# Dysfunctional automatic associations in anxiety and depression

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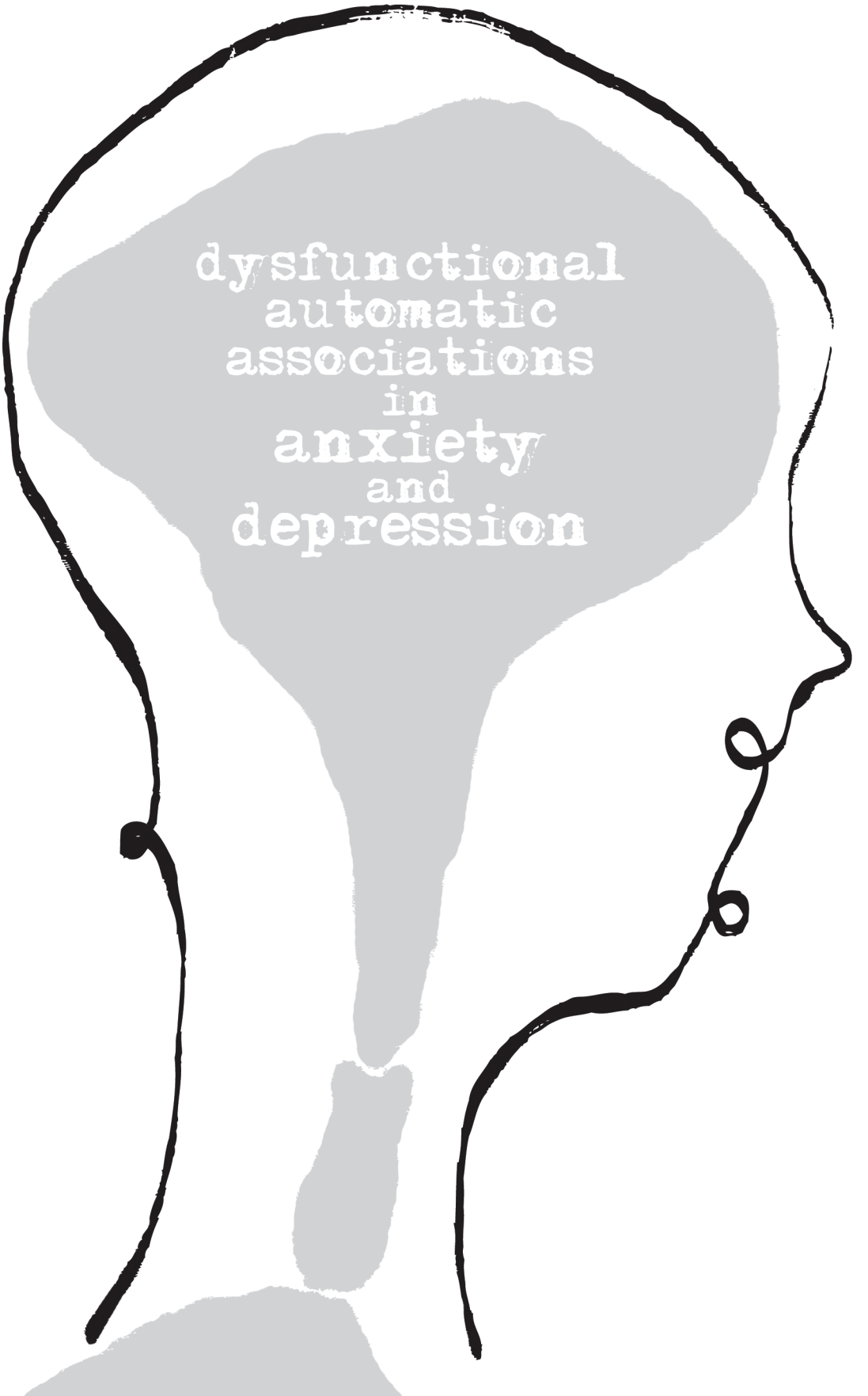
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# Dysfunctional automatic associations in anxiety and depression

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**Klaske Antje Glashouwer**
geboren op 26 maart 1982
te Achtkarspelen

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## Contents

# 1 Introduction

Which cognitive processes underlie the development and maintenance of anxiety disorders and depression? The present dissertation tries to shed a light on this question by specifically focusing on one type of cognitions, automatic associations, and their potential role in anxiety and depressive disorders. The introduction will start with an example of the role of automaticity in everyday live and will briefly describe dual process models, theoretical models that are used to understand automaticity. Next, the focus will depart from normal human behaviours and move towards behaviours that deviate from normality, even to the extent that individuals suffer from it like in depressive and anxiety disorders. I will explain how the dual process perspective might help us to better understand cognitive processes that play a role both in the development of these disorders and in the persistence of anxiety and depression once they have evolved. An overview of the empirical literature will be given, after which I will zoom in on the particular measurement instrument that we used, the Implicit Association Test. Finally, research questions and hypotheses will be lined up, which lead to conducting the studies in this dissertation.

## Glass floor

Recently, I was visiting Toronto to attend the annual congress of the American Association for Behavioral and Cognitive Therapies. After a long day of talks, we decided to enjoy the view over the city. The ultimate place to do this is the CN tower, for a long time the tallest building in the world being 533 meters high. One of the special features of the CN tower is a glass floor at about 350 meters through which you can look down on the tiny buildings, cars and people below. While I knew it was not actually dangerous (the floor is very well tested and it is supposed to hold at least 23 elephants), it was quite exciting to walk over this glass floor. My body immediately responded with an increase in heartbeat and my hand palms started sweating as if they tried to convince me not to do it. While I knew there was nothing to be afraid of, my body responded automatically with anxiety reactions, anxious thoughts popped-up in my mind ("this floor might not be strong enough") and I only dared to move slowly, carefully checking where I placed my feet, while I listened whether the floor might make any cracking sounds. Even after 20 minutes I still was not able to walk completely relaxed on the glass (see also Dijksterhuis, 2007).

Although this might seem an ordinary example, it is actually quite fascinating when you think about it further. The example brings up the question why people sometimes automatically behave in certain ways, without being able to control these behaviours. In the scenery of our everyday lives, we engage in a lot of behaviours automatically and sometimes even without being consciously aware. This is most of the time very adaptive

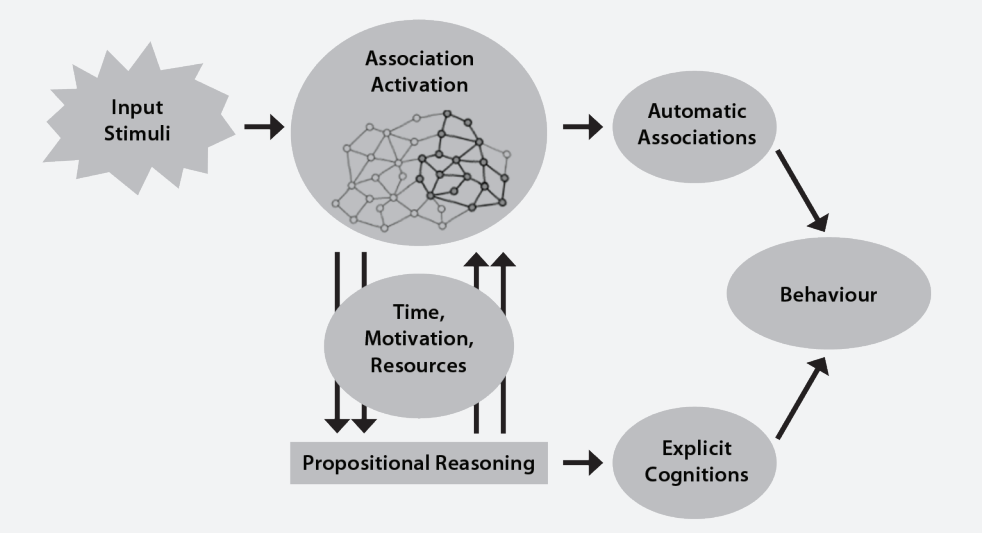
and makes it possible to perform all kinds of tasks simultaneously without needing to pay too much attention to them. For example, at this moment you are not only processing and interpreting the words you are reading, but you are also holding this 'book' in your hand, keeping yourself in an upright position in your chair, while you are breathing, your heart is beating and so forth. Being able to perform all these tasks without having to invest too much attention has an enormous survival value. It helps us to deal with the hectic of our daily lives, which is why the presumed underlying cognitive mechanisms of these automatic behaviours are referred to as 'the adaptive unconscious' (e.g., Wilson, 2002).

## Dual process models

How can we explain the phenomenon I experienced at the top of the CN tower? Theoretical models that could be useful here are so-called dual process models which focus on the question how cognitions guide behaviour (see Fig. 1.1; e.g., Fazio & Towles-Schwen, 1999; Gawronski & Bodenhausen, 2006; Strack & Deutsch, 2004). As the word dual already implies, these models start from the idea that two kinds of mental processes are operating behind the scenes of people's overt behaviours. To begin with, behaviours are assumed to be the result of automatic associations which stem from associative processes. In response to external or internal triggers, associations are activated spontaneously in memory within pre-existing associative networks. That is, as soon as one part of an associative network becomes activated, the activation spreads out to other parts of the network primarily driven by feature similarity and spatiotemporal contiguity (Gawronski & Bodenhausen, 2006). It might help to think of an associative network as a stone that is dropped in a pond. The surface of the water does not only wrinkle on the specific spot where the stone was dropped, but the entire surface of the pond gradually starts to wrinkle. Because activation automatically spreads through the associative network, associations can become activated irrespective of whether a person considers them as valid or invalid.

