and anxiety as aetiology for comorbidity might be different. Further, it is crucial to include a sad mood induction as scars may become particularly prominent during sad moods, and differences in how implicit SDA react to sad mood might highlight why some are more vulnerable for relapse than others. Importantly, explicit SDA did predict (time to) recurrence/relapse in those who were (on average) more recently remitted, regardless of how much improvement there was from levels of explicit SDA during the depression. This indicates that the extent that one associates oneself with negative depression-related adjectives following a depression predicts recurrence, and that this information is available for introspection and endorsed by the individual. Future studies should aim to reduce explicit SDA, in order to see whether this decreases the risk of recurrence, or increases the time to relapse. Such a study would also allow for inferences concerning the causal relationship between explicit SDA and recurrence in depression.

6. The Effect of Sad Mood on Implicit Self-Esteem

Abstract

Implicit self-esteem (ISE) refers to the valence of triggered associations when information about the self is activated. As context can influence implicit processing, it was hypothesized that sad mood would increase accessibility of negative associations therefore resulting in lower ISE. In this mixed-designed study, university students completed the self-esteem implicit association test (IAT) either at baseline (the control group; n = 46) or following a sad-mood induction (experimental condition; n = 49). Both conditions completed self-report measures of explicit self-esteem (ESE), and symptoms of depression and anxiety. There was no difference in ISE between the two groups, nor was there a relationship with self-reported sad mood and IAT scores. ESE did not moderate the relationship between sad mood and ISE. ISE was therefore concluded to be robust against increases in sad mood, regardless of ESE level. Results are limited to non-clinical participants, and results may differ in remitted depression.

Low self-esteem, often measured with self-report questionnaires has been linked to several dysfunctional outcomes including psychopathology (Zeigler-Hill, 2011). Self-esteem measured with self-report questionnaires is considered to measure the self that is endorsed (Zeigler-Hill & Jordan, 2010). However, dual-processing models of evaluation postulate that not all evaluations are done consciously or with purpose, and are thus not necessarily available for introspection. Implicit associations refer to the link between constructs which can vary in strength, and when associations are strong, it is possible that one construct can trigger another (Greenwald et al., 2002). Associations between constructs are thought to be strengthened by being simultaneously active (Greenwald et al., 2002), and through consistent and repetitive explicit associations that become default and automatic over time (Beevers, 2005). Implicit self-esteem (ISE) refers to the degree of overall positivity or negativity of associated constructs that are triggered when the self is triggered either by external stimuli or by the activation of other activated, associated constructs (Greenwald et al., 2002). It is pivotal to differentiate between more implicit and more explicit self-esteem, as they can lead to different types of behaviour (e.g., Rudolph et al., 2010).

Context is assumed to influence which associated constructs are triggered. Referring to pattern activation, Gawronski and Bodenhausen (2006) argue that the pre-existing network of associations and constructs, together with the external input stimuli (i.e., context), determine which associated constructs are triggered. They provide an example that "floating" is less likely to be triggered when a basketball is presented in a gym, but more likely when a basketball is presented by water. Context has been shown to be relevant for explicit evaluations as self-evaluations can differ when, for example, referring to academic performance or sporting ability. The effect of such a physical context on ISE has not yet been tested. It is feasible that completing a measure of ISE in the library might produce different results than completing the measure at home.

It is feasible that contexts not only refer to physical surroundings and situations, but that (negative) mood[7] also provides a context in which some associations become stronger than in other moods-contexts (e.g., happy mood, Beevers, 2005). Mood may influence ISE given that negative cognitive content and dysphoric moods are closely intertwined (Blaney, 1986). Therefore, if dysphoric mood is present, negative cognitive content may be more readily available and quicker to activate. Further, mood congruency has been observed in other implicit associations (e.g., anger increased implicit prejudice, DeSteno, Dasgupta, Bartlett, & Cajdric, 2004). Given that implicit processes are "quick and dirty", requiring less time, cognitive resources, and effort than explicit associations (Beevers, 2005), processing mood congruently would be most efficient. Regarding ISE, levels following a sad-mood induction were found to be lower in remitted depressed participants compared to never depressed participants (Gemar et al., 2001), although this effect disappeared when ISE differences before the mood induction were taken into account (Franck, De Raedt, et al., 2008). Regardless of group differences, in both studies, ISE did appear to drop following a sad-mood induction. Further, following a social threat invoking feelings of anxiety, those with a social anxiety disorder reported lower ISE than those without (Ritter, Ertel, Beil, Steffens, & Stangier, 2013).

These earlier studies focused on comparing (remitted) clinical groups with a never-depressed comparison group. Yet, whether sad mood influences ISE regardless of (previous) psychopathology has not yet been formerly tested. Further, in the case of Franck et al. (2008) and Gemar et al. (2001), the assumption is made that measures of ISE can be given twice in relatively quick succession. Reduced scores may therefore have been explained by test-retest effects rather than sad mood. As people generally score positively on most ISE measures (e.g., Bosson et al., 2000), a learning effect would manifest as differential scores becoming closer to zero which would also appear as a decrease in ISE. Therefore, the first aim of this study was to test the influence of a sad-mood induction on ISE in a group of non-

[7] To clarify, we define mood as being "longer, slower moving" as opposed to affect that refers broadly to valences, and emotion that refers to the valence of the feeling in response to a given stimuli/situation (Rottenberg & Gross, 2007).

clinical participants. To rule out the influence of possible learning effects, we followed a between-subjects design and compared participants who were tested before with those who were tested after a sad-mood induction. As a subsidiary issue, we also examined whether the assessment of ISE is robust against test-retest effects and can reliably be measured twice within a short period of time as was done in previous research (e.g., Franck, De Raedt, et al., 2008). Therefore, participants of the before mood induction condition were also tested after the sad-mood induction. This meant it was possible to examine whether post-mood induction ISE would differ between those who were completing it for the first time, and those who had completed it pre-mood induction.

Explicit self-esteem (ESE) may moderate how much ISE reacts to changes in mood. ISE in those with high ESE was less likely to decrease in response to more negative events in the preceding days than ISE in individuals with low ESE (DeHart & Pelham, 2007). However, given that those with lower ESE were more likely to report more negative events, and subsequently reported more negative affect, it could simply be the differences in mood present at the time of measurement that accounted for differences in ISE. ESE may still moderate the impact of sad mood on ISE, as those with higher levels are more likely to engage in mood repair (Heimpel, Wood, Marshall, & Brown, 2002). If sad mood decreases ISE, then it is feasible that improved moods will increase ISE. As such, a moderating effect of ESE may be observed when looking at the relationship between extent of sad mood and ISE. Therefore, the second aim of the current study was to test whether the impact of sad mood on ISE would be less pronounced for those with relatively high ESE.

If ISE is lowered by sad mood, it may explain how depression and anxiety affect processing at the implicit level (e.g., Beevers, 2005; Risch et al., 2010). Sad mood (and other negative moods) is central to both depression and anxiety (Watson, 2005), and prolonged periods of sad mood, and lack of positive mood, may give rise to lowered ISE. Further, it may also be pivotal to measure ISE following a sad-mood induction in order to detect any relationship between ISE and symptoms of depression and anxiety. Indeed, many have failed to find differences in ISE, in the absence of a sad-mood

induction, between those with a current depression or anxiety and those with no history of the disorders (Franck, De Raedt, et al., 2008), although some have (e.g., Risch et al., 2010). Therefore, given the strength and duration of negative mood in psychopathology, it might be pivotal to measure ISE following a sad-mood induction to detect the relationship with symptoms of depression and anxiety. As a first step to test this final hypothesis, we examined whether the relationship between internalising symptoms and ISE would be most pronounced when ISE was assessed following a sad-mood induction compared to relationships between the constructs in the absence of a sad-mood induction.

All in all, the following main hypotheses were tested: (i) ISE following a sad-mood induction will be lower than ISE in the absence of a sad-mood induction, particularly in those who have low ESE; (ii) symptoms of depression and social anxiety will be related to ISE when the latter is measured following a sad-mood induction, specifically. As a subsidiary issue, we explored the robustness of the ISE assessment for test-retest effects.

Method

Participants

One hundred and five participants took part in the present study, of which ten were dropped (e.g., technical errors). Of the final sample (n = 95; 63.2% female), 87 participants were undergraduate psychology students receiving course credit for participation, and the remaining eight were recruited through social contacts of the research assistants. The average age was 20.54 years (SD = 4.59). Participants were randomized into the experimental condition (n = 49) or the control condition (n = 46). The study was conducted in Dutch. Participants gave consent before taking part in the study, and were fully debriefed at the end.

Measures

Brief Fear of Negative Evaluation Scale II (BFNES; Carleton et al., 2006). BFNES is a self-report questionnaire measuring the extent of anxiety regarding (unfavourable) social judgements from others. Twelve items are

rated on a scale from 1 ("not at all characteristic of me") to 5 ("extremely characteristic of me"). Higher scores were reflective of more fear of negative evaluation. Missing answers were replaced with the mean for that individual (n = 4). Internal consistency was excellent (Cronbach's α = .96).

Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). The LSAS is a 24-item self-report measure and was used as a measure of social anxiety. For each item, participants rate how much they feel anxious or fear (from 0 "none" to 3 "severe"), and how much they try to avoid (from 0 "never" to 3 "usually") typical social scenarios (e.g., "making a call in a public place"). Total scores were used, with higher scores being indicative of more social anxiety. In the present sample, excellent internal consistency was observed (Cronbach's α = .93).

Centre for Epidemiologic Studies Depression Scale (CES-D; Bouma, Ranchor, Sanderman, & Sonderen, 1995; Radloff, 1977). Symptoms of depression were measured using the 20-item self-report CES-D. The prevalence of depressive symptoms in the past week was answered on a four-point scale from 0 ("rarely/never [less than one day]") to 3 ("often/always [5-7 days]"). Higher scores were indicative of more depressive symptoms. Missing items were replaced with the mean for that individual (n = 5). The CES-D showed excellent internal consistency (Cronbach's α = .94).

Implicit Association Test (IAT; Greenwald & Farnham, 2000). Implicit self-esteem was measured with a computer-based word-sorting task. Per trial, a single word was sorted using a two-buttoned response box that belonged to one of four categories: two target categories (*I* vs. *Other*; e.g., "me", "them") or two attribute categories (*positive* vs *negative*; e.g., "valuable", "useless"). The IAT has been described in full previously (Greenwald & Farnham, 2000). In short, the premise for the IAT is that those with higher ISE will sort words faster in the block where *I* and *positive* words (and *other* & *negative)* share the same response key than in the block where *I* and *negative* words (and *other* & *positive*) share the same response key. IAT scores were calculated using the D4 measure (Greenwald et al., 2003). IAT scores were voided when more than 10% of trials were faster than 300ms, more than 20% were answered wrongly, or more than 1% of trials

were longer than 10,000ms (neutral mood IAT: n = 3, sad mood IAT: n = 6). Spearman-Brown corrected correlations between test halves were .80 and .87 for the control condition, and .80 for the experimental condition. Higher scores were indicative of more positive ISE. The IAT given at baseline to approximately half of the participants (control condition) only differed to the IAT given to all participants following the sad-mood induction (both conditions) in the fixed random order of words[8]. Specific words, category labels and order of pairings were the same across all IATs.

Rosenberg Self-Esteem Scale (RSES; Franck, De Raedt, Barbez, & Rosseel, 2008; Rosenberg, 1989). The self-report RSES was used as a measure of explicit self-esteem. Participants answer 10 items on a 4-point Likert scale (from 1 "strongly agree" to 4 "strongly disagree"). Scores were reversed so that higher scores were indicative of higher self-esteem. The present sample showed high internal consistency (Cronbach's α = .88).

Mood Induction. For the sad-mood induction, participants were asked to write about a time when they were sad on the paper provided. While they wrote, "Theme from Schindler's List" (composed by John Williams, released 1994) was played through the headphones. A similar procedure has been adopted successfully before (Franck, De Raedt, et al., 2008; Gemar et al., 2001). The music was slowed to three quarter speed using Audacity[R] (v 2.0.2) and played for approximately 5 minutes. Participants were instructed to write for the full duration of the time, and that they could take what they wrote home with them. A similar happy-mood induction was given at the end of the study in order to reverse any sad mood that had been induced.

Visual Analogue Mood Scales (VAMS). Sets of four mood scales measuring sad, happy, calm and irritable on a horizontal line were given throughout the study. Participants clicked on a horizontal line (132 mm) anchored from Not (e.g., "Not happy") to Very (e.g., "Very happy") using the

[8] Previous studies usually present IAT stimuli in a random order (Franck et al., 2008; Gemar et al., 2001), and as such the order of words is assumed not to influence the IAT effect. Given that the previous studies involving a sad-mood induction did not involve identical order of words in the IATs before and after, the present study also mixed the order of the words across the two IATs.

mouse. The nearest hundredth was recorded (possible range 0 – 100). Scores on the sad VAMS are used in the present analysis to check whether the sad mood induction was effective, and that this effect remained for the duration of the IAT. For the correlational analyses, the influence of the mood manipulation was calculated by subtracting VAMS sad-mood score pre-mood induction from scores post-mood induction, with higher scores indicative of a greater increase in sad mood.

Other measures. The measures reported above are relevant for the analysis presented in this paper. Other questionnaires and measures were included, and information can be provided upon request. The IAT was the first before the sad-mood induction (control condition), and the first measure following the sad-mood induction. VAMS were given directly before the IATs, and the final VAMS was given directly after the (second) IAT.