In addition, behaviours are assumed to be the result of explicit cognitions which stem from propositional processes. Individuals only engage in propositional reasoning when they are highly motivated to perform accurately and when they have sufficient time and cognitive resources to do so (Fazio & Towles-Schwen, 1999; Gawronski & Bodenhausen, 2006; Strack & Deutsch, 2004). In propositional reasoning, the input of the associative network is used for logistic inferences. As result of the reasoning, additional associations can become activated which again are included in the propositional reasoning. The crucial difference between both kinds of mental processes is that propositional reasoning is generally concerned with the validation of propositions, whereas the activation of associations

Figure 1.1
*Summary of different dual process models (Fazio & Towles-Schwen, 1999; Gawronski & Bodenhausen, 2006; Strack & Deutch, 2004)*




can occur regardless of whether a person considers these associations as true or false. Because propositional processes are slow and require a high amount of cognitive capacity, they are assumed to be easily disturbed by for instance distraction or high/low states of arousal. In contrast, associative processes are considered to be fast and require little cognitive capacity. Associative processing is generally thought to be 'the default mode', although both kinds of mental processes will often work synchronously, because propositional processes are largely based on what is available from the associative network. However, when propositional processes lead to the rejection of automatic associations as valid basis for behavioural decisions, the explicit cognitions that arise can lead to behavioural outcomes that differ from the associative pathway.

Back to the CN tower example. If we look at it from a dual process model perspective, it seems that, while I was looking over the edge, automatic associations with threat were activated in my associative memory network. These automatic associations might have led to the physiological arousal and the tendency to carefully check every step I took. However, I paid quite some money to go up the tower, so I was pretty motivated to make the most out of the experience. In addition, I had enough time and cognitive capacity for weighing pros and cons ("this floor might not be strong enough", "but it can hold 23 elephants", "so it will be able to hold my weight") leading to the explicit cognition that the floor was safe to walk over. This cognition seemed to have guided my behaviour of walking on the glass floor.

## Abnormal behaviours

As already mentioned, often these automatic, fast behaviours are really adaptive. If the glass floor would not have been a solid glass plate, but a hole in the floor, it would have been lifesaving that my body warned me for this. However, in some cases automatic responses are not so adaptive and individuals can be really bothered by them. This might be the case in depressive and anxiety disorders. Major depressive disorder is a mental disorder characterized by an all-encompassing depressed mood and/or by a loss of interest or pleasure in normally enjoyable activities. Anxiety disorder is a more broad term that covers several different forms of abnormal and pathological fear and anxiety. In this dissertation, I will mainly focus on social anxiety disorder, generalized anxiety disorder and panic disorder, because these anxiety disorders are highly prevalent, within disorder relatively homogenous in phenotype and the disorders are found across different health care settings. Individuals who suffer from social anxiety disorder typically experience an intense and persistent fear of social situations in which they are exposed to unfamiliar people or to the possible scrutiny by others. Generalized anxiety disorder is characterized by long-lasting anxiety that is not focused on any particular object or situation and individuals who suffer from generalized anxiety are overly concerned with everyday matters. In panic disorder, a person suffers from recurrent unexpected panic attacks of intense fear, often marked by physiological reactions like trembling, dizziness, nausea or difficulty breathing. These panic attacks abruptly arise and peak in less than ten minutes and individuals with panic disorder worry over future attacks and/or their potential consequences (American Psychiatric Association, 2000).

Depressive and anxiety disorders are highly prevalent and represent a major problem for public health these days. Recently it was shown that 20.1% of the general Dutch population suffers from clinical significant depressive symptoms during their lives, whereas the lifetime prevalence of anxiety disorders is 19.6% (de Graaf, ten Have & van Dorsselaer, 2010). Having to deal with one of these disorders has an enormous impact on a patient's life with pervasive social and economic consequences. In addition, for many patients symptoms keep returning, even after successful treatment. Consequently, quite some research focuses on the underlying cognitive mechanisms that play a role in the development, maintenance and relapse of anxiety and depressive disorders.

The assumption is widely adopted that depressive and anxiety disorder symptoms for an important part are being maintained by dysfunctional cognitions (e.g., Beck, Rush, Shaw & Emery, 1979; Clark, Beck & Alford, 1999; Ingram, Miranda & Segal, 1998; Rapee & Heimberg, 1997; Wells & Clark, 1997). Likewise, correcting these dysfunctional cognitions

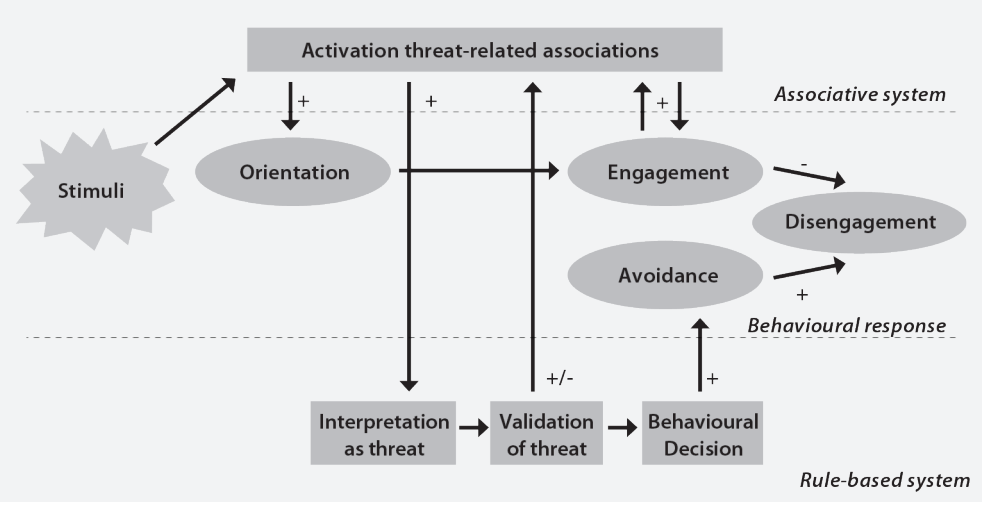
is often seen as a necessary precondition for recovery. However, when we put on the dual-process-model-glasses again, it seems likely that not only individuals' explicit cognitions might be important here, but also their dysfunctional automatic associations might contribute to the anxiety and depressive symptoms. Both depression and anxiety have very clear characteristics that point to automaticity in behaviours. For instance, anxiety disorder patients often know their fear is not in proportion with the feared situation or object, like talking to your neighbour in social anxiety disorder or going to the supermarket in agoraphobia. Yet, they are not able to prevent themselves from feeling and responding very anxious in these situations. Depressed individuals often react immediately with negative responses, without being able to stop these or even being aware of it. Since many symptoms of which anxious and depressed patients suffer seem to happen to them automatically, directly, and unintentionally, without having any control over it or even without being aware of it, it seems very logical that dysfunctional automatic processes might play an underlying role. Therefore, studying automatic associations seems crucial for a better understanding of how these disorders develop and why these disorders are so persistent once they have evolved. Moreover, looking at the role of automaticity might also have important implications for the treatment of these disorders. Interventions that target explicit cognitions might not necessarily change automatic associations as well (e.g., Gawronski & Bodenhausen, 2006; Grumm, Nestler & von Collani, 2009). This means that even when dysfunctional explicit cognitions change under the influence of treatment, dysfunctional automatic associations still could remain and keep having a negative influence on symptoms. That way, residual dysfunctional automatic associations after treatment could be an important predictor for reinstatement of symptoms. Consequently, getting more insight into the role of automatic associations in depressive and anxiety disorders could help to further improve already existing interventions for these disorders and 'break through' the persistent and recurrent nature of these disabling disorders.

## Disorder-specific dual process models: anxiety

Recently, Ouimet, Gawronski and Dozois (2009) proposed a multi-process model for anxiety disorders in which they use dual process models to explain information-processing biases (such as attention and interpretive biases) in anxiety disorders. The model is providing an integrated framework of how individual differences in the processing of threat information might contribute to the development and maintenance of anxiety disorders (see Fig. 1.2). In response to an anxiety-relevant stimulus, threat-related associations are thought to be directly activated as part of the associative system. The threat associations

lead directly to the orientation and engagement with the stimulus, which increases the activation of the associative system. In addition, information from the associative system is assumed to be used for the interpretation and validation of the stimulus through propositional processing (referred as the rule-based system). The outcomes of validation processes can lead to further activation of threat-related associations or to a deactivation of the associative system, depending on whether or not the threat-related associations are considered to be valid. When the stimulus is validated as a threat, this is assumed to lead to avoidance of the stimulus. However, a response conflict (whether or not to disengage) can develop, because threat-related associations actually promote engagement with the stimulus. This way, associative and propositional information processing systems jointly influence negative interpretive biases and attention biases which work together in a way to develop and/or maintain anxiety symptoms.

Figure 1.2
*Main elements of the multi-process model for anxiety disorders (Ouimet, Gawronski & Dozois, 2009)*




## *Available evidence*

Although not all anxiety disorders have been studied to the same extent, several studies support the view that threat-related associations might play a role within the cognitive vulnerability to anxiety disorders. One line of research focused on disorder-specific associations looking at threat-related associations that might be characteristic of one kind of anxiety disorder. Most studies in this area have been conducted in specific phobia, social phobia and panic disorder. High spider fearful individuals were shown to display more negative automatic associations towards spiders than non-fearful controls (Roefs

et al., 2011) and high socially anxious individuals displayed stronger negative automatic associations with social cues than low-anxious participants (de Hullu, de Jong, Sportel & Nauta, 2011; de Jong, Pasman, Kindt & van den Hout, 2001). However, in panic disorder, two studies failed to find group differences in automatic associations with bodily changes (Teachman, 2005; Teachman, Smith-Janik & Saporito, 2007), although stronger automatic catastrophic associations have been shown to significantly predict a smaller reduction in anxiety sensitivity in response to cognitive-behavioural therapy (CBT) in patients with panic disorder (Schneider & Schulte, 2008). Threat-related associations have also been studied in treatment and behaviour of spider phobia. It was found that automatic spider-associations were predictive of phobia-related behaviours beyond what explicit measures predicted (e.g., Huijding & de Jong, 2006a). In addition, automatic spider-associations seemed to decrease after cognitive behavioural treatment (Teachman & Woody, 2003), although this could also have been due to testing effects, since no waitlist-control group was included in this study. In line with the latter hypothesis, two later studies that did include a waitlist control group (Huijding & de Jong, 2007, 2009) showed similar decreases from pre-test to post-test in the treatment and the control group suggesting that the reduction in automatic spider-associations was the result of learning effects rather than treatment effects. However, recently, another controlled study found opposite results and showed that automatic spider-associations did normalize under the influence of CBT (Reinecke, Soltau, Hoyer, Becker & Rinck, 2012). Although the contrasting findings of these controlled studies seem puzzling, some methodological differences between the studies might account for the different outcomes, which I will discuss further in Chapter 6.