Procedure

First, both conditions completed a set of VAMS (baseline mood). Those in the control condition then completed the IAT (i.e., measured during a relatively neutral mood). Both conditions then completed the self-report questionnaires via Qualtrics. Both conditions then completed a set of VAMS (pre-mood induction), the sad-mood induction, and another set of VAMS (post-mood induction). Following the sad-mood induction, both conditions completed the IAT, and then a set of VAMS (final score). As such, the only difference between the control and experimental condition was the inclusion of a baseline IAT in the control condition.

Results

Descriptives

Means and standard deviations of the measures used, split by group, are presented in Table 6.1. Applying a Bonferroni correction (adjusted $\alpha = .0125$), scores on the RSES, CESD, LSAS and BFNES did not differ between the two conditions.

Table 6.1
Means and Standard Deviations, and Independent T-Tests between Groups per Variable

	Experimental (n = 49)	Control (n = 46)			
	M (SD)	M (SD)	*t*	*p*	Cohen's *d*
IAT neutral mood	-	.52 (.38)	-	-	-
IAT sad mood	.58 (.33)	.30 (.42)	3.55	<.001	0.76
RSES	30.49 (4.60)	29.65 (5.03)	0.85	.40	0.18
CES-D	11.09 (9.17)	15.11 (11.19)	1.92	.06	0.40
LSAS	31.63 (16.31)	36.74 (19.94)	1.37	.17	0.28
BFNES	32.23 (11.76)	36.60 (13.39)	1.70	.09	0.35

Note: IAT = implicit association test (self-esteem); RSES = Rosenberg's Self-Esteem Scale; CES-D = Centre for Epidemiologic Studies Depression Scale; LSAS = Liebowitz Social Anxiety Scale; BFNES = Brief Fear of Negative Evaluation Scale II.

Sad Mood Manipulation Checks

Square-rooted sad mood score at baseline, before the sad mood induction, following the sad mood induction, and following post-mood induction IAT are presented in Table 6.2 by condition. In order to see whether the sad-mood induction was effective an independent t-test was conducted to compare sad mood before the IAT between the conditions. Scores were square-rooted to correct for skewness before the analyses was run. Supporting the efficacy of the current sad mood manipulation, results showed that those in the experimental condition were sadder before completing the IAT (M = 6.67, SD = 1.83; raw mean score = 47.78, SD = 22.85) than those in the control condition before completing the baseline IAT (M = 3.50, SD = 2.33; raw mean score = 17.52, SD = 20.53), t(93) = 7.41, p<.001, d = 1.51. A 2 (condition) by 2 (time; pre- and post- sad-induction) ANOVA indicated that while there was an increase in sad mood following the induction, F(1, 93) = 155.86, p < .001), this did not differ between conditions, F(1, 93) = .05, p = .83. As such, the manipulation was considered equally

successful for both conditions.[9] Furthermore, a greater effect of the sad-mood induction (i.e., change scores) was related to lower ESE (r = -.33, p<.001), and more depressive symptoms (ρ = .26, p = .01), but not more symptoms of social anxiety (LSAS; r = .20, p = .051), nor fear of negative evaluation (BFNES; r = .14, p = .18) across all the participants (n = 95).

Table 6.2

Means and Standard Deviations of Square-Rooted Sad Mood Scores across Study

	Experimental (n = 49)	Control (n = 46)
Baseline	3.10 (2.18)	3.50 (2.33)
Pre Sad-Mood Induction	4.07 (2.43)	4.27 (2.46)
Post Sad-Mood Induction	6.67 (1.83)	6.96 (1.98)
Post Sad-Mood IAT	4.79 (2.23)	5.19 (2.62)

Note: IAT = implicit association test (self-esteem).

IAT Test-Retest Effects

In order to look at test-retest effects, IAT scores following the sad-mood induction were compared between the two conditions. An independent t-test indicated that those in the control condition had significantly lower scores than those in the experimental condition, t(87) = 3.55, p = .001, d = 0.76 (Ms & SDs in Table 6.1, see sad IAT). Results were not explained by a difference in sad mood before the IAT between conditions, t(93) = 0.92, p = .36, d = 0.19, and nor by the effectiveness of the sad-mood induction (see previous manipulation checks). Test-retest reliability between IAT scores at baseline and following a sad-mood induction was significant but unacceptable, r (41) = .40, p = .01. This may be because of the sad-mood

[9] A repeated-measures ANOVA in the experimental group comparing square-rooted sad-mood scores before the sad-mood induction (M = 4.07, SD = 2.43), after the sad-mood induction (M = 6.67, SD = 1.83) and after the IAT (M = 4.79, SD = 2.23) revealed a significant difference, F(2, 47) = 40.40, p<.001. Repeated contrasts revealed the sad mood induction to be effective, F(1,48) = 68.81, p <.001, with evidence of some mood repair during the IAT, F(1,48) = 68.75, p<.001. A repeated measures t-test revealed that sad mood following the IAT was still higher than before the sad-mood induction, t(48) = 3.08, p = .003, d = .31.

induction. As a test-retest effect could not be rejected, IAT scores following the sad-mood induction in the control condition were not involved in any further analysis.

Table 6.3
Spearman's Rho (CES-D) and Pearson's Bivariate (LSAS, RSES & BFNES) correlations with IAT scores

	IAT sad mood (Experimental; n = 48)		IAT neutral mood (Control; n = 43)	
	r/r_s	*p*	r/r_s	*p*
RSES	.16	.27	.18	.26
CES-D	-.14	.35	-.10	.53
LSAS	-.07	.62	-.08	.63
BFNES	-.30	.04	-.02	.90

Note: IAT = implicit association test (self-esteem); RSES = Rosenberg's Self-Esteem Scale; CES-D = Centre for Epidemiologic Studies Depression Scale; LSAS = Liebowitz Social Anxiety Scale; BFNES = Brief Fear of Negative Evaluation Scale II.

The Influence of Sad Mood on Implicit Self-Esteem

First an independent t-test revealed no significant difference in IAT scores between both groups, $t(89) = 0.85$, $p = .40$, $d = 0.17$, 95% CI [-0.25, 0.59]. Subsequent correlational analyses within the experimental condition indicated that there was no significant correlation between the influence of the sad-mood induction (based on change scores) and IAT scores, Spearman's rho (47) = -.05, $p = .74$, nor were sad mood scores directly before the IAT correlated with IAT scores, Spearman's rho (47) = -.19, $p = .20$. As such, (extent of increase in) sad mood was not related to ISE.

To increase the sensitivity of the analysis, we repeated the analysis without participants for whom the sad-mood induction appeared not to be effective based on decreases in sad mood (n = 6) and relatively low scores (< 20; n = 6). Likewise, a number of participants in the control condition reported relatively high levels of sad mood (i.e., > 40; n = 6) and were excluded. Increasing the saliency of sad and relatively neutral moods in the experimental and control conditions, respectively, did not change the results; there was no difference between conditions in IAT scores, $t(72) = .46$, $p = .64$, $d = 0.12$ (M = .54, SD = .33 vs. M = .58, SD = .31, experimental and control conditions, respectively).

In order to see whether explicit self-esteem may moderate the effect of sad mood on ISE, a hierarchical regression was conducted. With IAT scores as the dependent variable, and condition, RSES scores, and the interaction between condition and RSES scores as independent variables, the resulting model was not significant, $R^2 = .04$, $F(3,87) = 1.11$, $p < .35$. Neither RSES (B = 0.01, SE = .01; $p = .22$), nor condition, (B = 0.02, SE = .32; $p = .94$), nor their interaction (B = 0.002, SE = .02; $p = .92$) explained variance in IAT scores.

Correlations between IAT scores and self-report measures are presented in Table 6.3. Spearman's Rho correlations were calculated for CES-D because of skewed scores. IAT scores in the control condition were not related to scores on the BFNES, while IAT scores in the experimental condition were significantly related to BFNES scores. However, applying a Bonferroni correction to adjust for eight correlations (adjusted $\alpha = .00625$) implied that the correlation does not meet statistical significance.

Discussion

The key findings of the present study can be summed as follows: i) Participants in a sad mood do not have lower ISE than those in a relatively neutral mood; ii) Explicit self-esteem did not moderate the influence of sad mood on ISE; iii) Administering the IAT twice over a relatively short period of time seem to lead to test-retest effects.

In the present study, there was no evidence that sad mood influences ISE, suggesting that lower levels of ISE may not manifest through a decrease in mood. While it is feasible that more persistent and intense sad mood is required before a decrease in ISE is observed, there are other theories as to how low ISE manifests. Beevers (2005) highlighted how repetitive consistent processing at the explicit level becomes increasingly more ingrained and automatic with time. This would suggest that prolonged low ESE would lower ISE. DeHart, Pelham and Tennen (2006) argued that those who reported more negative interactions with their parents during their childhood also showed lower ISE currently (i.e., in adulthood), while those who reported more nurturing and caring parents had higher ISE. Indeed, disruptions during childhood may have an important role in how ISE is formed, given that in adults with a current depression, those with

childhood-onset had lower ISE than those with adulthood-onset (Rodebaugh, Weeks, Gordon, Langer, & Heimberg, 2012). The present study suggests that ISE is stable and appears resistant to increases in sad mood, and further research is needed to understand how ISE manifests.

Implicit self-esteem during a relatively neutral or following a sad-mood induction was not related to symptoms of depression. Results may be very different in a clinical population, and the influence of sad mood might even be different in those who have a history of psychopathology and are currently symptom-free. While current levels of depression and social anxiety were measured, we did not control for previous depression and social anxiety. This may be pivotal, as in those with a history of depression specifically, a sad mood is thought to trigger dormant depressive processing (Gemar et al., 2001). Indeed, those with more depressive symptoms showed a relatively large increase in sad mood following the induction). As allocation to condition is random, we assume that the number with a history of psychopathology was similar across groups. Regardless, repeating the study in a population with more variation in depression and anxiety symptoms may produce different results.

The present study observed test-retest effects when two IATs were given over a relatively short period, as IAT scores differed to those who were completing the IAT for the first time. Test-retest effects have also previously been observed in other versions of the IAT (Robusto, Cristante, & Vianello, 2008). Therefore, it is feasible that a second IAT is less sensitive to measuring self-esteem even when several of self-report questionnaires and other tasks separate it from the first IAT. It is unlikely that the differences can be explained by the presence of a sad-mood induction, as manipulation checks would suggest that the effectiveness of the sad-mood induction was similar between conditions. This has important consequences for previous studies who gave multiple measures of IAT with, perhaps, fewer other measures in-between (Franck, De Raedt, et al., 2008; Gemar et al., 2001), as reduced implicit self-esteem following a sad-mood induction might have been tainted by test-retest effects. Given that scores already differed before the mood induction (Franck, De Raedt, et al., 2008), there may have been more room for scores to drop in the previously depressed group, and as such, the

extent of test-retest effects may have differed between groups. Future studies aiming to compare remitted depression and never depressed individuals on ISE following a sad-mood induction may be better adopting a pure between-groups design, or introducing more tasks and time between IAT measurements. It is of course feasible that those in the experimental group had genuinely higher implicit self-esteem than those in the control group, and replication is needed. Further, future studies should consider employing a four-group design (absence/presence of pre-test IAT x presence/absence of sad-mood induction) for a more accurate analysis of the test-retest effects of the IAT and the potential influence of the sad-mood induction herein.

A number of limitations need to be addressed. First, while higher sad moods were reported after the sad-mood induction compared to sad mood reported at baseline, sad mood was low considering that the scale went to 100. Further, given the obvious intention of the sad-mood induction, it is also possible that some participants were inclined to rate their sad mood higher than what they felt due to demand characteristics. As such, the lack of findings in the present study may be attributable to the somewhat mediocre effect of the sad-mood induction. However, a slight increase in sad mood might be more representative of daily changes in mood in the absence of a specific life event. Finally, in the absence of a Bayes Factor analysis, it is not possible to make inferences concerning the lack of difference observed between the IAT scores in the experimental condition and IAT scores at baseline in the control condition. A post-hoc power analysis indicated that for the observed effect size (0.17) and the sample sizes, the power was .20 (alpha = 0.05). As such, replication with larger samples is necessary.

The present study did not find support for the influence of sad mood on implicit self-esteem as measured by the IAT, in a student sample. This would suggest sad mood does not provide a context where certain (negative) associations are more likely to be triggered when the self is activated. In conclusion, although ISE seems relatively insensitive to differences in sad mood, the assessment of ISE seems relatively sensitive to test-retest effects.

7. General Discussion

Recap of Aims and Findings

The present thesis aimed to look at the role of self-concepts within anxiety and depression. Specifically, implicit, explicit, discrepant, and stable self-esteem were analysed in (symptoms of) anxiety disorders, depressive disorder, and a combination of anxiety disorders and depressive disorder (i.e., comorbidity). Further, the presence of continued lowered self-esteem into remission and recovery phases of an anxiety disorder and depressive disorder was also explored. The validity of more specific self-depressed associations (SDA) in predicting depression recurrence and relapse was analysed, continuing earlier findings that highlighted relatively stronger levels of SDA in remitted depression. The final empirical chapter was an experimental study looking at the influence of sad mood on implicit self-esteem.

Implicit self-associations (implicit self-esteem and implicit self-depressed association) frequently lacked evidence of being related to depression and anxiety in the present thesis. Levels of implicit self-esteem (ISE) were not prognostically related to symptoms of social anxiety and depression in relatively healthy adolescents two years later (chapter 2). In addition, there was no evidence of a difference in levels of ISE between the comparison group and current, remitted and recovered individuals with an anxiety disorder or depression. However, those who had concurrent depression and anxiety (comorbidity) did have lower ISE than the comparison group (chapter 3). There was no evidence that implicit SDA in remittance and recovery from MDD were related to (time to) recurrence (chapter 5). Finally, there was no evidence that those who had received a sad-mood induction displayed lower ISE than those who had not (chapter 6). As such, except for comorbid depression and anxiety, there was consistently a lack of evidence that self-evaluations at the more automatic and less controllable level were related to depression and anxiety.

Consistent across chapters, explicit self-associations (explicit self-esteem and explicit self-depressed associations) were found to be related to depression and anxiety. Low ESE was related to more symptoms of social anxiety and depression in adolescents two years later (chapter 2). A

comparison group had higher ESE than individuals with MDD, anxiety disorders (AD), and comorbid MDD/AD. This was true for those who currently had the disorder, were remitted from the disorder, and (for MDD and AD separately) who had recovered from the disorder (chapter 3). Finally, explicit SDA shortly following an MDD episode predicted (time to) recurrence (chapter 5). As such, dysfunctional self-related processing at a more conscious level was consistently found to be involved in depression and anxiety.

Furthermore, the results of the studies described in this thesis showed that low self-esteem stability was present in depression and anxiety, with no evidence to support a presence of self-esteem discrepancy between ISE and ESE. Participants with no history of depression and anxiety had more stable self-esteem than those with a current or remitted MDD, AD, and comorbid MDD/AD. Further, those who had recovered from MDD or AD still reported less stable self-esteem than those without a history of depression and anxiety. However, once controlling for ESE, statistical testing failed to support a difference in self-esteem stability between the comparison group and comorbid MDD/AD at both the current and remitted level. Differences between the comparison group and current/remitted/recovered MDD, and current/recovered AD were still supported (chapter 4). Evidence for the role of discrepant self-esteem (i.e., quantified either by the interaction or the difference between ISE and ESE) in depression and anxiety was consistently lacking. An interaction between ISE and ESE did not explain additional variance in social anxiety or depressive symptomatology two years later in healthy adolescents (chapter 2). Variables based on difference scores suggested that in both directions (i.e., ESE > ISE, and ISE > ESE), discrepant self-esteem predicted current MDD and current AD from relatively healthy controls. However, these effects disappeared once ESE was included in the model (chapter 3). As such, support for the role of self-esteem stability in depression and anxiety was found, while support for the role of self-esteem discrepancy was lacking.

Implicit, explicit, discrepant, and stable self-esteem within the context of depression and anxiety are discussed in the main body of this final chapter. For each facet of self-esteem, explanations of the findings,

particularly where those conflict with previous studies, limitations and future directions are covered. Depression and anxiety more generally are discussed, with a focus on several key aspects that may influence the interpretation of the findings in this thesis. Possible future directions are postulated before, finally, deriving at a conclusion concerning *Self-Esteem in Depression and Anxiety: Implicit, Explicit, Unstable, and Discrepant.*

Implicit Self-Esteem (ISE)

The previous findings concerning implicit self-esteem in depression and anxiety are few and mixed. Some studies found lowered levels (compared to a comparison group) in depression and anxiety (e.g., Risch et al., 2010), while others failed to find any differences (e.g., Lemmens et al., 2014). Common across all these studies using clinical samples are the limited sample sizes. This is particularly problematic as small effect sizes are anticipated due to different method-related error variance involved in reaction-time based computer measures (e.g., IAT) versus self-report measures of symptomatology and structured clinical interviews. As such, the power needed to detect an association between measures that have different method-related error (i.e., the IAT and symptomatology) is higher than the power needed to detect associations between measures that have similar method-related error (i.e., ESE and self-reported symptomatology). As such, limited sample sizes in previous studies may explain why effects were detected for ESE, but not for ISE. In both chapters 2 and 3, relatively large sample sizes were used to look at the longitudinal relationship between ISE and symptoms of depression and social anxiety in healthy adolescents, and differences in ISE between clinical groups of depression, anxiety and comorbidity. Using large sample sizes, results suggest that ISE was not related to symptoms of depression or social anxiety in either direction in healthy adolescents, and that those with a current depression or anxiety, on average, did not have lower levels of ISE than a never clinically depressed or anxious comparison group in adults. As such, the lack of positive findings in previous studies is unlikely to be explained by the lack of power to detect small effects.

Another shortcoming of previous studies that was addressed in chapter 3 was differentiating between comorbidity and more singular forms of either depression or anxiety. For example, if low ISE is unique to depression, then the failure of excluding those with a comorbid anxiety in previous studies may explain the null findings. Given that low self-esteem ("feelings of worthlessness") is a DSM criterion for depression, but not necessarily anxiety, it is feasible that low ISE might be unique for depression. Particularly as models of dual processing would suggest that low ISE develops through persistent low ESE (Beevers, 2005). Alternatively, given that those with comorbid depression and anxiety tend to have more severe and persistent symptomatology, it is also feasible that those with a comorbid depression and anxiety have lower ISE, particularly when ESE is also lower. Indeed, the latter was observed in chapter 3. However, it is still pivotal to see whether the presence of both depression and anxiety resulted in lowered ISE, or whether lower ISE in the comorbid group stemmed from longer periods of stable low ESE. As repetitive explicit associations become increasingly ingrained (i.e., more implicit; Beevers, 2005), it may simply be those with more persistent, severer symptoms who have low ISE, rather than those who have both symptoms of anxiety and depression. Further research could address this by controlling for the number of months with (severe) symptoms.

There are a number of limitations to the IAT which have already been covered to some extent in the discussion section of chapter 3. Criticisms of the self-esteem IAT include the low convergent validity with other ISE measures and the lack of clarification of how self-concepts and attributes are related. Remue and colleagues (2013; 2014) addressed this latter point by differentiating between actual ISE (i.e., "I am") and ideal ISE (i.e., "I want to be") using the Implicit Relational Assessment Procedure (IRAP). Results from differentiating between these concepts suggested that high dysphoric students had higher ideal ISE and lower actual ISE than a low-dysphoric comparison group. Therefore, the lack of clarification between how the concepts are related (i.e., actual or ideal) may explain the mixed and lack of findings concerning ISE. However, the IRAP involves the presence of "I am" or "I want to be" on the screen during the task, and it is possible that such

primes may trigger explicit processing to some extent, particularly when followed by an attribute. Further, the somewhat more obvious nature of the task may also introduce demand biases (e.g., presentation bias). However, that is not to say that the IRAP fails to capture a more realistic ISE than the IAT. Perhaps a completely implicit self-esteem that is devoid of any explicit aspect is unrepresentative of what happens in day-to-day life.

It is crucial to remember that ESE and ISE are polar ends on the same spectrum, and that a given evaluation falls on other overlapping spectrums too. In forgetting this, the aim of many researchers is to capture "clear-cut, all-or-none" constructs (Moors & De Houwer, 2006). Indeed, measures of implicit attitudes often aim to capture evaluations at the most implicit end of this spectrum, while totally implicit evaluations may rarely occur. Often the example of driving a car is provided to differentiate between implicit and explicit processing (e.g., Beevers, 2005). While driving is effortful and takes up much cognitive resources when first learning to drive, driving becomes increasingly less conscious and more automatic with time and practise (e.g., changing gears without deliberating where the gear is), thereby taking up less cognitive resources. However, it would be surprising if driving became completely non-conscious and automatic, and cognitive resources are still likely to be used, albeit to a lesser extent. Likewise, while repetitively evaluating the self negatively might make access to negative propositions easier and quicker, thereby being less conscious of the processes involved or even how a negative self-evaluation is derived (i.e., more implicit), it is unlikely that there is no degree of consciousness involved, and that no cognitive resources are involved. Future studies should aim to capture self-esteem at several points along the spectrum of awareness, automaticity and effort, as relatively implicit self-evaluations may still play a role in depression and anxiety if captured at a more ecologically valid level. The IAT is highly likely to measure a very implicit self-esteem as there is very little subjective evaluation involved. This contrasts with, for example, the Name Letter Task (Nuttin, 1985), an alternative ISE measure, where participants are asked to evaluate the attractiveness of each letter, and many participants are able to guess the nature of the task (Krizan, 2008). This task may capture ISE closer to ESE on the spectrum than the IAT.

Explicit Self-Esteem (ESE)

Explicit self-esteem has consistently been shown to be lower in both anxiety and depression (e.g., Sowislo & Orth, 2013). In chapter 3 it was explicitly tested whether there were differences in self-esteem between those with a depressive disorder and those with an anxiety disorder. Results suggest that there was no support of a difference in self-esteem between the two disorders. As such, an intervention targeting self-esteem may have transdiagnostic applicability given that self-esteem is equally low in depression and anxiety. Indeed, one such intervention, Competitive Memory Training (COMET, Korrelboom et al., 2012), already seems to be effective in reducing depressive symptoms when combined with treatment as usual. COMET was also found to be effective in panic disorder as an add-on therapy (Korrelboom, Peeters, Blom, & Huijbrechts, 2014). More common interventions, like Cognitive Behavioural Therapy, often include aspects that involve challenging negative self-thoughts. However, whether self-esteem interventions like COMET are effective as stand-alone therapies, and whether self-esteem related aspects of more common interventions are key processes of change, remains an important question. If the answer is yes, then given the high rates of co-morbidity, low self-esteem may highlight an important symptom to target, and might be a cost-effective approach to reducing both depressive and anxiety symptoms. Further research is required to see whether the effects of COMET hold as a stand-alone treatment.

It is important to note that most measures of ESE are self-report questionnaires, and indeed, self-report measures of ESE were used throughout this thesis. Argued in chapter 3 is that self-report measures of ESE are not perfect measures of self-esteem, with which other self-esteem measures (e.g., ISE indices) must compete with to prove validity and reliability (Buhrmester et al., 2011). Shortcomings of self-report measures include measurement error common across self-report questionnaires more generally that overinflate relationships, and self-presentation biases. As such, many argue that findings should be replicated using behavioural measures of self-esteem which may limit the influence of self-presentation biases. Ideally a behavioural measure of ESE will involve measuring the behaviour of a participant that is thought to reflect solely self-esteem while minimizing

the influence of other processes. However, if self-esteem acts as a filter that influences the way external information is processed, and how the self is placed in the environment (Zeigler-Hill, 2013), then it is likely to influence most processes. ESE is not a process in itself, but an evaluation. Where other evaluations are likely to influence processes specific to that evaluation (e.g., one who holds a sexist opinion will process gender-relevant information in a biased way that is consistent with opinion and also enforces this opinion), ESE may be a unique evaluation as it can be relevant in all contexts and also influence other more specific evaluations. It is feasible that the construct of self-esteem is too abstract and broad to be captured completely in one self-report or behavioural measure.

What exactly causes low ESE remains an important question. ESE definitions usually involve words like conscious, purposeful, and controllable – but if this were the case, then it somewhat implies that low ESE can be increased by simply altering the content of our thoughts (e.g., thinking ourselves positive; however, see Hulme, Hirsch, & Stopa, 2012). While low self-esteem can have a rational explanation (e.g., a break-up, loss of a job), there often seems to be an inconsistency between reported self-esteem and actual abilities, achievements, etc. This points to a dysfunction in processing the available information, and is somewhat reminiscent of cognitive biases that have been researched extensively in both depression and anxiety. Theory postulates that anxiety and depression results in processing biases that favour threatening or negative information. These biases have been observed in attention, interpretation, and memory (e.g., Hallion & Ruscio, 2011; MacLeod, Mathews, & Tata, 1986). Negative thoughts persist and are prevalent as a result of increased attending to negative information, the tendency to interpret ambiguous scenarios as negative, and the relative ease with which negative information is recalled. However, many have looked at the direct relationship between the presence of these biases and psychopathology, while ESE may be a feasible mediator in these relationships. For example, if one is quick to attend to negative facial expressions like disgust, in comparison to a positive face (attention), and is also likely to interpret successes as luck and failures as incompetence (interpretation), and is more likely to remember the one time they failed an

exam rather than the several times they received a top mark (memory), then it is feasible to anticipate that negative self-related propositions will enter the mind. In such a scenario, while the resulting self-esteem propositions (e.g., "I am stupid") are explicit in that the individual is aware of these thoughts and may actively think further about them, the processes that triggered these thoughts might be relatively implicit (i.e., non-controllable and requiring little effort). This would explain why low self-esteem thoughts cannot simply be thought away. There is some support for this theory in that those with low self-esteem have been shown to have a dysfunctional interpretation bias (Tafarodi, 1998), attentional bias (Li & Yang, 2013), and memory bias (Ross & Wilson, 2002; see Tafarodi, Marshall, & Milne, 2003, for a review of the models concerning self-esteem and memory). However, this has often been interpreted to be the result of low self-esteem, rather than the cause. Future research should manipulate these biases to see whether this changes ESE. Further how these processes relate to ISE would also be of interest. It could be that low ISE leads to these biases by processing in a ISE-congruent way.