Other studies focused on more general threat-related associations that might play a role in the cognitive vulnerability across different anxiety disorders. Especially cognitions concerning the self might be important in the aetiology and maintenance of anxiety disorders (e.g. Acarturk et al., 2009; Batelaan et al., 2010; Rapee & Heimberg, 1997; Wells & Clark, 1997). In line with this hypothesis, several cross-sectional studies demonstrated that anxious individuals showed stronger dysfunctional automatic self-associations than non-anxious controls (social anxiety, implicit self-esteem: de Jong, 2002; Tanner, Stopa & De Houwer, 2006; social anxiety, automatic self-anxious associations: Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008; panic disorder, self-panic associations: Teachman, 2005; Teachman et al., 2007; post-traumatic stress disorder, self–invulnerable associations: Engelhard, Huijding, van den Hout & de Jong, 2007; for an extensive review, see Roefs et al., 2011). In addition, positive changes in automatic self-associations over the course of CBT for panic disorder were found to predict greater symptom reduction, but not the other way around

(Teachman, Marker & Smith-Janik, 2008). Finally, it was shown that automatic self-associations were predictive of experimentally-provoked anxiety behaviours in unselected student samples in the laboratory (e.g., Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002; Spalding & Hardin, 1999).

### *Open questions*

Although the available evidence overall suggests that both dysfunctional automatic associations with respect to the self and more specific threat-related associations might be involved in anxiety disorders, some important questions still remain unanswered. First of all, the research into dysfunctional self-associations included different types of self-associations in different anxiety disorders, which makes it hard to determine whether dysfunctional self-associations might be a shared vulnerability factor across several anxiety disorders. Secondly, prior studies in the field typically included analogue groups of high anxious individuals and low anxious individuals. Since there may be quantitative as well as qualitative differences between analogue and clinical samples (Emmelkamp, 1982), it seems important to replicate findings in clinical samples. Thirdly, prior studies mainly compared anxious groups with healthy control groups, leaving open the question whether differences regarding dysfunctional automatic self-associations can indeed be attributed to anxiety disorders or have to be seen as more general characteristics of psychopathology. Relatedly, with respect to more specific threat-related associations that might be particularly relevant for a particular anxiety disorder, it would be important to include other anxiety disorders as clinical controls to be able to really 'check' the disorder-specificity of the threat-related associations. Finally, up to now, studies mainly used cross-sectional designs or were conducted in the context of treatment. This leaves undecided whether dysfunctional automatic self-associations are also involved in the onset of anxiety symptoms and its naturalistic course over a longer period of time.

Consequently, the existing literature in this area should be further extended by research focusing on (1) the comparison of one kind of dysfunctional self-associations across different anxiety disorders. Automatic self-anxious associations might be a good candidate for this purpose, since self-anxious associations were already found to predict anxious behaviours (Egloff & Schmukle, 2002). Moreover, self-anxious associations might be 'broad enough' to serve as general vulnerability factor for different anxiety disorders, but 'specific enough' for anxiety disorders, rather than for several other psychopathological disorders. Hereby, it would be important (2) not only to include clinical samples of different anxiety disorders, but also non-anxious controls as well as clinical controls. In

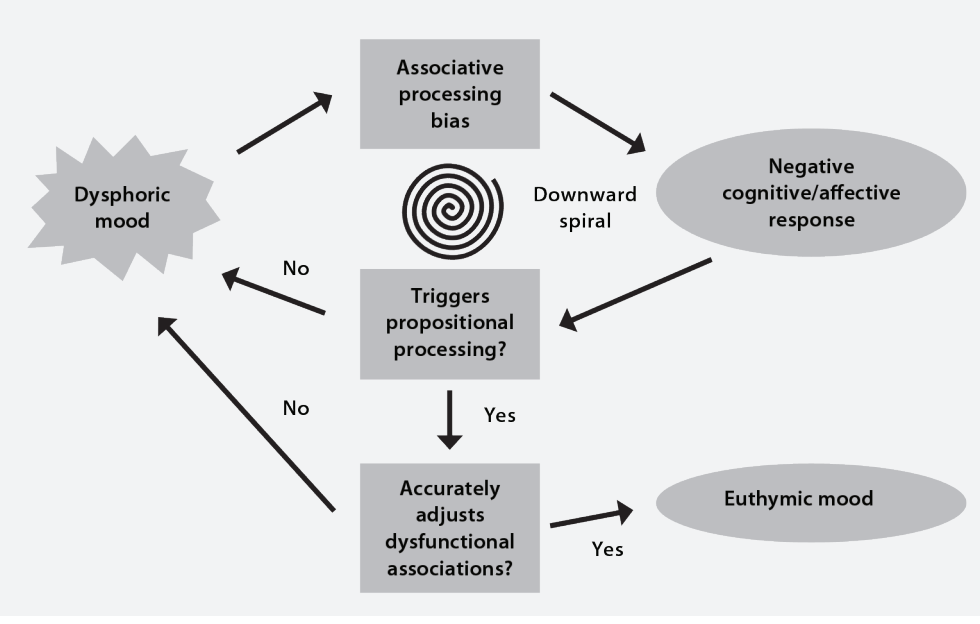
addition, (3) the role of dysfunctional associations should be examined for maintaining anxiety disorders over time. Finally, (4) it would be interesting to prospectively study the predictive validity of dysfunctional automatic associations for the onset of anxiety disorders, to test whether they can be seen as a pre-morbid vulnerability factor. In line with the multi-process model (Ouimet et al., 2009) one would expect that anxious individuals are characterized by stronger dysfunctional automatic self-associations and more specific threat-related associations. Also within anxious individuals, stronger dysfunctional automatic self-associations might be related to the persistence of anxiety, thus, to an unfavourable course of the anxiety disorder. If dysfunctional automatic self-associations are indeed a pre-morbid vulnerability factor for anxiety disorders, they should be able to predict which individuals develop an anxiety disorder over time.

## Disorder-specific dual process models: depression

In 2005, Christopher Beevers proposed a dual process model for the cognitive vulnerability to depression (see Fig. 1.3). He presumes that in individuals vulnerable to depression, dysphoric mood serves as a trigger for negatively biased associative processing. Dysfunctional automatic associations that become activated lead to negative cognitive and affective responses. Since associative processing is occurring by default, a negative feedback loop can develop over time maintaining the dysphoric mood. Only when expectations are violated, propositional processes are being triggered which can interrupt the feedback loop (Beevers uses the term reflective processing). However, not all propositional processes necessarily lead to an adjustment of the dysphoric mood. That is, when the propositional reasoning does not effectively correct the associative bias, like for example in rumination, again it works in a way to maintain the dysphoric mood. The same happens when propositional processes are triggered, but insufficient cognitive recourses are available. In addition, the negative mood state itself can even further deplete the available cognitive recourses, decreasing the chance of disrupting the dysphoric mood. Through these cognitive mechanisms a downward spiral can develop which results in the maintenance and/or aggravation of the negative mood state. As a consequence, it becomes increasingly difficult for a person to disengage from negative thinking, eventually resulting in a depressive episode. Only when propositional processes are triggered, sufficient cognitive resources are available, and when reasoning leads to an adjustment of the associative bias, a more neutral/positive mood can be restored.

In the dual process model for depression, Beevers builds further on other cognitive models of depression, such as the diathesis-stress theory (e.g., Clark et al., 1999) in which

Figure 1.3
*Main elements of Beevers (2005) dual process model for depression*




dysfunctional processing biases (negative schema's) are seen as latent vulnerability factors for depression that can get activated under the influence of certain (contextual) triggers. Schemas are assumed to be more or less enduring cognitive structures which organize people's thoughts, feelings, and behaviours into stable patterns. In depression, dysfunctional schemas towards the self are considered to be especially important concerning themes of loss and worthlessness (e.g., Beck et al., 1979; Beevers, 2005; Ingram et al., 1998). The repeated activation of negative schemas during a depressive episode might result in an associative memory network in which the self becomes increasingly and automatically linked to negative attributes. This way, negatively biased automatic self-associations might form an important factor in the maintenance of depression.

## *Available evidence*

Since dysfunctional schemas towards the self are assumed to play an important role in depression, most research in this area focused on automatic self-associations, particularly global affective self-associations (so-called *implicit self-esteem*). In line with the cognitive model of depression, self-report measures provided overwhelming evidence that depressed individuals indeed show lower 'explicit self-esteem' than healthy controls (Ingram et al., 1998). However, with respect to implicit self-esteem no consistent differences were found between depressed and healthy individuals. Whereas two studies found evidence

for a lowered implicit self-esteem in individuals with current depression (Franck, De Raedt, Dereu & Van den Abbeele, 2007; Risch et al., 2010) and one study in students at risk of becoming depressed (Steinberg, Karpinski & Alloy, 2007), the majority of studies showed no systematic differences in implicit self-esteem between depressed individuals and healthy controls (De Raedt, Schacht, Franck & De Houwer, 2006; Franck, De Raedt & De Houwer, 2007, 2008; Gemar, Segal, Sagrati & Kennedy, 2001; Valiente et al., 2011). Moreover, these studies not only failed to find evidence for negative implicit self-esteem in depressed patients, but even showed enhanced implicit self-esteem in people who were remitted from depression (e.g., Franck, De Raedt & De Houwer, 2008; Gemar, Segal, Sagrati & Kennedy, 2001). In contrast, an experimental study among non-clinical undergraduates showed that low implicit self-esteem had predictive validity for immediate affective reactions after a lab stressor, which is in line with the cognitive model (Haeffel et al., 2007: study 1). In addition, lower implicit self-esteem interacted with life stress in predicting depressive symptoms over time (Haeffel et al., 2007: study 2; Steinberg, Karpinski & Alloy, 2007). However, surprisingly, another study among formerly depressed individuals and never depressed controls showed exactly the opposite, namely that higher levels of implicit self-esteem were associated with higher levels of depressive symptoms six months later (Franck, De Raedt & De Houwer, 2007).

### *Open questions*

It is difficult to explain these mixed findings with the dual process model of depression (Beevers, 2005) or with the diathesis-stress model (Clark et al., 1999). Perhaps the outcomes are the result of specific study samples that were selected. For example, there is some evidence indicating that implicit self-esteem becomes lowered when individuals have had several depressive episodes (Risch et al., 2010). When most of the study samples included individuals with a first depressive episode, this factor or other confounding factors can have severely influenced the findings, since the sample sizes of these studies generally were small. In addition, there are some important questions in this field that still remain unanswered. Up to now, all studies in this field almost exclusively focused on global self-associations (implicit self-esteem), but implicit self-esteem somehow might not capture the automatic self-associations that are important in depression. Perhaps, analogous to self-anxious associations in anxiety disorders, more specific self-depressed associations may be relevant in depression. In line with this hypothesis, one study already showed that former depressed patients had relatively strong automatic associations between self and depressed mood than healthy controls (Meites, Deveney, Steele, Holmes & Pizzagalli,

2008). Furthermore, prior studies typically compared (remitted) depressed groups with healthy control groups, making it impossible to compare dysfunctional automatic self-associations of depressed individuals with other psychopathological disorders, such as anxiety disorders. Finally, prior studies were mostly conducted over a relatively short time period, leaving undecided whether dysfunctional automatic self-associations are also involved in the naturalistic course and maintenance of depression over a longer period of time. Based on the dual process model of Beevers (2005), one would not only expect that depressed individuals are characterized by stronger dysfunctional automatic self-associations than healthy controls and clinical controls, but also that within depressed individuals, stronger dysfunctional automatic self-associations might be related to the persistence of dysphoric mood and, thus, to an unfavourable course of the depressive symptoms.

Therefore, it seems paramount to further expand the existing literature in this field with research focusing on (1) more specific automatic self-depressed associations (2) in a large sample of (remitted) depressed patients (3) including both healthy controls and clinical controls. When depressed patients are indeed characterized by more specific automatic self-depressed associations, (4) an important next step would be to prospectively examine the long-term predictive validity of dysfunctional automatic self-associations for the course of depression.

## Implicit Association Test

During the past decades, several measurement instruments have been developed to measure automatic associations (see for an overview: Fazio & Olson, 2003). In the present series of studies, we consistently used one task, the Implicit Association Test (IAT), and a modified version of this task, the single-target Implicit Association Test (stIAT). The IAT was developed over a decade ago by Greenwald, McGhee and Schwartz (1998). The IAT is a computer task that was designed to measure the relative strength of automatic associations between two contrasted target concepts (e.g. *me* and *other*) and two attribute concepts (e.g. *anxious* and *calm*). The participants' task is to sort words (stimuli) from these four concepts which appear in the middle of a computer screen. The words have to be sorted by means of two response buttons: two concepts share one button (e.g. *me* and *anxious*) and two concepts share the other button (e.g. *other* and *calm*). During the task the concept labels stay visible in the upper right and left corners of the screen. After a block of trials, the concepts switch (e.g. now *me* and *calm* share one button and *other* and *anxious* share the other button). The idea is that it is easier to sort words of two concepts with the same button when these concepts are strongly associated in memory resulting

in faster reaction times. By comparing the mean reaction times for the different pairings of concepts, one can calculate the so-called IAT-effect, which is thought to be a measure for the relative strength of the association.

With respect to psychometric properties, the IAT is one of the better implicit measures (e.g., Bosson, Swann & Pennebaker, 2000), although the task shows lower test-retest reliability than most self-report measures (Greenwald & Nosek, 2001). However, higher test-retest reliability of self-report measures could also be an overestimation of true reliability of self-report measures, caused by social desirability and memory-effects. Correlations between the IAT and explicit equivalents are usually low (Hofmann, Gawronski, Gschwendner, Le & Schmitt, 2005), which could reflect that both types of measures measure genuinely different cognitive processes. The concepts and stimuli of the IAT can be easily adjusted to the specific construct of interest. It can be successfully administered in children and adults with a minimum of education. The task is simple, takes only ten minutes and most participants find it fun to do. As already was discussed, the task successfully seems to distinguish between experimental groups and controls. Furthermore, there are some indications that the task is sensitive to the influence of treatment and has specific predictive validity for dysfunctional behaviours.

## Outline thesis

The main aim of this dissertation is to get more insight into the role of dysfunctional automatic associations in the cognitive vulnerability to depressive and anxiety disorders. In order to do this, I will present seven studies in clinical samples of treatment-seeking anxious and depressed patients using both cross-sectional and prospective designs.

Chapter 2 *IAT algorithms* will start off discussing an important methodological issue, that is, which algorithm should be used to calculate the so-called IAT effect. Although an influential study of Greenwald, Nosek and Banaji (2003) showed that so-called D-measures perform best in internet samples, it is still unclear whether this also yields for laboratory studies. Consequently, we decided to compare different algorithms for the Implicit Association Test in a laboratory setting.

Chapter 3 *dysfunctional automatic self-associations in anxiety and depression* contains two studies. In Study 1 *group differences*, we examine whether patients with anxiety disorders and/or depressive disorders are characterized by dysfunctional automatic self-associations. Following the design of Egloff and Schmukle (2002), we studied automatic self-anxious associations as a shared vulnerability factor across different anxiety disorders and equivalently, automatic self-depressed associations as vulnerability factor for depression.

We hypothesize that when automatic self-anxious and self-depressed associations would be vulnerability factors for developing/maintaining anxiety and depression respectively, individuals with current anxiety disorder and/or depression would differ on these associations from healthy controls. In addition, because we include both patients with anxiety and depressive disorders, we can test whether these self-associations are specific to anxiety and depression, or a more general characteristic of psychopathology. Furthermore, we investigate whether dysfunctional automatic self-associations represent a relatively stable characteristic that remains unchanged even after recovery of anxiety and/or depressive disorder. Therefore, we test whether remitted individuals are still characterized by enhanced automatic self-depressed/anxious associations. If dysfunctional automatic self-associations are a stable cognitive feature of these disorders, we would expect them to be still present in remitted individuals. In Study 2 *suicidal ideation*, we explored whether automatic self-associations could also help to improve our understanding of suicidal ideation, a psychopathological symptom that occurs both in depressive disorders and in anxiety disorders. We were inspired by the findings that automatic self-associations seem to display predictive validity specifically for more spontaneous, uncontrollable kind of behaviours such as autonomic responding and nonverbal behaviours (e.g., Asendorpf et al., 2002; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a; Spalding & Hardin, 1999). Building on this, we hypothesized that dysfunctional automatic self-associations might also contribute to the onset and maintenance of suicidal ideation, since suicidal patients often report difficulties in controlling their suicidal thoughts and preventing them from repetitively entering their awareness. Because not only depression, but also anxiety has been linked to suicidal ideation (e.g., Ellis, 2006, Norton, Temple & Pettit, 2008; Sareen et al., 2005), we hypothesize that automatic self-depressed associations as well as automatic self-anxious associations might relate to suicidal ideation.

Chapter 4 *dysfunctional automatic associations in social anxiety disorder* focuses on more disorder-specific automatic associations in the context of social anxiety disorder. In Study 1 *fear of blushing*, we examine automatic blushing-association in socially anxious individuals with a fear of blushing, whereas in Study 2 *social anxiety disorder* implicit self-esteem and automatic associations with social cues are studied in social anxiety disorder. In the second study we not only include a healthy control group, but also a clinical control group of panic disorder patients. If lowered implicit self-esteem and dysfunctional automatic associations with social cues are specific vulnerability factors to social anxiety disorder, they should not also be present in panic disorder. Both studies are the first to examine these automatic associations in clinical samples of treatment-seeking individuals

with social anxiety disorder.

Chapter 5 *dysfunctional automatic self-associations over time* covers two prospective studies. Study 1 *maintenance of anxiety and depression* focuses on the predictive validity of dysfunctional automatic self-associations for the maintenance of both anxiety and depressive disorders. In line with both dual process models of the vulnerability to anxiety and depression (Beevers, 2005; Ouimet et al., 2009), we would expect that stronger dysfunctional automatic self-associations are related to the persistence of anxiety and depressive disorders, and thus, to an unfavourable course of these disorders. In Study 2 *onset of anxiety disorders*, we zoom in further on automatic self-anxious associations as a vulnerability factor for developing anxiety disorders by testing their prognostic value for the onset of anxiety disorders between baseline and 2-year follow-up. If dysfunctional automatic self-anxious associations are indeed a pre-morbid vulnerability factor for anxiety disorders, they should be able to predict which individuals develop an anxiety disorder over time.