Discrepant Self-Esteem

Consistent in chapter 2 and 3 was the inability to reject the null hypothesis concerning the association between self-esteem discrepancy and (symptoms of) depression and anxiety. Previous research suggests that there are two common methods to quantifying self-esteem discrepancy. In one method, the interaction between ISE and ESE is included in the analysis, and, when statistically significant, plotted to differentiate between high/low ESE and high/low ISE (chapter 2). Indeed, this taps into the idea that specific combinations (i.e., high ESE and low ISE; low ESE and high ISE) may put an individual at risk for dysfunctional outcomes (e.g., depressive attributional style; Schröder-Abé et al., 2007), as it may represent a vulnerability that is pronounced when explicit processing cannot be engaged to overrule initial negative implicit processing (e.g., during times of stress; low ISE and high ESE), or the tendency for explicit processing to result in negative conclusions despite initial positive implicit processing (e.g., high ISE and low ESE). Another approach employs the method outlined by Briñol and colleagues (2006), where the absolute difference is calculated between standardized

measures of ISE and ESE, and the direction of the discrepancy is represented by a dummy variable. Although theoretical underpinnings are similar to those who use the interaction method, it has also been argued that larger differences result in more (implicit) self-doubt (Briñol et al., 2006). Further, that those with larger differences are more inclined to try and resolve the discrepancy by potentially dysfunctional means (e.g., rumination, Phillips & Hine, 2016), and are more vulnerable to external information related to this discrepancy (Briñol et al., 2006). Advocates of this method argue that the interaction approach fails to acknowledge the importance of the direction of the discrepancy independent of how large the discrepancy actually is (Leeuwis et al., 2015). However, both methods have methodological shortcomings, and a number of theoretical questions remain.

One issue of using the interaction method in linear regression analyses (as is often done; e.g., Schröder-Abé et al., 2007) is that the assumption of a linear relationship between dependent and independent variable is violated. The range of values when creating an interaction variable are likely to have congruent low self-esteem at one end (i.e. low ESE score x low ISE score) and congruent high scores at the other (i.e., high ESE score x high ESE score). Indeed, discrepancy scores where, for example, ESE is high and ISE is low, are likely to fall somewhere in the middle (i.e., high ISE score x low ESE score, or low ISE score x high ESE score). Even if transformation was successful in creating a linear interaction variable, the direction of the discrepancy is also not acknowledged in an interaction value as, hypothetically, high ESE x low ESE may give the same value as low ESE x high ESE. As such, the lack of a significant interaction might not mean that there is no role for discrepant self-esteem in one specific direction.

While the interaction method has both ESE and ISE included as main effects, the method highlighted by Briñol and colleagues (2006) does not. This poses an issue particularly when one type of self-esteem (e.g., ESE) is more strongly correlated with the outcome variable (e.g., symptoms of depression) than the other self-esteem type (e.g., ISE). Previous research and the findings of the present thesis consistently found stronger correlations between ESE and psychopathology than between ISE and psychopathology. While this larger correlation might be an overestimation of the actual

relationship between the constructs because of, for example, shared method variance (i.e., method variance common across self-report measures), it nonetheless influences statistical analysis using difference scores. Previous findings that suggest that damaged self-esteem (low ESE and high ISE), specifically, is related to psychopathology (Creemers et al. 2012, Leeuwis et al., 2015) may simply be an artefact of the relatively strong relationship between ESE and the outcome variable, particularly when the interaction between damaged self-esteem and the extent of discrepancy is observed to be statistically significant (i.e. even lower ESE, and/or higher ISE, is related to more psychopathology). In the original article of this discrepancy method by Briñol et al., correlations between the outcome variables and the variables used to derive at discrepancy variables were not reported. Further, many studies have used the method by Briñol et al. without excluding those who have relatively congruent self-esteem. Those with slightly higher ESE than ISE are then assigned to the damaged category, while the reverse is labelled as having fragile self-esteem (e.g., Briñol et al., 2006; Creemers et al., 2012; Creemers, Scholte, Engels, Prinstein, & Wiers, 2013; Leeuwis et al., 2015). Although the interaction between discrepancy direction and the extent of the discrepancy would somewhat account for this, many studies still draw conclusions over the discrepancy direction independent of the extent of discrepancy (e.g., Creemers et al., 2013). However, the theoretical argument for Briñol et al. method is that larger discrepancies give rise to self-doubt and other dysfunctional processes that aim to reduce discrepancies (Briñol et al., 2006; Phillips & Hine, 2016). As such, it is unclear what, theoretically, discrepancy direction represents when this effect is not quantified by the extent of magnitude. In sum, while the interaction method may fail to capture the direction of the discrepancy, the use of a variable representing discrepancy direction, independent of the magnitude of the discrepancy, lacks theoretical justification. Further, it is doubtful as to whether previous positive findings using the method by Briñol et al. would remain if main effects were taken into consideration.

The issues of variables that are difficult to interpret (direction of discrepancy independent of extent of discrepancy) and the lack of controlling for the relatively large correlation between ESE and the outcome

variable were addressed in chapter 3. Here, variables were created that incorporated both the direction and the magnitude of the discrepancy, and included ESE in the model. These variables technically represent the interaction between ESE and ISE (as quantified by difference scores), split by the direction of difference. Results indicated that both damaged and fragile self-esteem predicted current depression and anxiety from a comparison group (no history of a depression or anxiety disorder), with stronger effects observed for damaged self-esteem (based on Wald test). The latter is consistent with many previous findings (e.g., Creemers et al., 2013). However, with the inclusion of ESE in the model, there was no longer evidence for either discrepancy variable. It is not possible to conclude that the null hypothesis (i.e., self-esteem discrepancy does not play a role in depression and anxiety) is accepted, and it is necessary to repeat these analyses with, for example, Bayes factor. Further, this method also did not allow for inclusion of both main effects (i.e., also ISE).

A number of broad methodological flaws remain in discrepant self-esteem research. Some methods use standardised scores of ESE and ISE (e.g., RSES and IAT) in order to place two very different measures on the same hypothetical scale, thus allowing for the calculation of difference values. The mean then becomes zero and values fall either above or below this value. However, if a sample were recruited who generally have relatively high ESE and relatively low ISE (in other words, fragile self-esteem is already evident in the whole group), then a large portion of the sample will erroneously be labelled as having damaged self-esteem. Using z-scores to derive at discrepancies would only be appropriate if the whole group represented a sample from the general population. Combining clinical and control groups (as done in chapter 3) is likely to over represent the clinical disorder where ESE is consistently found to be low. Further, a normal variance in score needs to be evident in both ESE and ISE as skewed data will result in distorted means. Skewed scores on ESE are often evident in (relatively) healthy samples. One way to create discrepancy groups would be to apply pre-determined cut-offs to ISE and ESE measures thereby highlighting who has high and who has low self-esteem, independent of the mean group self-esteem. Not only would establishing suitable cut-offs be difficult, but

applying cut-offs to IAT scores has been criticised based on the random variation which is incorporated in some scoring methods (including the scoring method used in this thesis; Blanton, Jaccard, & Burrows, 2015).

Another broad flaw of self-esteem discrepancy research is that measures of ESE and ISE often do not only differ on the construct they seek to capture. As highlighted in the discussion of chapter 2, the timing and context of the IAT and RSES also differ. While RSES, arguably the most common measure of ESE, asks participants about their level of self-esteem over the last week, the IAT, and most other performance-based tasks, measures ISE at that moment. Also, context has been argued to be an important influencing factor of implicit associations (Gawronski & Bodenhausen, 2006; although see chapter 6 where sad mood was not an influencing context), and therefore the context of a lab and being "tested" might be too specific, compared to the context imposed on measures of ESE, which allow participants to reflect back to more real-life occurrences. Finally, method variance that is associated with self-response measures (e.g., response bias), and method variance associated with reaction time-based tasks differ. Combining measures may therefore create a score including a lot of method variance.

Therefore, the conclusion of this thesis, and based on previous findings, is that we currently do not have the means to adequately test discrepant self-esteem. Further, the potential relevance of such a construct also requires further justification, as implicitness and explicitness of evaluations would be better considered as being opposites on a single spectrum (Moors, 2014). As such an evaluation at a single moment is likely to fall somewhere on the spectrum, rather than being concurrently at the implicit and at the explicit end. There may be an initial implicit evaluation, but with motivation and time, the evaluation may become increasingly more explicit (i.e., more conscious, purposeful, and, potentially, malleable). Once it reaches awareness, initial implicit evaluations become a proposition (e.g., gut-feeling) involved in explicit processing. Indeed, propositions may conflict with one another, and result in doubt. Therefore, not only the methodological shortcomings of self-esteem discrepancy need further research, but also the theoretical underpinnings of discrepancies between

more implicit evaluations and more explicit evaluations need further clarification. Indeed, it is currently unclear as to when such a discrepancy may occur, and what it actually means. It is feasible that discrepancies between propositions at the explicit level do result in indecision, doubt, and potentially, self-esteem instability.

Self-Esteem Stability (SE-S)

The relative stability of self-esteem in comorbid depression and anxiety, and lack of self-esteem stability in singular forms of depression and anxiety found in chapter 4 may explain the findings concerning ISE found in chapter 3. Specifically, that lower ISE was observed in comorbid depression and anxiety, and not singular forms of depression and anxiety disorders. Indeed, it is assumed that low ISE forms through consistently evaluating the self negatively at the explicit level. The more often this occurs, the quicker and easier the negative propositions are recalled, thus becoming increasingly implicit (e.g., Beevers, 2005). However, if ESE is not particularly stable, then it is unlikely that consistent ISE forms. This is reflected in the findings of chapter 3 and 4 in that groups who displayed less stable self-esteem (once controlling for differences in ESE; e.g., current depression, current anxiety) did not show lower ISE, while low ISE and relatively stable low ESE were observed in current comorbidity. Indeed, an important question then arises: if ESE is not stable, what determines self-evaluations when more explicit forms of processing cannot be engaged (i.e., limited cognitive resources)? Research has long observed increased self-focused attention in clinical disorders (e.g., Ingram, 1990), and may highlight continued processing at the explicit level (e.g., rumination, worry), thereby suggesting that ISE plays a minimal or no role in behaviour or mood. This might even go on to explain some of the somatic symptoms of depression and anxiety, like poor concentration and sleep disturbances, that may be explained by drained or otherwise-occupied cognitive resources. This does not mean that because self-esteem is consistently lower in comorbidity that there is less self-focused attention, but perhaps that the low ISE observed in comorbidity has little practical significance if ESE is often engaged. Indeed, further research is required to see whether the low ISE in co-morbidity actually predicts anything (e.g., treatment resistance) over and above low ESE. The higher

resilience to treatment and remittance in comorbidity, relative to singular forms of depression and anxiety (Penninx et al., 2011), may also be explained by the even lower and stable ESE rather than the presence of low ISE.

As with all facets of self-esteem discussed thus far, there are a number of flaws to the way self-esteem stability has been measured, both in previous research and in this thesis. As highlighted in the discussion of chapter 4, both self-report measures of self-esteem stability at a single moment and looking at variation in scores across multiple measures of ESE has limitations including self-report biases and lack of possible variation for those who score consistently high or consistently low. The definition of self-esteem stability is the extent and frequency of short-term self-esteem fluctuations (e.g., Roberts & Monroe, 1994). Also, most definitions highlight that fluctuations occur in response to other factors (e.g., mood states; Clasen et al., 2015), suggesting that fluctuations are not necessarily random. Presumably, extent and frequency are related to one another, in that if self-esteem is very responsive to changes in mood, it will also fluctuate more often. If this is indeed the case, then self-esteem instability may be quantified by the extent that self-esteem responses to a lab-induced stressor (i.e., as argued in chapter 6 concerning ISE instability). As such, self-esteem reactivity may be a better measure of self-esteem stability. Future studies should aim to see whether self-esteem reactivity and self-esteem instability are indeed related, and as such, whether lab-induced self-esteem reactivity differ across clinical groups.

In the present thesis, and in many previous studies (see Okada 2010 for meta-analysis), a strong relationship between trait (ESE) and fluctuations in state (SE-S) was observed. However, it is unclear whether this relationship is causal, and if so, in what direction. It is feasible that increases in trait high self-esteem leads to more stable self-esteem because of a higher resilience of negative factors (e.g., negative feedback; Ford & Collins, 2010). It is also possible that increased self-esteem stability is required before an increase at the trait level occurs. Indeed, if self-esteem reactivity is less, then it is possible that an individual will engage in more situations that may boost self-esteem (Baumeister et al., 1989). Indeed, if there is a causal association between ESE and SE-S, it suggests that interventions need only target either

ESE or SE-S, and not both necessarily, although it may be pivotal to monitor both.

Self-esteem instability in the present thesis refers to fluctuations of ESE. It was hypothesized in chapter 6 that ISE may also fluctuate, specifically, in response to sad mood. It seems very plausible that sad mood would activate negative content in memory, thus increasing the speed and ease with which negative content associated with the self are triggered. However, results suggest that those who received a sad-mood induction did not report lower ISE than those who did not receive such an induction. On the basis of these findings, one could argue that ISE might be a stable construct, not influenced by external factors or processes, including mood. However, there are limitations to the design in chapter 6 that may have introduced other influencing factors not accounted for in the analysis (e.g., lack of neutral-mood induction similar to the sad-mood induction), and the analysis used does not allow for confirmation of the null hypothesis (i.e., that sad mood does not influence ISE). Further, it could be that sad mood influences ideal and actual self-esteem in different ways, (e.g., while actual ISE decreases, ideal ISE increases) which would not have been picked up by the IAT (Remue et al., 2013). A stronger design is required to test the influence of mood on implicit associations, and comparing the effects of a sad-mood induction to a happy-mood induction may show differences in ISE better, particularly as the sad-mood induction used seemed only to have a slight, albeit statistically significant, effect on self-reported sad mood. If indeed ISE does fluctuate in response to changes in mood, it may be crucial to control for individual differences in current mood state prior to comparing groups.