Finally, Chapter 6 provides an integration and discussion of the results presented in the empirical chapters (Chapters 2 to 5). The outcomes of this thesis will be discussed focussing on the following four questions: (1) which algorithm performs best in calculating the IAT- effect in a laboratory setting? (2) are dysfunctional automatic self-associations vulnerability factors for developing anxiety and/or depressive disorders? (3) are dysfunctional automatic self-associations maintaining factors for anxiety and/or depressive disorders? and (4) are dysfunctional automatic associations specific characteristics of certain anxiety and/or depressive disorders or shared characteristics across several disorders?

# 2 IAT algorithms

## Abstract

In their paper, "Understanding and using the Implicit Association Test: I. An improved scoring algorithm", Greenwald, Nosek and Banaji (2003) investigated different ways to calculate the IAT-effect. However, up to now, it remained unclear whether these findings -based on internet data-, also generalize to laboratory settings. Therefore, the main goal of the present study was to cross-validate scoring algorithms for the IAT in a laboratory setting, specifically in the domain of psychopathology. Three known IAT algorithms and six alternative IAT algorithms were evaluated on several performance criteria in the large-scale laboratory sample of the Netherlands Study of Depression and Anxiety ($N = 2981$) in which two IATs were included to obtain measurements of automatic self-anxious and automatic self-depressed associations. Results clearly demonstrated that the $D_{600}$-measure is suitable to be used in a laboratory setting for IATs with a fixed order of category combinations. It remains important to further replicate these findings, especially in studies that include outcome measures of more spontaneous kinds of behaviours.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## Introduction

During the past two decades, an increased interest for implicit associations has also spread to the field of psychopathology (e.g., de Houwer, 2002) with the Implicit Association Test (IAT; Greenwald, McGhee & Schwartz, 1998) as one of the most frequently used measurement instruments (for an example of an IAT design see block 1 – 7 of Table 2.1). This kind of research is inspired by recent information-processing models that emphasize the importance to distinguish between more explicit and more automatically activated cognitions. Both types of cognitions are believed to have different functional qualities (e.g. Gawronski & Bodenhausen, 2006) and influence different kinds of behaviours. While explicit cognitions are assumed to predict more deliberate, controlled behaviours, automatic associations are thought to play an important role in guiding relatively spontaneous, uncontrollable behaviours (e.g. Spalding & Hardin, 1999; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a). The latter kinds of behaviours are also critically involved in psychopathology where patients often report symptoms being unpredictable and uncontrollable (e.g., Mayer et al. 2000).

Table 2.1
*Arrangement of Implicit Association Test blocks*

| Block | No. of trials | Function | Labels assigned to left-key response | Labels assigned to right-key response |
|---|---|---|---|---|
| 1 | 20 | Practice | me | other |
| 2 | 20 | Practice | anxious | calm |
| 3 | 20 | Practice | me + anxious | other + calm |
| 4 | 60 | Test | me + anxious | other + calm |
| 5 | 20 | Practice | calm | anxious |
| 6 | 20 | Practice | me + calm | other + anxious |
| 7 | 60 | Test | me + calm | other + anxious |
| 8 | 20 | Practice | depressed | elated |
| 9 | 20 | Practice | me + depressed | other + elated |
| 10 | 60 | Test | me + depressed | other + elated |
| 11 | 20 | Practice | elated | depressed |
| 12 | 20 | Practice | me + elated | other + depressed |
| 13 | 60 | Test | me + elated | other + depressed |

Despite the frequent use of the IAT within psychopathology research (review: Roefs et al., 2011), there are still several unsolved methodological and conceptual issues regarding what the IAT actually measures (e.g., Conrey, Sherman, Gawronski, Hugenberg & Groom, 2005; Fiedler, Messner & Bluemke, 2006) and to what extent IAT-effects really reflect 'im-

plicit' or 'automatic' cognitive processes (De Houwer, 2009). The present paper focuses on one specific methodological issue, namely how response latencies of the IAT could be transformed into an 'IAT-effect'. In their paper, 'Understanding and using the Implicit Association Test: I. An improved scoring algorithm', Greenwald, Nosek and Banaji (2003) thoroughly investigated different ways to calculate the IAT-effect. They showed that in large datasets collected through the internet the so-called D-measures perform best. However, it is still unclear whether these findings also generalize to laboratory settings, which are common in psychopathology research. Therefore, the main goal of the present study was to cross-validate scoring algorithms for the IAT in a laboratory setting in the domain of psychopathology. Given the dominant influence of the IAT in this field, a better understanding of its scoring procedures seems crucial.

In contrast to laboratory settings, internet studies almost completely lack experimental control, which could lower the commitment of participants to the task and by this, create more lapses of attention. Because short periods of inattention probably increase both the average and the variability of reaction times (RTs), it might be that the superior performance of D-measures (that correct for variability by dividing by the pooled SD) is limited to situations without experimental control. That is, D-measures not necessarily also improve the sensitivity of the IAT-effect under laboratory conditions. Consistent with the suggestion that the D-measure might in fact be suboptimal for indexing IAT effects of laboratory studies, some studies in the alcohol-domain (Wiers, Rinck, Kordts, Houben & Strack, 2010; Wiers, van de Luitgaarden, van den Wildenberg & Smulders, 2005), found expected changes in IAT-scores as a result of a cognitive behavioural intervention using the original algorithm, but not with D-measures. This could be related to the more controlled lab-circumstances, to the within-subjects designs used (which were not used in the original validation study of Greenwald et al., 2003), or just reflect chance findings, given the relatively small sample sizes of these studies. In any case, currently, most studies using the IAT only report results of D-measures, which makes it hard to compare the performance of various scoring algorithms across different settings.

How should we judge which IAT algorithm performs best? Greenwald and colleagues (2003) formulated several criteria on which they compared the performance of different IAT algorithms: correlation with explicit measures; correlation with average response latency; internal consistency; sensitivity to undesired influence of order effects of the combined task; resistance to the effect of prior IAT experience; effect size; and magnitude of the implicit-explicit path. The correlation with the explicit equivalent was identified as one of the most important performance criteria. Based on the assumption that implicit and

explicit measures share one underlying association they assumed that a higher correlation is preferable. As a second important performance criterion, the correlation with general response speed was used, because people with a slower overall response tendency generally show larger IAT-effects. As a consequence, conceptually unrelated IAT-effects can have substantial correlations (McFarland & Crouch, 2002; Mierke & Klauer, 2003). The IAT-effect is confounded by general cognitive abilities that are unrelated to the construct of interest (Mierke & Klauer, 2003), and therefore, an IAT-effect that shows smaller correlations with general response speed seems preferable (Greenwald et al., 2003).

In addition to the criteria that Greenwald and colleagues formulated (2003), we used two other performance criteria to evaluate the performance of IAT algorithms. First of all, we included predictive validity as criterion by examining the ability of the IAT to predict relevant outcome measures (Blanton & Jaccard, 2006). Greenwald, Poehlman, Uhlman and Banaji (2009) already conducted a large meta-analysis of the predictive validity of the IAT. Although the meta-analysis included IAT scoring method as a methodological moderator, the performance of different IAT algorithms with respect to predictive validity could not be compared within one sample. Second, reliability is considered an important characteristic of psychological measures. In situations where there is no reason to assume that the construct of interest has changed between two points in time, the correlation between the outcomes should typically be high. In the present study, test-retest reliability will be tested in a control group that was repeatedly assessed but did not receive an intervention between the time points.

To summarize, the present study is an extension of the work of Greenwald and colleagues (2003) with as main goal to cross-validate scoring algorithms for the IAT in a laboratory setting in the domain of psychopathology. Therefore, the three known IAT algorithms will be compared on several performance criteria, including predictive validity and test-retest reliability. In addition, this study will explore the performance of six alternative IAT algorithms. It is very hard to find laboratory data-sets that are sufficiently large to achieve the required power for the purpose of the present enterprise. Fortunately, we had access to the unique, large-scale sample of Netherlands Study of Depression and Anxiety (NESDA; $N = 2981$) in which participants carried out two IATs in a laboratory setting. The IATs were designed to measure automatic self-anxious and automatic self-depressed associations, respectively (cf. Egloff et al., 2002). Data were collected among both patients and non-clinical controls and the assessment was repeated after two years (Penninx et al., 2008).

# Method

## *IAT Algorithms*

Nine different IAT algorithms were tested: three were the same as in the study of Greenwald and colleagues (2003): $D_{600}$-measure, $C_1$-measure and $C_3$-measure. Furthermore, six alternative algorithms were tested: $D_{noSD}$-measure, $D_{noSD+log}$-measure, GRS-measure, d-measure, S-measure and P-measure. In line with the recommendations of Greenwald and colleagues, we decided to apply two basic principles to all the algorithms: 1) participants with more than 10 % of the RTs below 300 milliseconds (ms) were discarded from the analyses; 2) error trials were replaced with mean reaction times of correct responses in the block in which the error occurred plus a penalty of 600 ms, because the present IAT design did not record the second correct response after a mistake, so no built-in error penalty could be used. In addition, subjects with scores diverging more than 4 SDs from the mean were discarded from the analyses (see *Missing data and construction of groups*). Main characteristics of the measures and rationales behind the new measures will be briefly discussed below. A detailed overview of the characteristics of the algorithms can be found in Table 2.2 and Table 2.3.

Table 2.2
*Characteristics of known IAT algorithms*

| | **$D_{600}$-measure (Greenwald et al., 2003)** | **$C_1$-measure (Greenwald et al., 1998)** | **$C_3$-measure (Greenwald et al., 2003)** |
|---|---|---|---|
| Which trials? | Practice and test<br>Include first trials | Test only<br>Exclude first trials | Practice and test<br>Include first trials |
| Treatment extremes | Exclude trials > 10,000 ms | Recode RTs < 300 ms and > 3000 ms | Recode RTs < 300 ms and > 3000 ms |
| Latency transformation | None | Natural log-transformation | Natural log-transformation |
| Other transformation | Divide practice and test difference scores by pooled SD, before taking unweighted mean | None | Unweighted mean practice and test effect |

*Note*. In all algorithms, subjects with more than 10% of their responses below 300 ms were excluded from analysis and error trials were replaced with mean reaction times of correct responses in the block in which the error occurred plus a penalty of 600 ms.

Table 2.3
*Characteristics of new IAT algorithms*

| | $D_{noSD}$-measure | $D_{noSD+log}$-measure | GRS-measure | d-measure | S-measure | P-measure |
|---|---|---|---|---|---|---|
| Which trials? | Practice and test Include first trials | Practice and test Include first trials | Practice and test Include first trials | Practice and test Include first trials | Practice and test Include first trials | Practice only Include first trials |
| Treatment extremes | Exclude trials > 10,000 ms | Exclude trials > 10,000 ms | Exclude trials > 10,000 ms | Exclude trials > 10,000 ms | Exclude trials > 10,000 ms | Exclude trials > 10,000 ms |
| Latency transformation | None | Natural log-transformation | None | None | None | None |
| Other transformations | Unweighted mean practice and test effect | Unweighted mean practice and test effect | Unweighted mean practice and test effect, before dividing this mean by GRS[a] | Calculate Cohen's *d*[b] for practice and test blocks, before taking unweighted mean | Unweighted mean practice and test effect, before recoding effects > 0 into +1 and < 0 and -1 | None |

*Note*. GRS, general response speed; S, sign; P, practice. In all algorithms, subjects with more than 10% of their responses below 300 ms were excluded from analyses. Error trials were replaced with mean reaction times of correct responses in the block in which the error occurred plus a penalty of 600 ms. [a] GRS is defined as the mean RT of the single target practice trials in blocks 1,2, and 5; [b] Cohen's *d* is defined as

$$d = \frac{\bar{x}_1 - \bar{x}_2}{s}, s = \sqrt{\frac{(n_1 - 1)s_1^2 + (n_2 - 1)s_2^2}{n_1 + n_2}}$$

**$D_{600}$-measure.** In the $D_{600}$-measure, both the practice and the test trials were included as well as the first trials of each of these blocks. RTs above 10.000 ms were excluded before mean RTs are calculated for all blocks. For practice trials and test trials separately, the difference scores between the congruent and incongruent blocks were divided by the SDs of these blocks, i.e. the difference score of the practice blocks was divided by the SD based on all practice trials and the difference score of the test blocks was divided by the SD based on all test trials. Then, the unweighted mean of both difference scores was calculated. In addition to the $D_{600}$-measure, Greenwald and colleagues (2003) calculated five other D-measures. However, in the present study no built-in error penalty could be used, which means that $D_1$-measure and $D_2$-measure could not be calculated. Greenwald

and colleagues state that "…for the four D-measures ($D_3$, $D_4$, $D_5$, $D_6$) that replaced error latencies with computed penalties, there were virtually no differences between the two measures that deleted latencies below 400 ms ($D_5$ and $D_6$) and the two that did not ($D_3$ and $D_4$)." In addition, they conclude that "…for IAT procedures that contain no built-in penalty, either of the two penalty formulas used in Study 6 (the 600-ms penalty or the 2x SD penalty) should perform approximately equally." Consequently, we decided to use the $D_4$-measure (that we refer to as '$D_{600}$-measure'), because it keeps the error penalty constant (600 ms) instead of variable (2x SD).

**$C_1$-measure.** The $C_1$-measure ('C' for 'conventional') was only based on the test trials, only excluding the first two trials of the blocks as 'warm-ups'. RTs below 300 ms and above 3000 ms were recoded to respectively 300 and 3000 ms. RTs were log-transformed before the mean score for each block was calculated.

**$C_3$-measure.** The $C_3$-measure was almost similar to the $C_1$-measure. The only difference was that both the test and the practice trials were included in the algorithm, instead of the test trials only. The unweighted mean of both difference scores (practice and test) formed the $C_3$-measure.

**$D_{noSD}$-measure.** To investigate whether dividing by the SD was indeed improving the IAT-effect, or whether the other ingredients of the D-measure caused this effect, the D-measure was tested without dividing by the SD. The remaining transformations were the same as with the $D_{600}$-measure.

**$D_{noSD+log}$-measure.** This measure was very similar to the $D_{noSD}$-measure and the $C_3$-measure. The difference with the $D_{noSD}$-measure is that the RTs were log-transformed before averaging and the difference with the $C_3$-measure is that RTs above 10,000 ms were excluded instead of recoding RTs below 300 ms and above 3000 ms.

**GRS-measure.** If correcting for general response speed is an important element of the IAT algorithm, it might be useful to directly divide the difference score by the general response speed (GRS), instead of correcting for it by dividing by the SD. The GRS-measure was otherwise similar to the $D_{600}$-measure, but instead of dividing each difference score by its SD, the unweighted mean difference scores of the practice and test blocks were divided by general response speed. GRS was defined here as the mean RT of the single target practice trials.

**d-measure.** Cohen's *d* is a widely used way to calculate effect size. Therefore, it makes sense to test the performance of this measure as well. The d-measure was again similar to the $D_{600}$-measure, but now Cohen's *d* was calculated. For the d-measure, the difference score was divided by the standard deviation. This was done for the test and practice trials

separately, before the unweighted mean was taken. The difference between the present d-measure and the $D_{600}$-measure is that the SD in the denominator of the $D_{600}$-measure is computed from all scores in both conditions (congruent and incongruent blocks taken together), ignoring the condition membership of each score. By contrast, the SD used in computing the effect size of Cohen's *d* is a within-condition SD (congruent and incongruent blocks separately).

**S-measure.** S stands for 'sign'. In this algorithm, practice as well as test trials were included. After the unweighted mean of the difference scores was calculated, these effects were being recoded in a dichotomous variable according to their sign. Values above zero were recoded into +1 and values below zero were recoded into -1. The advantage of this measure could be that it is quite robust against how exactly the IAT-effect was calculated. The S-measure only shows which combination (congruent or incongruent) someone finds more difficult. Therefore, task-specific variance probably will have less influence, because the chance that it will change the sign is small.

**P-measure.** Greenwald and colleagues (2003) found that correlations with explicit measures were higher for the IAT measures based on the practice blocks than for the measures based on test blocks. Perhaps this has to do with the tendency of IAT-effects to decrease when individuals have more experience conducting the IAT (e.g., Greenwald & Nosek, 2001; Wiers et al., 2005). It might be that the most important information can be obtained from the first few trials. Following this perspective, we decided to test an algorithm that only includes the practice trials, i.e. the P-measure was calculated as the difference score between the congruent and incongruent practice blocks.

### *Criteria and corresponding data analyses*

The IAT-algorithms were examined according to the following seven performance criteria: 1) IAT correlations with explicit measures (high values desired); 2) Correlations of IAT with response latency (approaching zero desired); 3) Internal consistency (high values desired); 4) Resistance to undesired influence of prior IAT experience; 5) IAT effect size (high values desired); 6) Predictive validity (high values desired); 7) Test-retest reliability (high values desired).

**1. IAT correlations with explicit measures.** Correlations between IAT measures and explicit equivalent measures were calculated.

**2. Correlations of IAT with response latency.** We used the mean response speed of the four combined blocks as measure of general response speed (GRS). Greenwald and colleagues (2003) calculated GRS without excluding trials above 10,000 ms. However, we

did not want extreme RTs to influence GRS and excluded trials above 10,000 ms before calculating GRS. Correlations were calculated between the absolute IAT effects and GRS.

**3. Internal consistency.** Greenwald and colleagues (2003) defined the internal consistency of the IAT as the correlation between IAT-effects based on two mutually exclusive subsets. As subsets they used the practice- and test trials, and when this was not possible, the first and the second half of the blocks were used. A higher correlation points to a stronger internal consistency and indicates a better measure. However, during the administration of the IAT, usually learning effects occur, and therefore, the correlation between IAT-effects based on test and practice trials (or first and second block halves) could in fact underestimate the internal consistency. Therefore, we applied a slightly different definition and calculated the Spearman-Brown corrected correlations between IAT-effects based on two mutually exclusive subsets of 'odd and even trials' (test-halves were based on trials 1, 2, 5, 6, 9, 10 etc. vs. 3, 4, 7, 8, 11, 12 etc.).

**4. Resistance to undesired influence of prior IAT experience.** Additionally, Greenwald and colleagues (2003) looked at the correlation of the IAT-effect with prior IAT experience. IAT effects tend to decrease with the number of IATs presented to a participant; therefore the algorithm should reduce this influence as much as possible. We tested this in the healthy control group, with repeated measures ANOVA in which Time was included as within-subject factor. The effect of time ($\eta^2$) is preferably small.

**5. IAT effect size.** The IAT should be sensitive enough to detect individual or group differences. This means that an algorithm that maximizes IAT effect sizes is preferable. Sensitivity for differences between groups (anxious/depressed vs. controls) was evaluated using t-tests and one-sample t-tests were used to test which algorithm was most sensitive to pick up differences between the congruent and incongruent condition of the IAT. For all t-tests Cohen's *d* was calculated.

**6. Predictive validity.** To test the predictive validity, multiple regression analyses were conducted for the different algorithms separately using depressive and anxiety symptoms as dependent variables. In the result section $R^2$ is reported. A description of the symptom measures can be found below.

**7. Test-retest reliability.** This criterion was tested in the control group that did not have a depressive or anxiety disorder. The control group was repeatedly assessed but received no intervention or treatment in between the two assessments. Correlations were calculated between the first and second IAT-effect.