Self-Depressed Associations (SDA)

Explicit SDA, and not implicit SDA, predicted (time to) relapse in those who had recently remitted from a depression (< 2 years). This was not true for everyone with a history of depression. However, in the present study, and previous studies looking at SDA, (e.g., Elgersma et al., 2013), it is not possible to conclude that explicit SDA reflect a scar following an episode of depression. Indeed, it has been argued that it is important to distinguish vulnerability factors that were present before the onset of the first depressive

episode which may still be present following the end of an episode (i.e., pre-morbid vulnerability factors) from vulnerability factors that appear during and following a period of depression (i.e., scar; Burcusa & Iacono, 2007). It is also feasible that vulnerability factors existing before depression onset already can predict who will have recurrent depression and who may experience fewer or just one episode (e.g., those with more than one MDD episode had stronger pre-morbid cognitive reactivity, Elgersma et al., 2015; or were more likely to have had a previous anxiety disorder, Wilhelm, Parker, Dewhurst-Savellis, & Asghari, 1999). As such, future research may focus on testing whether explicit SDA preceding the first depressive episode predicts a recurrent prognosis. However, such a study would involve a lot of (potentially unnecessary) work as never depressed individuals would have to be recruited, and presumably only a percentage would develop depression, of which only a percentage would both remit and relapse. Not only would this result in a small portion of usable data, but follow-up would have to continue for years in order to capture relapse and recurrence. While this may help in developing our understanding of depression, it may have little clinical application.

To be of any clinical significance, it is perhaps not necessary to determine whether explicit SDA were already present before the first episode of depression. Identifying scars is clinically useful as it highlights possible avenues of relapse prevention. This is of key importance in depression research given the high rates of recurrence and relapse (e.g., 42% - 85%; Hardeveld et al., 2013; Mueller et al., 1999), and the often somewhat disappointing results of current relapse prevention treatments that have been reported. For example, although a treatment effect was observed specifically in those with more than 3 previous episodes, 75% of remitted recurrent depressed individuals who received preventative cognitive therapy still relapsed within 5.5 years (Bockting et al., 2005). As such, having observed a relationship between explicit SDA and an increased risk of (quicker) recurrence, the next clinically useful step may involve targeting explicit SDA, specifically, to see whether the risk for relapse reduces. While explicit SDA may already have been present before the onset of the disorder and

therefore cannot be considered a scar, targeting these may still be effective in preventing relapse and recurrence.

It is worth noting that it might be more cost and time efficient, in the long run, to first further research whether low ESE following depression (and anxiety) increases the risk for relapse than continuing with the more specific forms of self-associations (e.g., SDA). In the present thesis, there was some support for this, as both recovered and remitted depressed or anxious individuals had lower ESE than the relative healthy controls (chapter 3). However, studies looking at whether low ESE in remittance predicts relapse could not be found. It might therefore be worth further investigating whether ESE can predict relapse over a longer period, in both depression and anxiety, thereby highlighting a possible area for preventative treatment that can be used across disorders. This would not only be cost and time efficient with regards to the research involved (e.g., it would be feasible for a study to recruit both remitted depressed and remitted anxiety participants), but given the high rates of comorbidity within and between disorders (e.g., 57%, Brown et al., 2001), a self-esteem based relapse prevention might offer more broad protection from psychopathology in general. A preventative treatment targeting self-depressed associations is unlikely to be effective in those with an anxiety disorder only, and ineffective in preventing the development of anxiety symptoms in remitted depression, as self-depressed and self-anxious associations have been found to be disorder specific (Glashouwer & de Jong, 2010). Regardless, the findings of chapter 5 suggest that explicit SDA may be a useful target in treatment preventing relapse for remitted depressed individuals.

Limitations of Depression and Anxiety Definitions

It is important to note some key criticisms regarding depression and anxiety research more generally, which may influence the interpretation of the findings. There has been a movement over the last decade in research to consider disorders as a network of somatic and psychological complaints rather than a latent variable causing symptoms (Borsboom & Cramer, 2013). Not only does this highlight the heterogeneity of depression (and anxiety), but it also highlights that symptoms are distinct behavioural and physical

outputs that can cause and influence other symptoms. For example, lack of sleep may lead to issues in concentration, and less sleep and less concentration may result in a reduced ability to connect with more positive situations, thus resulting in reduced positive affect. Network models are interesting because they also highlight which symptoms (or "nodes") to target that may have a subsequent influence on other associated symptoms. Indeed, in a network analysis of depressive symptoms (Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015), feelings of worthlessness ranked third out of 21 symptoms on "outdegree" (i.e., the likeliness that other symptoms increase following increases in self-reported worthlessness). A network approach would also reveal whether fluctuations in self-esteem (i.e., low SE-S) are partially explained by other symptoms. In this thesis, self-esteem has been compared between clinical groups (e.g., those meeting the criteria for an MDD diagnosis) and controls, or self-esteem and the associations with depressive symptoms, as represented by a sum score, has been calculated. With the exception of chapter two, where self-esteem differences between MDD with suicidal ideation and MDD without suicidal ideation were investigated, depression has been considered as a homogeneous construct causing symptoms. As such, it may be pivotal to look at the individual level if self-esteem is always lower in depression and anxiety, rather than considering the group mean. If self-esteem is consistently lower, showing little individual variation, then it may highlight a key node to target that may, subsequently, reduce other symptoms. There is some support that self-esteem might precede other symptoms of depression and anxiety (Sowislo & Orth, 2013). Further, given that low self-esteem is present in both depression and anxiety, it is feasible that self-esteem represents a "bridge symptom", thereby explaining the high rates of comorbidity (Cramer, Waldorp, van der Maas, & Borsboom, 2010).

A second criticism refers to variations in definitions of remittance and recovery across research. Indeed, this led Frank et al., (1991) to provide recommendations for what defines remittance and recovery in depression based on the number of months where the criteria for a diagnosis is not met. This provided some consistency across research, thus allowing for comparison of results. However, concerning anxiety disorders, previous

research has often applied different cut-offs (e.g., remittance = 3 months of no anxiety symptoms; Penninx et al., 2011). In this thesis, cut-offs defining remittance and recovery were the same across anxiety, depression, and comorbidity. This did allow for comparisons across clinical groups without having to factor in differences in time since meeting the criteria for the disorder. If different cut-offs had been applied, findings may have been more difficult to explain. For example, if low self-esteem were found in remitted depression and not in remitted anxiety, then this may have been explained by the number of symptom-free months, or by differences between remitted depression and remitted anxiety. Given that those with a current depression and current anxiety did not differ in self-esteem (neither explicit, implicit, nor stability), it was pivotal to apply the same criteria for remission and recovery, particularly as differences observed between recovery and remittances (chapter 3 & 4) implies some continued improvement. Further, terms like remittance and recovery seem a little arbitrary given the high rates of relapse and recurrence, or perhaps, just wrongly labelled. Even the word "episode" would suggest a clean-cut end, rather than the slow tapering off that is more frequently observed. As such, perhaps it would be better to quantify the period following an "episode" in the number of months/years that the criteria for a depression or anxiety was not completely met. Much like previous alcohol dependence is quantified in the number of years "clean" thereby highlighting the vulnerability that exists but is increasingly decreasing.

Conclusion

Low explicit self-esteem was consistently observed in depression and anxiety in this thesis, and in previous research, with some support of a causal role, as lower ESE predicted more symptoms of depression and anxiety two years later. It would therefore be most fruitful, in terms of developing effective interventions and preventions, to continue with this facet of self-esteem in research. The next step, and already it has been undertaken by some, is to look at why low explicit self-esteem arises. Just like anxiety and depression, there will likely be several different pathways, and each may require a unique intervention. Further, it is pivotal to move on from existing self-report measures of self-esteem, and to establish behavioural measures

that are resistant to presentation biases. A lack of evidence for implicit self-esteem in depression and anxiety is a common finding, both in this thesis and in previous studies. However, more research is needed to better understand when ISE is engaged and whether some explicit processing, and thus ESE, is ever absent. If this is rarely the case, and ESE plays a more central role in behaviour and emotion, attempts at improving ISE may have little clinical significance, particularly if low ESE persists. Possibly this is not only specific to self-esteem, but also other self-related constructs, as explicit SDA were related to quicker recurrence in recently remitted, while implicit SDA was not. Discrepant self-esteem may also be a research avenue with little clinical significance, as it appears that low ESE plays a much larger role in depression and anxiety. One could argue that an intervention for ISE following successful improvement of ESE would be valuable given that fragile forms of self-esteem (i.e., high ESE and low ISE) in previous research has been linked to undesirable personality traits like narcissism. However, many theories of how ISE develops would suggest that ISE will improve following sustained improvement of ESE. Finally, there appears to be a relationship between stable and high ESE, suggesting that any increases in ESE may also result in a more stable self-esteem. Previous studies have suggested that low stable self-esteem is more reluctant to change, and therefore instability might indicate individuals who are more likely to respond to self-esteem interventions. Only through the manipulation of explicit self-esteem (and explicit SDA) will it become evident whether there is a causal relationship with depression and anxiety. Future studies should focus on processes that might give rise, and maintain, low explicit self-esteem, to develop effective interventions.

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Nederlandse Samenvatting

Eigenwaarde bij Depressie en Angst: Laag, Onstabiel, en Discrepant?

Deze these richt zich op eigenwaarde en de rol die eigenwaarde mogelijk speelt in het ontstaan, het voortduren en de terugkeer van (symptomen van) angst en depressie. In deze samenvatting wordt het woord eigenwaarde gebruikt als vertaling van het Engelse begrip "self-esteem". Over het algemeen refereert hoge eigenwaarde aan positieve zelf-gerelateerde evaluaties en lage eigenwaarde aan negatieve zelf-gerelateerde evaluaties. Echter, onderzoek heeft laten zien dat er ook specifieke facetten van eigenwaarde zijn, met ieder hun eigen relatie tot gedrag en emoties. Deze verschillende facetten hebben mogelijk ook elk een unieke relatie tot depressie en angst. In deze these wordt in meerdere studies een vergelijking gemaakt tussen de eigenwaarde van mensen met of zonder depressieve- of angststoornis, gebruikmakend van grote steekproeven. De verschillende facetten van eigenwaarde die in deze these onderzocht worden, zijn expliciete eigenwaarde, impliciete eigenwaarde, de discrepantie tussen de impliciete en expliciete eigenwaarde en, tot slot, de impliciete of expliciete associaties met 'het zelf' die specifiek lijken voor depressie zoals ik-waardeloos, ik-hopeloos. Het laatste empirische hoofdstuk van deze these richt zich op factoren die impliciete eigenwaarde kunnen beinvloeden. Meer specifiek is in dit hoofdstuk onderzocht hoe impliciete eigenwaarde verandert in reactie op een negatieve stemmingsinductie.

Alvorens per studie de kern van de uitkomsten van deze these samen te vatten, is het van belang om eerst de relevante facetten van eigenwaarde te definiëren die in deze these aan bod komen. *Impliciete eigenwaarde* (IE) verwijst naar de valentie van constructen die sterk geassocieerd zijn met het zelf, waarbij de kans groot is dat ze ook geactiveerd worden als het zelf-construct geactiveerd wordt. Wanneer ik bijvoorbeeld vaak een tentamen niet haal en iedere keer denk dat ik een mislukkeling ben, dan wordt de associatie tussen het zelf en 'een mislukkeling zijn' sterker. Als de associatie sterk genoeg is dan kan met een simpele activatie van het zelf, automatisch het construct van mislukken geactiveerd worden, zelfs wanneer er geen sprake is van een tentamen. *Expliciete eigenwaarde* (EE), aan de andere kant, verwijst naar de uitkomst van het actief en bewust evalueren van bepaalde zelf-gerelateerde

proposities (bijvoorbeeld: 'Ik ben lelijk'). De automatisch geactiveerde associaties kunnen in dit proces als irrelevant of onjuist ter zijde worden geschoven (bijvoorbeeld: in vergelijking met de meeste anderen ben ik niet speciaal lelijk), maar ook bevestigd ("ik ben –inderdaad- lelijker dan anderen"). IE en EE kunnen daarom van elkaar verschillen. In het geval IE en EE van elkaar afwijken wordt in de literatuur wel gesproken van *discrepante eigenwaarde* (discrepant self-esteem). Men spreekt van een beschadigde eigenwaarde als een positieve IE samengaat met een negatieve EE, en van een kwetsbare eigenwaarde als een negatieve IE samengaat met een positieve EE. Er zijn aanwijzingen dat beschadigde eigenwaarde betrokken zou kunnen zijn bij depressie. *Eigenwaarde stabiliteit* (ES) verwijst naar de mate waarin eigenwaarde stabiel is over de tijd. Mensen verschillen in de mate waarin eigenwaarde al dan niet fluctueert bijvoorbeeld in reactie op omgevingsfactoren en stemming. ES lijkt overigens niet geheel onafhankelijk van EE; mensen met een hogere eigenwaarde als eigenschap (EE) hebben ook vaker meer stabiele situatie onafhankelijke eigenwaarde (ES). *Zelf-depressie associaties* (ZDA) verwijzen naar zelf-associaties met specifiek aan depressie gerelateerde constructen (bijvoorbeeld: hopeloosheid).

Studie 1 (Hoofdstuk 2)

Studie 1 onderzocht twee mogelijke relaties tussen eigenwaarde en symptomen van depressie/sociale angst. Ten eerste is onderzocht of adolescenten met lage IE en/of EE een hoger risico lopen op het ontwikkelen van symptomen van depressie of angst. Ten tweede is onderzocht of de aanwezigheid van relatief intense symptomen van depressie en/of sociale angst bijdragen aan het ontwikkelen van laag IE/EE. Periodes van depressie (en angst) zouden littekens kunnen achterlaten, zoals lage eigenwaarde ('scar hypothese'). Dit zou dan mede kunnen verklaren waarom mensen na initieel herstel vaak weer terugvallen en nieuwe episodes van depressie (of angst) ontwikkelen. Om beide relaties te onderzoeken is gekozen voor een longitudinaal design en is een grote groep adolescenten (N = 1631) tweemaal gemeten met een interval van 2 jaar. IE is gemeten met de zogenaamde Implicite Associatie Test (IAT) en EE met de Rosenberg Self Esteem Scale (RSES). IE bleek niet samen te hangen met meer symptomen van depressie/sociale angst na twee jaar; evenmin bleek IE na twee jaar lager

bij adolescenten die tijdens de eerste meting relatief hoog scoorden op symptomen van depressie of sociale angst. Dus IE leek adolescenten niet kwetsbaar te maken voor het ontwikkelen van depressie/sociale angst. Evenmin bleek sociale angst/depressie een laag-IE-litteken achter te laten, wat hen mogelijk op termijn kwestbaar zou kunnen maken voor het ontwikkelen van (een hernieuwde periode van) sociale angst/depressie. Laag EE tijdens de eerste meting bleek daarentegen wel voorspellende waarde te hebben voor relatief hoge scores op symptomen van depressie en sociale angst op de tweede meting twee jaar later, ook als statistisch werd gecontroleerd voor de symptoomscores tijdens de eerste meting. De hoogte van de symptoomscores tijdens de eerste meting bleken niet voorspellend voor de hoogte van EE tijdens de tweede meting. Dus de resultaten boden geen ondersteuning voor het idee dat een periode van relatief intense symptomen van depressie en/of sociale angst een litteken zouden achterlaten in de vorm van lage EE. De resultaten zijn dus in overeenstemming met de theorie dat lage eigenwaarde een rol kan spelen in de ontwikkeling van intensere symptomen, maar niet met het idee dat symptomen toekomstige niveaus van eigenwaarde beïnvloeden. Deze resultaten suggereren dat interventies ter verhoging van EE behulpzaam zouden kunnen zijn bij het voorkomen van het ontwikkelen van sociale angst en depressie onder adolescenten

Studie 3 (Hoofdstuk 4)

Duale proces modellen van psychopathologie benadrukken het belang onderscheid te maken tussen meer automatisch geactiveerde zelf-associaties (IE) en meer bewuste, weloverwogen zelf-associaties (EE). Zowel IE als EE zouden een rol kunnen spelen bij het ontstaan van een depressieve stoornis (DS) of angststoornis (AS); Daarnaast is betoogd dat tengevolge van DS en AS een laag IE litteken zou ontstaan dat zou kunnen bijdragen aan de terugkeer van symptomen na herstel. De beschikbare evidentie biedt echter geen eenduidige ondersteuning voor de relevantie van laag IE in DS en AS en studies die zich richtten op discrepante eigenwaarde lieten zelfs zien dat juist hoog IE in combinatie met laag EE predictief zou zijn voor het ontwikkelen van angst/depressie. De eerdere studies kenmerkten zich echter zonder uitzondering door kleine steekproeven, onduidelijke definities van

onderzoeksgroepen in termen van comorbiditeit en fase van de stoornis en problematische indices van discrepante eigenwaarde die niet toestaan te controleren voor IE en EE sec. Om te komen tot beter onderbouwde conclusies maakte Studie 3 daarom gebruik van een grootschalige studie (de Netherlands Study of Depression and Anxiety; NESDA), die het mogelijk maakte heel strikte groepsindelingen te maken en beter te controleren voor de fase van de stoornis. Daarnaast maakte studie 3 gebruik van een nieuwe maat voor discrepante eigenwaarde die toestaat te controleren voor het deel in AS en MS dat al kan worden verklaard door verschil in EE. Voor deze studie zijn deelnemers geselecteerd met een huidige DS (n = 60), een huidige AS (n = 111), een huidige comorbide AS/DS (n = 71), deelnemers die kortdurend hersteld waren (remitted) van DS (n = 41), AD (n = 29), of een comorbide DS/AS (n = 14), deelnemers die minstens 6 maanden hersteld waren van MDD (n = 136), of AD (n = 98), en een vergelijkingsgroep die nooit een DS of AS heeft gehad (n = 382). Net als in studie 2 is IE gemeten met de Impliciete Associatie Test en EE met de Rosenberg Self-Esteem Scale. De vergelijkingsgroep die nooit een depressie of angststoornis heeft gehad, rapporteerde de hoogste EE (hoger dan alle andere groepen). De comorbide DS/AS groep rapporteerde de laagste EE (lager dan alle andere groepen). Het was ook deze groep die als enige klinische groep lagere IE liet zien dan de vergelijkingsgroep. De bevinding dat IE in de herstelde groepen niet lager was dan in de vergelijkingsgroep trekt het idee dat AS/DS zou leiden tot een laag IE litteken in twijfel. Discrepante eigenwaarde (verschil tussen EE en IE) bleek onafhankelijk van groep zodra statistisch werd gecontroleerd voor de verschillen in EE. Bijelkaar genomen bieden de resultaten geen ondersteuning voor de hypothese dat AS/DS zou leiden tot een laag IE (of EE) litteken. Tegelijkertijd ondersteunen de resultaten wel het idee dat verlaagde EE een rol kan spelen in zowel DS als AS. Alleen in de comorbide groep was er ook sprake van verlaagd IE hetgeen mogelijk de ongunstige prognose van deze groep mede kan verklaren. Het moet echter wel benadrukt dat studie 3 cross-sectioneel van aard was; om inzicht te krijgen in de richting van de relaties tussen eigenwaarde en symptomen van AS/MS is het belangrijk deze studie op te volgen met een longitudinaal design.

Studie 4 (Hoofdstuk 5)

Er niet alleen evidentie dat EE in het algemeen verlaagd is in DS en AS, maar EE zou mogelijk ook relatief instabiel zijn (laag ES). Laag ES zou mensen extra kwestbaar kunnen maken voor alledaagse stress en interpersoonlijke reacties die je zou kunnen interpreteren als afwijzend. Op die manier zou laag ES bij kunnen dragen aan het ontwikkelen van depressie en angststoornissen zoals sociale angst. Als eerste stap is in deze cross-sectionele studie gekeken of ES inderdaad lager is in mensen met DS of AS, en in welke mate laag ES persisteert in mensen die gedurende een korte periode of gedurende een wat langere periode zijn hersteld van DS of AS. Net als studie 3 maakte ook studie 4 gebruik van de NESDA. Studie 4 vergeleek deelnemers met een huidige DS (n = 60), AS (n =111) of comorbide AS/DS (n = 71), deelnemers die sinds kort waren hersteld van DS (n = 41), AS (n =29), of comorbide DS/AS (n = 14) en deelnemers die al langere tijd waren hersteld van DS (n= 136) of AS (n = 98) en tenslotte een groep deelnemers die nooit een DS of AS heeft gehad (n = 382). EE was gemeten met de RSES en ES met twee vragen die specifiek vroegen naar stabiliteit en instabiliteit van de eigenwaarde. De vergelijkingsgroep die nooit DS of AS had gehad vertoonde de hoogste ES (hoger dan alle andere groepen). Ook wanneer statistisch werd gecontroleerd voor EE, vertoonden de huidige DS/AS, kort herstelde DS/AS en langer herstelde DS/AS een geringere eigenwaarde stabiliteit dan de vergelijkingsgroep. ES in de comorbide groep vertoonde echter geen verschil met de vergelijkingsgroep. Bijelkaar ondersteunen de bevindingen van studie 4 het idee dat naast laag EE per se ook lage ES kan bijdragen aan het ontstaan en voortduren van affectieve stoornissen. De verlaagde ES was ook nog steeds zichtbaar in de groepen die hersteld waren van DS/AS. Het is belangrijk in vervolg onderzoek met longitudinaal design te onderzoeken of die verlaagde ES mogelijk ook bijdraagt aan de terugkeer van symptomen. Als dat inderdaad het geval zou blijken te zijn, zou het vanuit klinisch persectief niet alleen relevant zijn EE te verhogen maar ook in te zetten op het verhogen van de stabiliteit (ES). The combinatie van laag EE met hoog ES in de comorbide groep kan mogelijk ook (mede) verklaren waarom mensen met comorbide klachten als regel minder goed reageren op behandeling. Er zijn aanwijzingen dat enige mate van flexibiliteit in eigenwaarde belangrijk is voor behandelsucces. Specifiek voor mensen met een comorbide DS/AS is

het derhalve mogelijk cruciaal eerst in te zetten op het verhogen van de flexibiliteit in EE alvorens met succes een EE-interventie te kunnen toepassen.

Studie 5 (Hoofdstuk 6)

Eerdere studies hebben reeds laten zien dat impliciete en expliciete ZDA nog steeds sterker waren in mensen die hersteld waren van DS dan in mensen die nooit een DS hebben gehad, dat de ZDA sterker waren bij mensen met relatief veel eerdere episodes, en dat een langere periode van depressieve klachten samenging met sterkere ZDA. Studie 5 testte de hypothese dat die relatief sterke ZDA na herstel mogelijk een litteken is dat de kans op terugval vergroot. Hiertoe zijn longitudinale analyses uitgevoerd binnen de NESDA om vast te stellen of impliciete ZDA zoals gemeten met de Impliciete Associatie Test (IAT) en expliciete ZDA (zelf rapportage equivalent van de IAT) voorspellende waarde hebben voor de (tijd tot) terugkeer van DS. In de eerste analyse is getoetst of expliciete en impliciete ZDA tijdens de baseline van NESDA in personen die tijdens de baseline hersteld waren van minstens één depressieve episode (n =387) voorspellende waarde hadden voor (tijd tot) terugkeer symptomen binnen een 6-jaar follow up periode. De tweede analyse richtte zich op deelnemers met DS tijdens de baseline en die waren hersteld tijdens de 2-jaar follow up vervolgmeting (n = 279). Deze analyse onderzocht of de mate waarin expliciete en impliciete ZDA persisteerden na herstel voorspellend was voor de (tijd tot) terugkeer van DS in de 4-jaar follow up periode. Het percentage deelnemers met een terugkeer van DS was 42.4% binnen het tijdsinterval van de eerste analyse (6 jaar), en 54.5% voor de tweede analyse (4 jaar). De eerste analyse liet zien dat noch expliciete noch impliciete ZDA prognostische waarde hadden voor de terugkeer van DS. In de tweede analyse bleek dat expliciete ZDA maar niet impliciete ZDA predictieve waarde hadden voor de (tijd tot) terugkeer van DS ook als statistisch werd gecontroleerd voor de mate van residuele symptomen van depressie zoals gemeten met de IDS. Bijelkaar genomen boden de bevindingen geen ondersteuning voor het idee dat verhoogde impliciete ZDA na herstel van een depressieve episode kunnen worden gezien als een actief litteken (een litteken dat er toe doet). Verhoogde expliciete ZDA vertoonden daarentegen wel kenmerken van een actief litteken, met name als er nog relatief weinig tijd was verlopen sinds het

herstel van DS. Deze bevinding suggereert dat het uit oogmerk van terugval-preventie relevant is in te zetten op het bijstellen van expliciete ZDA.