### *Study sample*

The present study was carried out in the context of the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008), a multi-center, ongoing cohort study, designed to examine the long-term course and consequences of anxiety and depressive disorders. A total of 2981 persons aged 18 through 65 were included, including healthy controls, individuals at risk because of prior episodes, sub threshold symptoms or family history, and individuals with a current first or recurrent depressive and/or anxiety disorder. The inclusion was restricted to Major Depressive Disorder, Dysthymia, General Anxiety Disorder, Panic Disorder, Social Phobia, and Agoraphobia, because these disorders are relatively homogeneous in phenotype and are found across different health care settings. Recruitment of respondents took place in the general population, in general practices, and in mental health care institutions. General exclusion criteria were having a primary clinical diagnosis of a psychiatric disorder not subject of NESDA which would importantly affect course trajectory (i.e., psychotic disorder or bipolar disorder) and not being fluent in Dutch. The present study concerns baseline and 2-year follow-up measurements conducted from September 2004 until April 2009. The study protocol was approved centrally by the Ethical Review Board of VU Medical Center Amsterdam and subsequently by local review boards of each participating center/institute, and all participants provided written informed consent.

After two years, a face-to-face follow-up assessment was conducted with a response of 87.1 % ($N$ = 2596). Non-response was significantly higher among those with younger age, lower education, non-European ancestry, and depressive disorder, but was not associated with gender or anxiety disorder (Lamers et al., 2011). The presence of depressive or anxiety disorders was established with the Composite International Diagnostic Interview (CIDI; WHO version 2.1) which classifies diagnoses according to DSM-IV criteria (American Psychiatric Association, 2000).

### *Measures*

**Implicit Association Test.** The Implicit Association Test (IAT) is a computerized reaction time task originally designed by Greenwald and colleagues (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left or right response key. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are

weakly associated. The category labels are visible in upper left and right-hand corners of the screen during the whole task. For both IATs target labels were *me* and *others*. Following the design of Egloff and Schmukle (2002), an anxiety IAT was constructed with attribute labels *anxious* and *calm*. Analogously, attribute labels were *depressed* and *elated* for the depression IAT. Each category consisted of five stimuli (see Appendix 2.A). Attribute stimuli of the anxiety IAT were the same self-descriptors as used by Egloff and Schmukle (2002) who based their IAT on trait anxiety. Furthermore, we designed a self-depressed IAT in an equivalent way and selected trait self-descriptors of depressed persons that were also used in previous work on attentional bias in (remitted) depression (e.g., McCabe, Gotlib & Martin, 2000). Both IATs consisted of two critical test blocks that were preceded by practice blocks (see Table 2.1). The order of category combinations was fixed across participants to reduce method variance.

**Explicit self-associations.** To obtain explicit self-associations equivalently to the automatic self-associations, participants rated all IAT attribute stimuli on a 5-point scale (1 = hardly/not at all, 5 = very much) (i.e., "For each word please indicate to what extent you think it generally applies to you").

**Questionnaire data.** Severity of anxiety symptoms was measured with the 21-item Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988), whereas fearful avoidance behaviour was measured using the 15-item Fear Questionnaire (FQ; Marks & Mathews, 1979). Severity of depressive symptoms was measured with the 30-item Inventory of Depressive Symptoms self-report version (IDS-SR; Rush, Gullion, Basco & Jarrett, 1996). Total scale scores were used for all questionnaires.

### *Procedure*

Baseline and follow-up assessments were similar and lasted between 3 and 5 hours. During assessments, other measurements were collected as well, but these are not of interest for the present study (for a detailed description, see Penninx et al., 2008). Each participant completed the anxiety IAT, followed by the depression IAT. After that, participants explicitly rated attribute words that were used in the IATs. Respondents were compensated with an €15,- gift certificate and travel expenses.

### *Missing data and construction of groups*

Although the present sample was particularly large, the drop-out was considerable. In addition to the 'regular attrition' of the NESDA study, there was extra attrition for the IAT. Sometimes individuals were willing to participate in the follow-up assessment, but were

measured at home or via the telephone, resulting in a loss of IAT data. Consequently, IAT data and explicit self-associations for 129 participants were missing at t1 and 564 were missing at t2. Participants with more than 10% of the trials below 300 ms were discarded from all analyses (IAT anxiety: $n_{t1} = 7$, $n_{t2} = 7$; IAT depression: $n_{t1} = 1$, $n_{t2} = 1$). Consequently, the total sample consisted of 2845 participants at t1 and 2030 participants at t2. In addition, for the analyses of criterion 1 *predictive validity* 35 individuals were discarded because of missing data on the BAI and FQ (100 missings at t2) and 39 on the IDS-SR (93 missings at t2). Criterion 2 *test-retest reliability* and criterion 4 *resistance to undesired influence of prior IAT experience* were tested in the group of control participants that did not have a disorder during or in between baseline and follow-up ($n = 821$). Finally, for criterion 5 *IAT effect size* the sensitivity for differences between groups was tested (anxious group: $n_{t1} = 512$, $n_{t2} = 439$; depressed group: $n_{t1} = 283$, $n_{t2} = 274$; controls: $n_{t1} = 648$, $n_{t2} = 439$). The control group was smaller in these analyses, because we additionally excluded individuals that had a prior depressive or anxiety disorder (cf. Glashouwer & de Jong, 2010).

In addition, subjects with scores diverging more than 4 SDs from the mean were discarded from the analyses (IAT anxiety: $D_{600}$-measure: $n_{t1} = 0$, $n_{t2} = 0$; $C_1$-measure: $n_{t1} = 10$, $n_{t2} = 3$; $C_3$-measure: $n_{t1} = 1$, $n_{t2} = 3$; $D_{noSD}$-measure: $n_{t1} = 20$, $n_{t2} = 15$; $D_{noSD+log}$-measure: $n_{t1} = 3$, $n_{t2} = 4$; GRS-measure: $n_{t1} = 8$, $n_{t2} = 5$; d-measure: $n_{t1} = 21$, $n_{t2} = 14$; S-measure: $n_{t1} = 0$, $n_{t2} = 0$; P-measure: $n_{t1} = 15$, $n_{t2} = 12$; IAT depression: $D_{600}$-measure: $n_{t1} = 2$, $n_{t2} = 3$; $C_1$-measure: $n_{t1} = 8$, $n_{t2} = 3$; $C_3$-measure: $n_{t1} = 3$, $n_{t2} = 4$; $D_{noSD}$-measure: $n_{t1} = 18$, $n_{t2} = 11$; $D_{noSD+log}$-measure: $n_{t1} = 5$, $n_{t2} = 4$; GRS-measure: $n_{t1} = 7$, $n_{t2} = 7$; d-measure: $n_{t1} = 13$, $n_{t2} = 9$; S-measure: $n_{t1} = 0$, $n_{t2} = 0$; P-measure: $n_{t1} = 9$, $n_{t2} = 8$).

## Results

The results of both IATs separately can be found in Table 2.4 and 2.5. In Table 2.6 the mean performance of the different algorithms are shown.

### *1) IAT correlations with explicit measures*

The $D_{600}$-measure consistently showed the highest correlation with the explicit equivalent. The correlations of the $C_3$-measure and the d-measure were closest to that of the $D_{600}$-measure.

### *2) Correlations of IAT with response latency*

The $D_{600}$-measure showed the lowest mean correlation with GRS. The performance of the $C_3$-measure and the d-measure were closest to that of the $D_{600}$-measure.

Table 2.4

*Performance of 9 IAT algorithms on 7 criteria in NESDA study, IAT anxiety*

| | Known algorithms | | | New algorithms | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Performance criteria** | **$D_{600}$** | **$C_1$** | **$C_3$** | **$D_{noSD}$** | **$D_{noSD+log}$** | **GRS** | **d** | **S** | **P** |
| 1. Correlation w explicit equivalent | | | | | | | | | |
| T1 EA anxiety | .374 | .329 | .356 | .297 | .350 | .336 | .364 | .308 | .276 |
| T2 EA anxiety | .355 | .295 | .334 | .287 | .333 | .328 | .334 | .263 | .217 |
| 2. Correlation w response speed | | | | | | | | | |
| T1 | .091 | .201 | .195 | .502 | .279 | .259 | .132 | | .509 |
| T2 | .041 | .199 | .175 | .473 | .223 | .227 | .060 | | .443 |
| 3. Internal consistency | | | | | | | | | |
| T1 | .919 | .910 | .938 | .896 | .934 | .896 | .864 | .729 | .821 |
| T2 | .910 | .907 | .927 | .878 | .922 | .882 | .856 | .717 | .776 |
| 4. Prior IAT experience (in controls) | .021 | .002 | .005 | .000 | .003 | .007 | .015 | .010 | .000 |
| 5. IAT effect size | | | | | | | | | |
| T1 One-sample t-test | .583 | .712 | .563 | .446 | .544 | .500 | .345 | .514 | .319 |
| T1 Group differences | .824 | .657 | .750 | .601 | .733 | .719 | .763 | .664 | .569 |
| T2 One-sample t-test | .841 | .929 | .821 | .698 | .800 | .734 | .620 | .752 | .525 |
| T2 Group differences | .878 | .719 | .832 | .713 | .827 | .809 | .831 | .650 | .679 |
| 6. Predictive validity ($R^2$) | | | | | | | | | |
| T1 BAI | .110 | .090 | .102 | .073 | .098 | .091 | .103 | .080 | .060 |
| T1 FQ | .080 | .059 | .071 | .048 | .068 | .063 | .074 | .054 | .044 |
| T2 BAI | .088 | .058 | .075 | .054 | .073 | .074 | .079 | .058 | .053 |
| T2 FQ | .077 | .049 | .066 | .045 | .066 | .066 | .065 | .048 | .049 |
| 7. Test-retest reliability (in controls) | .540 | .508 | .535 | .427 | .523 | .467 | .496 | .385 | .349 |

*Note*. Abbreviations for 9 measures and 7 performance criteria are explained in detail in the Method section. IAT = Implicit Association Test; BAI = Beck Anxiety Inventory; FQ = Fear Questionnaire; GRS = General Response Speed; EA = Explicit Association.