Studie 6 (Hoofdstuk 7)

Er zijn aanwijzingen dat een sombere stemming de drempel verlaagd voor het activeren van negatieve associaties. Op basis hiervan zou het goed kunnen dat een sombere stemming ook de drempel verlaagd voor het activeren van negatieve zelf-associaties (IE). Omgekeerd zou een neutrale stemming de drempel juist kunnen verhogen om negatieve automatisch zelf-associaties te activeren hetgeen mogelijk zou kunnen verklaren waarom in studie 2 geen verlaagde IE werd gevonden bij mensen met DS of AS. Fluctuaties in IE in reactie op stemmingswisselingen zou kunnen bijdragen aan het in gang zetten van een negatieve spiraal die uiteindelijk kan uitmonden in DS. Studie 6 was ontworpen om te toetsen of een experimenteel uitgelokte negatieve stemming inderdaad zou resulteren in minder positief/meer negatief IE dan een neutrale stemming. De IAT was weer gebruikt als maat voor IE en een de RSES als maat voor EE. Proefpersonen werden random toegewezen aan de controle groep (n = 46) of de experimentele groep (n = 49). In de experimenele conditie werd de IAT afgenomen na een sombere stemmingsinductie. Ter ondersteuning van de experimentele manipulatie bleek de zelf- gerapporteerde stemming minder positief in de experimentele dan in de controle groep. IE bleek echter niet te verschillen tussen beide condities en evenmin was er meer algemeen een relatie tussen de zelfgerapporteerde stemming en IE scores. De relatie tussen stemming en IE score bleek ook onafhankelijk van EE. Studie 6 liet al met al zien dat IE relatief immuun is voor een tijdelijk geinduceerde negatieve stemming. Belangrijk hierbij aan te tekenen dat deze studie zich richtte op proefpersonen zonder klachten of historie van klachten. Het is denkbaar dat een negatieve stemming juist een invloed heeft op IE bij mensen met DS of die zijn hersteld van DS

Conclusie

In deze these zijn verschillende facetten van eigenwaarde onderzocht; de grootste en meest systematische effecten werden gevonden voor expliciete eigenwaarde (EE). In overeenstemming met het idee dat lage

EE een rol zou kunnen spelen bij DS/AS vertoonden mensen met een depressie, angststoornis of beide stoornissen een lagere expliciete eigenwaarde dan mensen in een klachtvrije controlegroep. Deze verschillen werden niet alleen gevonden tijdens de acute fase van de stoornis, maar ook na remissie of herstel hetgeen in overeenstemming is met het idee dat verlaagde EE na herstel mogelijk zou kunnen bijdragen aan de terugkeer van de klachten. Prognostisch onderzoek in studie 2 bood verdere ondersteuning voor de veronderstelde rol van EE bij de ontwikkeling van DS en AS en liet zien dat lage EE prognostische waarde had voor toekomstige symptomen van depressie en sociale angst in een steekproef van gezonde adolescenten. Ook de meer depressie-specifieke zelf-associaties bleken prognostische waarde te hebben voor een hernieuwde depressieve episode in recent herstelde personen. Cruciale vervolgstap is om te onderzoeken of EE daadwerkelijk een causale invloed heeft op het ontwikkelen van (symptomen van) depressie en angststoornissen. Experimenteel verlagen van EE om te toetsen of dat zou leiden tot DS/AS is natuurlijk geen reële optie en ethisch onacceptabel. Het is daarentegen wel mogelijk om technieken te ontwikkelen die er op zijn gericht specifiek eigenwaarde te verhogen en om vervolgens te toetsen of dergelijke interventies de ontwikkeling van symptomen kunnen voorkomen, en mogelijk ook een transdiagnostische interventie zouden kunnen vormen voor depressie en angststoornissen. Veel van de huidige interventies voor depressie en angst (zoals cognitieve gedragstherapie) richten zich op het uitdagen van negatieve, catestrofale overtuigingen, terwijl het nog niet is vastgesteld of dit ook het veranderingsmechanisme is dat ten grondslag ligt aan de effectiviteit van deze interventies. Mogelijk zou een interventie die specifiek gericht is op het verhogen van eigenwaarde al kunnen resulteren in afdoende symptoomreductie. Specifiek voor personen met een comorbide AS/DS is het daarnaast mogelijk relevant tevens in te zetten op het bijstellen van IE.

English Summary

Self-Esteem in Depression and Anxiety: Low, Unstable, and Discrepant?

This thesis discusses several studies looking at self-esteem, and the role that it plays in the onset, maintenance, and re-occurrence of anxiety and depression (symptoms). Self-esteem is frequent in lay conversation and literature. Common across definitions of self-esteem is that it refers to the valence of self-related evaluations. High self-esteem therefore refers to relatively positive self-evaluations. Literature concerning self-esteem research suggests that several facets of self-esteem exist, each with a unique relationship to different behaviour and mood. Further, each self-esteem facet has also been argued to have a potentially unique association with depression and anxiety. Throughout the majority of this thesis, large sample sizes were used to look at the extent of self-esteem in depression and anxiety, in comparison to a group without MDD/AD. Self-esteem facets considered in this thesis are explicit self-esteem, implicit self-esteem, self-esteem discrepancy, self-esteem stability, and implicit/explicit self-depressed associations. Although the latter is not considered a facet of self-esteem, per se, the theoretical underpinning has large overlap with implicit/explicit self-esteem, and can be considered as a more disorder-specific facet of self-esteem. The last empirical chapter of this thesis aimed to look at how low implicit self-esteem may develop. Specifically, this chapter looked at changes in implicit self-esteem in response to sad mood.

Before continuing onto an overview of the specific research questions addressed in this thesis, it is necessary to define what is meant by implicit/explicit self-esteem, discrepant self-esteem, self-esteem stability, and implicit/explicit self-depressed associations. *Implicit self-esteem* (ISE) refers to the valence of constructs that are strongly associated with the self, and are activated when the self-construct is activated. It is theorized that certain associations develop through repetitively processing self-related information in certain ways. For example, if I often fail an exam, and I think each time about what a failure I am, then the association between the self and failure would become increasingly stronger. If the association is strong enough, then simply the activation of the self may also automatically activate the construct of failure, even outside the context of an exam. *Explicit self-esteem* (ESE), on the other hand, refers to the relatively slower and more conscious

process of evaluating propositions (e.g., "I am ugly") on the degree of certitude. Memory may be used for evaluating the extent that a proposition is true. Crucial to ESE is that it is highly subjective and victim to all sorts of biases. For example, a negative memory bias may lead to remembering more negative situations, rather than positive ones, thereby increasing the likeliness that negative self-propositions are considered valid, and positive self-propositions are considered invalid.

Theory suggests that ESE may influence ISE if it is consistent and enduring. Likewise, ISE may influence ESE in those who consider their "gut-feeling" or initial reaction to have value when making evaluations. However, ISE and ESE need not be congruent. *Discrepant self-esteem* refers to the extent that ISE and ESE differ, and larger differences have been suggested to result in self-doubt. Further, when ISE is higher than ESE, this is considered to be representative of damaged self-esteem, while those who show the reverse pattern are considered to have fragile forms of self-esteem. Previous research has suggested that damaged and fragile forms of self-esteem have different consequences.

Self-esteem stability (SE-S) refers to fluctuation in state levels of ESE, and suggests that individual differences exist in how often, and the extent to which, self-esteem fluctuates. SE-S refers to the extent of self-esteem fluctuations on a moment-to-moment basis in response to environmental factors and mood. Although SE-S and ESE have often been considered as distinct constructs, there does seem to be a relationship between the two, with ESE and stability being positively related. In other words, those with higher trait levels of self-esteem (ESE) are also more likely to have more stable state levels of self-esteem (SE-S).

Self-depressed associations (SDA) only differ to the definitions of self-esteem in that they refer to the extent that constructs related, specifically, to depression (e.g., "hopelessness") are associated to the self. That is, for implicit SDA, the strength of association between the self and depression constructs. If implicit SDA are strong then the likeliness that depression-related constructs are triggered when the self-construct is activated is increased. For explicit SDA, this would refer to the extent that

self-depressed propositions are considered valid (e.g., "I am worthless"). Previous research has suggested that disorder-specific self-associations exist for anxiety and depression at both the implicit and explicit level.

Depression & Anxiety

This thesis focused predominantly on (symptoms of) depression and anxiety: mental health disorders that are common, persistent, and often characterised by low self-esteem. However, since the emergence of new self-esteem facets in research, findings have not always been consistent, and it is feasible that not all facets of self-esteem are involved in depression and anxiety. Knowing specifically which aspects of self-esteem are relevant would highlight which aspects to target in depression and anxiety treatment. Further, the persistence of dysfunctional self-esteem into remittance and even recovery might explain the high rates of relapse and recurrence in these disorders.

Thesis summary: Chapter by Chapter

For implicit and explicit self-esteem, two research questions were addressed. The first research question referred to the longitudinal associations between ISE and ESE and symptoms of depression and social anxiety in adolescents (chapter two). This question stems from observations that adolescence marks a period of low and vulnerable self-esteem. This has been theorized to result from a decrease in positivity bias that is present during childhood, and an increased pressure to create an identity for oneself. Further the pressure in school to do well, and the pressure of meeting parent and peer expectations, all stipulate a period of life where self-criticism is likely to be higher. This decrease in self-esteem may therefore highlight a period where an individual is particularly at risk for developing depression or anxiety. Indeed, for social anxiety disorder, onset is often observed before adulthood is reached. Depression onset during adolescence is also related to more persistent and severer depression aetiology later on in life. It is feasible that adolescents with low levels of implicit and explicit self-esteem are more at risk of developing symptoms of depression or anxiety. A reverse association between symptoms and self-esteem is also possible. Scar hypotheses highlight that periods of depression (and anxiety) can leave

scars, like low self-esteem. Through the presence of scars, a new episode of depression or anxiety is more likely to occur compared to those without a history of depression or anxiety.

In chapter two, levels of ISE and ESE were measured around the age of 13, along with symptoms of depression and social anxiety. Approximately two years later, these measures were completed again by the participants. Asking participants to also complete the self-esteem and symptomatology measures at follow-up and baseline, respectively, meant that it was possible to test the association between symptoms of depression and social anxiety at baseline and self-esteem at the 2-year follow-up, which may be indicative of a scarring effect. Results indicated that for ISE, there was no relationship with symptoms in neither direction (i.e., preceding and proceeding symptoms). ESE however was related to both symptoms of depression and symptoms of social anxiety two years later. This suggests that those with lower ESE at baseline were more likely to have relatively many/intense symptoms of depression and social anxiety disorder two years later, even when controlling for baseline symptoms. The reverse pattern was not observed. Symptoms of depression and anxiety at baseline were not related to self-esteem two years later. Thus the results are consistent with the view that low self-esteem may promote the development of more intense symptoms, whereas there was no evidence for symptoms impacting on adolescents' future levels of self-esteem.

The second question that was tackled in the present thesis concerned to what extent implicit and explicit self-esteem are involved in anxiety and depressive disorders. The previous findings concerning ISE are very mixed, with some observing a difference between clinical groups and a comparison group, and many failing to replicate this. It is possible that small sample sizes may have limited the power to find a difference, while increasing the likeliness of chance findings (i.e., type II and type I errors, respectively). Further, many previous studies have not controlled for the possible influence of a co-morbid anxiety or depression. This might be particularly pivotal as low ISE might be more prominent in comorbid depression and anxiety, rather than more singular forms of the disorder, as more severe and persistent symptoms are often reported in comorbidity.

Therefore, ESE may be lower and more enduring in comorbidity, thereby lowering ISE over time. As such, large sample of individuals without a history of anxiety disorder or depression was contrasted with several large samples of clinical groups (chapter three).

The study in chapter three (also four and five) was part of the Netherlands Study of Depression and Anxiety (NESDA), a large ongoing study into the course of depression and anxiety. Groups were formed representing current depression, current anxiety and current comorbid depression and anxiety, remitted depression, remitted anxiety, and remitted from both, recovered depression, and recovered anxiety. Results indicated that those in the comparison group had higher ESE than all clinical groups, including those who had recovered from a depression or anxiety. Further, those with both a depression and an anxiety had lower ESE than all other clinical groups. Regarding ISE, lower levels were only observed in those with a current comorbid depression and anxiety in comparison to those who had never been diagnosed with a depression or anxiety disorder. This is in keeping with previous theories that comorbidity represents a disorder with a unique aetiology to depression or anxiety, alone, and may also be more than just the sum of the parts. This would also call for interventions specifically tailored for those who have both depression and anxiety as simply combining existing treatments may not target the unique symptoms that arise in comorbidity. Theory concerning ISE and ESE would suggest that the low ISE observed in comorbidity may be explained by the lower ESE that was also observed. Further research is needed to determine the causal role of ESE in ISE, as this may be an important avenue for prevention interventions, because theory would suggest that ISE is slow and reluctant to change. This may also explain, in part, the treatment resistant nature of comorbid depression and anxiety.

The relevance of discrepant self-esteem in (symptoms of) depression and anxiety was also analysed in chapters two and three. Previous research indicated two common methodological approaches to quantify discrepant self-esteem: by an interaction variable (i.e., ISE X ESE), or by calculating difference scores (i.e., absolute difference ISE – ESE). In chapter two, an interaction between ISE and ESE was included in the regression model to see

if additional variance in symptoms of depression and social anxiety two years later could be explained (over and above ISE and ESE as main effects). This interaction was found not to be significant, suggesting no further variance was explained by self-esteem discrepancy over and above ESE and ISE entered separately. However, some argue that including an interaction in the model does not adequately represent self-esteem discrepancy. Specifically, that by not differentiating between damaged and fragile self-esteem, the role of particular forms of discrepant self-esteem is not accounted for. However, the alternative method of deriving discrepancy scores (i.e., calculating differences), does not allow for the inclusion of either ESE or ISE as a main effect, as this leads to multicollinearity. As such, any observed effects involving discrepancy scores may actually be driven by ISE or ESE, rather than discrepancy per se. In chapter three, a novel analysis was conducted that not only allowed for distinction between damaged and fragile forms of discrepant self-esteem, but also allowed for the inclusion of one main effect (i.e., ISE or ESE). As ESE is often observed as having stronger associations with depression and anxiety, this was included in the model together with the measures of discrepancy. Results did not support the hypothesis that self-esteem discrepancy (neither damaged, nor fragile) differentiated those with a current depression or current anxiety disorder from the healthy comparison group once ESE was included in the model. As such, the findings would suggest that low ESE should be targeted in depression and anxiety regardless of whether ISE is congruently low or not. In other words, low ESE does not appear to be particularly involved in depression and anxiety when ISE is high. Nor is high ESE a distinguishing factor in depression and anxiety when ISE is low. Consistent with the effect sizes observed in chapters two and three, depression and anxiety are associated with particularly low levels of ESE.

While there was support that trait level of self-esteem (ESE) is lower in clinical groups of depression and anxiety than a healthy comparison group, many have argued that the presence of large fluctuations around this trait level makes a person particularly susceptible to depression and anxiety. The next research question pertained to the stability of self-esteem at a state level (chapter four). Previous research concerning SE-S has often used

analogue samples with some results indicating that low levels of SE-S are associated with symptoms of depression and anxiety, regardless of ESE levels. Others have argued that low SE-S in addition to low ESE is related to many and severer symptoms, while stable levels of self-esteem add some protection. In the few studies that have looked at SE-S in clinical levels of depression and anxiety, sample sizes were small and differences in ESE were not always controlled for. This latter point is particularly poignant given that SE-S and ESE are highly correlated. As such, differences between clinical groups and comparison groups in the absence of controlling for ESE may simply have reflected the often-observed differences in ESE. As such, using the same sample as in chapter three, SE-S was compared across current depression, remitted depression, recovered depression, current anxiety, remitted anxiety, recovered anxiety, current comorbid depression and anxiety, remitted comorbid depression and anxiety, and a healthy comparison group. Results indicated that the healthy comparison group had more stable self-esteem than all clinical groups. Further, both those who had recovered from an anxiety disorder, and those who had recovered from depression showed more stable self-esteem than all other clinical groups, with the exception of remitted anxiety. However, when controlling for ESE, results revealed that the healthy comparison group still had more stable self-esteem than those with a current depression, remitted depression, current anxiety disorder, recovered depression, and recovered anxiety disorder. This again points to a unique aetiology in co-morbid depression and anxiety, as a difference in SE-S was not observed when compared with the healthy comparison group. This is also consistent with a previous finding that suggested that lower self-esteem stability was related to more improvement following therapy, and the authors argued that some instability in self-esteem was reflective of some flexibility. As such, high stable low ESE in co-morbidity may partly explain the difficult-to-treat nature of comorbidity. This may point to an important indicating factor when treating comorbid depression and anxiety as increased instability might reflect an increasing flexibility. Further research is required to look at how ESE and SE-S are connected as increasing ESE might already increase SE-S in those with a depression or anxiety, and may, at first, decrease SE-S in comorbid depression and anxiety.

While self-esteem refers to the positivity of self-evaluations in general, disorder-specific self-evaluations may be more sensitive to group differences. Previous research has observed low levels of implicit and explicit SDA in those remitted and recovered from a depression. Further, it appeared that a history of more depression episodes, and more months of depressive symptoms in the previous two years, were related to stronger implicit SDA. As such, it is highly feasible that persistent implicit and explicit SDA represent a scar following depression. Theoretical papers stipulate that one of the criteria of a depression scar is that it is related to relapse and recurrence. Therefore, the research question as to whether residual SDA following a depression predicts (time to) relapse and recurrence was addressed in chapter five. Results indicated that explicit SDA was related to higher risk for relapse and recurrence. Further, stronger explicit SDA was related to quicker relapse and recurrence. However, these findings were not observed when including all those with a history of depression (i.e., recovered and remitted depressed), but only those who had remitted or recovered in the past two years. Consistent with ESE differences observed between recovered and remitted depression in chapter three, it is highly likely that improvement continues through recovery. There was no support that stronger implicit SDA was associated with (time to) relapse or recurrence. These results justify further research in this area with the next step to look at manipulating explicit SDA in order to prevent relapse and recurrence, or promote recovery.

While the notion of state levels of ESE is well documented in previous research (i.e., SE-S), studies looking at fluctuations in ISE in response to mood changes have largely been ignored. Theoretically, it is feasible that with sad mood, negative associations are more easily triggered as negative constructs might already be slightly more activated than positive ones. As such, when the self is triggered, relatively more associated negative constructs are more likely to be activated than during a relatively neutral mood. Further, previous theoretical articles suggest that depressive cognitions can lie dormant and are triggered with sad mood and stress. Although this is usually applied within the context of remitted and recovered depression, it is feasible that how reactive self-esteem is to sad mood relates to symptoms of depression and anxiety. This may explain why lowered ISE

was not observed in depression and anxiety in chapter three, as ISE was measured during a relatively neutral mood. To test these hypotheses, in chapter six, all participants received self-report measures of ESE, depression and anxiety. Participants were then randomized into receiving a measure of ISE straight away, or receiving a measure of ISE following a sad-mood induction. Results indicated that those who completed the measure of ISE following a sad-mood induction did not show lower ISE than those who completed it in the absence of a sad mood induction. These findings would suggest that ISE does not react to sad mood in a normal, non-clinical sample. While several design limitations need to be addressed before drawing conclusions, it is possible that ISE represents a construct that is robust to changes in mood. This is in keeping with the notion that ISE is relatively more stable and more reluctant to change than ESE. It is important to further explore which factors may influence ESE and ISE, as these may indicate signals for when an individual is at risk for lower self-esteem, and potential, increased risk in developing symptoms of depression and anxiety.

Limitations

It is important to note a few key limitations that may have influenced the findings. First, we relied on self-report measures of explicit self-esteem, self-esteem stability, and explicit self-depressed associations. While this introduces the possibility of presentation biases, suitable behavioural measures that may limit these biases are largely missing from the literature. As such, correlational findings between self-report measures of self-esteem and symptomatology in current study should be taken with a pinch of salt, as common method variance and biases may overinflate the relationship between the two constructs. Implicit self-esteem and implicit self-depressed associations were both measured with the implicit association test (IAT). There have been many criticisms of the IAT to adequately capture implicit associations. One main criticism is the lack of validity and reliability studies of the self-esteem and self-depressed IATs. Null findings are then quickly attributed to the inability of the IAT to capture the implicit construct. However, in other fields (e.g., stereotypes), the IAT does seem to fare better. While there is no doubt room for improvement concerning measurement of implicit self-concepts, perhaps a revision of theory is also necessary. Implicit

and explicit associations have often been considered to be distinct constructs, and a lack of correlation between the two has been taken as evidence of this. However, it might be more accurate to think of implicit and explicit associations as polar ends on a spectrum. That is, an evaluation can fall anywhere on the spectrum based on the amount of cognitive resources involved and the degree of awareness of the evaluation (to name but a few). Associations that are very implicit or very explicit are thus extreme in nature, and it is unclear as to how representative this is in daily life. It is feasible that associations concerning the self are rarely completely implicit, and there is always some degree of awareness or effort involved. Likewise, self-esteem measured on a questionnaire might represent self-evaluations that are considered and contemplated for longer than in daily life. Further, these are usually without a specific context, and therefore might be too abstract.

Clinical Implications

While several facets of self-esteem were considered in this thesis, the largest effects were found for explicit self-esteem. Specifically, those with a depression, anxiety disorder or comorbid depression and anxiety have lower ESE than a healthy comparison group. This was not only observed for those who currently met the diagnosis for these disorders, but also for those who were in remission and recovery. Further, in a healthy sample of adolescents, ESE was related to symptoms of depression and social anxiety two years later. In order to test the causal relationship of ESE and (symptoms) of depression and anxiety, it is pivotal to develop ways of boosting self-esteem. This may not only help prevent the development of symptoms, but may be a transdiagnostic intervention for both depression and anxiety. It is currently unclear as to whether challenging negative self-beliefs, an aspect in many common therapies of depression and anxiety (e.g., cognitive behavioural therapy), is a key mechanism of change. Perhaps an intervention targeting self-esteem solely and specifically is already enough to reduce symptoms.

While the other facets of self-esteem may play a unique role in depression and anxiety, it may not be necessary to target these, too. Facets of self-esteem are likely to be highly correlated with one another, and indeed, this was observed between SE-S and ESE. Further, theory suggests

that ISE forms following consistent and repetitive explicit self-evaluations. As such, targeting ESE, specifically, may also improve ISE, discrepant self-esteem, SE-S, and self-depressed associations.

Thesis Take Home Message

Several self-esteem facets have been identified in previous research. For each facet there is varying support of whether low levels exist in depression and anxiety. However, both in the present thesis and in previous research, the largest effects were consistently observed for explicit self-esteem. An intervention that corrects low ESE, and prevents future dips in self-esteem, may not only help those with a depression, but also those with an anxiety or comorbidity too. Many therapies already address negative self-evaluations to some extent, but it is unclear whether addressing self-esteem only may already be effective enough. Network analysis methods may highlight the centrality of self-esteem in the network of depression and anxiety symptoms. How self-esteem can be effectively improved remains an important question, and hints may be found in establishing factors that cause low self-esteem. Future research should not focus on how to further split the concept of self-esteem, but identify at the process-level what causes and maintains irrational and critical thoughts about the self. This in itself may prove vital and sufficient in reducing symptoms of depression and anxiety that are becoming increasingly as common as the common cold.

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"The best work that anybody ever writes is the work that is on the verge of embarrassing him, always"
Arthur Miller

With that in mind:

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Having moved from Scotland to a city in the Netherlands I had never heard of before, and that still, quite frankly, sounds like an ailment of the throat whenever anyone speaks it aloud, I am indebted to those who shaped my time here. Unlike ailments of the throat, I have come to love Groningen, and I can even say that I enjoyed working on my PhD. There are numerous people who have played a role in this, but I want to pay due attention to a few key contributors.

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I have found that the best cure for writers' block, frustrating statistics results, unexpected design flaws, and all the other negative things that inevitably crop up in research, is kicking a ball (and the occasional teammate or opponent – unintentionally, of course!) around the field. Oranje Nassau dames 4, Oranje Nassau dames 5, Helpman vrouwen 3 – our name has changed often over the last four years, but the laughs, comfort, and much-needed distraction has not. Ladies, I'm indebted to you, you truly are a wonderful bunch!

Marjolijn, I was somewhat dubious about including you in the acknowledgements as you have proved to be a major distraction[10]. But what a beautiful and delightful distraction you are! I am so glad that you are you, and that you encourage me to be me. With you, everything is possible, and nothing else really matters – not the London smog, nor the crazy rent prices.

And finally, you were the start of everything, and therefore it is also appropriate that you are the end. My loving parents. I know that it's been hard having an amazing daughter move to another country, and that you have found it difficult at times. Even though you're still not actually sure of what I am doing, your support has been constant and unwavering. No, I might not be doing a PhD in statistics, although you told a cousin otherwise. No, you can't buy the journal where my article is published in the local shop. Ok, so my first article lay on the table for weeks before dad finally admitted that he could not get past the first paragraph. And yes, I will write my project title down on a piece of paper, so that you can refer to it when others enquire about what I'm doing. And I suppose I could put my thesis on eBay if I have many copies spare. My wonderful and unintentionally-hilarious parents, thanks for the unconditional love and support that you both have provided in all my life. I have never doubted or questioned it for a second, and what a wonderful gift that is! (Although, you know… a car would *also* be a wonderful gift…).

[10] Actually, I'm pretty certain writing this thesis would have taken me at least another year if it had not been for you.

A Short Biography

Lonneke Aniek van Tuijl was born in Stavanger (Norway) on the 20th of May, 1987, and moved to the "Granite City" and "Oil Capital of Europe" in North-East Scotland before she turned two. Attending both primary and secondary school in Aberdeen, Lonneke went on to study Psychology at the University of Aberdeen (2005 – 2009). In 2010, Lonneke started studying at the University of Bath for a Master of Research in Psychology. This was completed with Merit in 2011, following various research projects on impulsiveness, self-esteem, and behavioural addictions. Having always been Dutch by nationality, but having never actually lived in the country, Lonneke was pleased to start a PhD in the department of Clinical and Experimental Psychopathology at the University of Groningen in November, 2011, under the supervision of Professor Peter de Jong. Since June 2016, Lonneke has been conducting post-doctoral research with Professor Richard Brown and Dr Colette Hirsch in the Department of Psychology at King's College London. This research looks at attentional bias (modification) in high worrying people with Parkinson's Disease. In her spare time, Lonneke enjoys eating, cooking, reading, board games, and football.

Publications

van Tuijl, L. A., de Jong, P. J., Sportel, B. E., de Hullu, E., & Nauta, M. H. (2014). Implicit and explicit self-esteem and their reciprocal relationship with symptoms of depression and social anxiety: A longitudinal study in adolescents. *Journal of Behavior Therapy and Experimental Psychiatry*, 45(1), 113-121. http://dx.doi.org/10.1016/j.jbtep.2013.09.007

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Elgersma, H. J., Koster, E. H., van Tuijl, L. A., Penninx, B. W. J. H, Bockting, C. L. H., & de Jong, P. J. Attentional bias for positive, negative, and threat words in current and remitted depression.

van Tuijl, L. A., Verwoerd, J. R. L., & de Jong, P. J. Attentional bias for angry and happy faces: Effects of sad mood on attentional engagement and disengagement.

van Tuijl, L. A., Verwoerd, J. R. L., & de Jong, P. J. The effect of sad mood on implicit self-esteem.

van Tuijl, L. A., Glashouwer, K. A., Bockting, C. L. H., Penninx, B. W. J. H, & de Jong, P. J. Predicting Depression Relapse and Recurrence with Implicit and Explicit Self-Depressed Associations.

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