### *3) Internal consistency*

The $C_3$-measure consistently showed the highest internal consistency. The internal consistencies of the $D_{600}$-measure, the $C_1$-measure and the $D_{noSD+log}$-measure were closest to that of the $C_3$-measure.

### *4) Resistance to undesired influence of prior IAT experience*

The $D_{noSD}$-measure and P-measure were consistently the most resistant to undesired influence of prior IAT experience. $C_1$-measure and the $D_{noSD+log}$-measure performed second and third best on this criterion.

Table 2.5

*Performance of 9 IAT algorithms on 7 criteria in NESDA study, IAT depression*

| | Known algorithms | | | New algorithms | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Performance criteria** | **$D_{600}$** | **$C_1$** | **$C_3$** | **$D_{noSD}$** | **$D_{noSD+log}$** | **GRS** | **d** | **S** | **P** |
| 1. Correlation w explicit equivalent | | | | | | | | | |
| T1 EA depression | .377 | .312 | .369 | .319 | .362 | .360 | .370 | .315 | .302 |
| T2 EA depression | .357 | .291 | .337 | .298 | .336 | .334 | .328 | .277 | .244 |
| 2. Correlation w response speed | | | | | | | | | |
| T1 | .082 | .198 | .189 | .470 | .259 | .244 | .114 | | .502 |
| T2 | .072 | .235 | .234 | .462 | .265 | .219 | .083 | | .451 |
| 3. Internal consistency | | | | | | | | | |
| T1 | .864 | .854 | .890 | .825 | .885 | .840 | .800 | .659 | .718 |
| T2 | .843 | .866 | .884 | .827 | .876 | .839 | .763 | .646 | .683 |
| 4. Prior IAT experience (in controls) | .010 | .002 | .003 | .000 | .002 | .007 | .010 | .008 | .000 |
| 5. IAT effect size | | | | | | | | | |
| T1 One-sample t-test | .588 | .493 | .587 | .495 | .578 | .530 | .404 | .531 | .445 |
| T1 Group differences | .822 | .624 | .767 | .653 | .745 | .710 | .817 | .669 | .591 |
| T2 One-sample t-test | .768 | .588 | .744 | .637 | .742 | .689 | .620 | .680 | .632 |
| T2 Group differences | .953 | .793 | .896 | .739 | .885 | .865 | .885 | .745 | .645 |
| 6. Predictive validity ($R^2$) | | | | | | | | | |
| T1 IDS | .114 | .078 | .107 | .076 | .103 | .102 | .105 | .081 | .066 |
| T2 IDS | .087 | .063 | .075 | .052 | .073 | .074 | .075 | .059 | .040 |
| 7. Test-retest reliability (in controls) | .540 | .509 | .530 | .485 | .530 | .503 | .476 | .285 | .414 |

*Note*. Abbreviations for 9 measures and 7 performance criteria are explained in detail in the Method section. IAT = Implicit Association Test; IDS = Inventory of Depressive Symptomatology; GRS = General Response Speed; EA = Explicit Association.

### *5) IAT effect size*

**One sample t-test.** The $D_{600}$-measure showed the greatest mean effect-size of the one sample t-tests. The effect sizes of the $C_1$-measure and the $C_3$-measure were closest to that of the $D_{600}$-measure.

**Group differences.** The $D_{600}$-measure consistently showed the greatest mean effect-size of the between sample t-tests. The effect sizes of the d-measure and the $C_3$-measure were closest to that of the $D_{600}$-measure.

### *6) Predictive validity*

The $D_{600}$-measure consistently showed the highest predictive validity. The predictive validity of the $C_3$-measure and the d-measure were closest to that of the $D_{600}$-measure.

Table 2.6
*Average performance of 9 IAT algorithms on 7 criteria in NESDA study*

| | Known algorithms | | | New algorithms | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Performance criteria** | **$D_{600}$** | **$C_1$** | **$C_3$** | **$D_{noSD}$** | **$D_{noSD+log}$** | **GRS** | **d** | **S** | **P** |
| 1. Correlation w explicit equivalent | **.366** | .307 | .349 | .300 | .345 | .340 | .349 | .291 | .260 |
| 2. Correlation w response speed | **.072** | .208 | .198 | .477 | .257 | .237 | .097 | | .476 |
| 3. Internal consistency | .884 | .884 | **.910** | .857 | .904 | .864 | .821 | .688 | .750 |
| 4. Prior IAT experience (in controls) | .016 | .002 | .004 | **.000** | .003 | .007 | .013 | .009 | **.000** |
| 5. IAT effect size | | | | | | | | | |
| One-sample t-test | **.695** | .681 | .679 | .569 | .666 | .613 | .497 | .619 | .480 |
| Group differences | **.869** | .698 | .811 | .677 | .798 | .776 | .824 | .682 | .621 |
| 6. Predictive validity ($R^2$) | **.093** | .067 | .083 | .058 | .080 | .078 | .084 | .063 | .052 |
| 7. Test-retest reliability (in controls) | **.540** | .509 | .533 | .456 | .527 | .485 | .486 | .335 | .382 |

*Note*. Abbreviations for 9 measures and 7 performance criteria are explained in detail in the Method section. IAT = Implicit Association Test; GRS = General Response Speed. **Bold** = best mean performance. Underlined: second and third best performance

### *7) Test-retest reliability*

The $D_{600}$-measure consistently showed the highest test-retest reliability. The test-retest reliability of the $C_3$-measure and the $D_{noSD+log}$-measure were closest to that of the $D_{600}$-measure.

## Discussion

The main purpose of the present study was to extend the findings of Greenwald and colleagues (2003) and validate scoring algorithms for the IAT in a laboratory setting in the domain of psychopathology. Therefore, three known IAT algorithms ($D_{600}$-measure, $C_1$-measure and $C_3$-measure; Greenwald et al., 2003) were evaluated on seven performance criteria in the large-scale laboratory sample of the NESDA. In line with the study of Greenwald and colleagues (2003), results demonstrate that the $D_{600}$-measure shows the highest correlation with explicit equivalents and the lowest correlation with general response speed, the two criteria that Greenwald and colleagues identified as most important. In addition, the $D_{600}$-measure shows similar internal consistencies as the $C_1$-measure and C3-measure and the best performance in terms of effect sizes. In contrast to the study of Greenwald and colleagues, the $D_{600}$-measure seems somewhat more sensitive to prior IAT experience than the other measures, since the effect of Time on IAT-effects is four to eight times larger for the $D_{600}$-measure than for the $C_3$-measure and the $C_1$-measure respectively. However, the effect of Time is still rather small ($\eta^2 = .016$). Finally, the $D_{600}$-measure shows the best performance for predictive validity and test-retest reliability, the two criteria that were

added to the original criteria of Greenwald and colleagues (2003).

Since there seem to be considerable differences between data collected via the internet and in the laboratory (e.g., with respect to experimental control or commitment of participants), we hypothesized that scoring algorithms for the IAT might perform differently in both settings and/or in within-subjects designs. However, present findings disprove these hypotheses by replicating prior findings of Greenwald and colleagues (2003). In line with this study, the results suggest that the $D_{600}$-measure shows generally the best performance on the criteria that were used for evaluating the various algorithms. This was not only the case for criteria that were identified by Greenwald and colleagues (2003) as most important, i.e. correlation with explicit equivalent and correlation with general response speed, but also for the additional criteria that were included in the present study being predictive validity and test-retest reliability. All in all, the present findings lead to the conclusion that the $D_{600}$-measure is suitable for use in a laboratory setting in the domain of psychopathology, when using an IAT in which the order of category combination is fixed.

In addition, this study explored the performance of six alternative IAT algorithms. However, most of the alternative algorithms performed actually (much) worse than the $D_{600}$-measure. The outcomes point to the conclusion that the success of the $D_{600}$-measure stems from the combination of different ingredients being both the division by the pooled standard deviation and the inclusion of practice trials (and also the inclusion of error trials, but we did not specifically investigate this in the present design). By this specific combination of ingredients, up to now the $D_{600}$-measure is the IAT algorithm filtering out the most meaningful information, at least for this specific IAT design. This does not necessarily imply that all other algorithms should be discarded. Future studies will have to illuminate whether the present positive results for the D-measure also holds for laboratory studies with a different design. Therefore, it would be important for coming studies to report results of alternative algorithms next to the D-measure.

### *Limitations and Considerations*

The evaluation of scoring algorithms of the IAT is a complicated undertaking and several aspects could not be investigated in the present design. Unfortunately, the present design did not contain an outcome measure of spontaneous behaviours that could be used to assess the predictive validity of the various algorithms. It is often assumed that automatic associations are especially relevant for guiding more spontaneous kinds of behaviours (e.g., Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002; Huijding & de

Jong, 2006a). By using a self-report measure as outcome measure to test predictive validity, we run the risk of letting in the influence of explicit 'strategic processes' (e.g., Rothermund, Wentura & De Houwer, 2005; Wentura & Rothermund, 2007). Consequently, future research should also validate IAT-scoring algorithms against outcome measures of more spontaneous behaviours, preferably behaviours that are known to be driven primarily by automatic, but not by explicit processes.

Furthermore, because the NESDA sample was not specifically designed for the purpose of the present study, some factors have influenced the results. First of all, the blocks and the order of both IATs were not counterbalanced between participants. This was done to reduce method variance in consideration of the prospective design of the NESDA. Although other studies chose similar designs (e.g., Asendorpf et al., 2002; Schnabel et al., 2006; Steffens & König, 2006), it might limit the generalizability of the present findings to laboratory studies without fixed blocks / orders of IAT. In addition, the IAT design did not contain a built-in error penalty, which made it impossible to calculate the $D_1$-measure and the $D_2$-measure. Greenwald and colleagues (2003) showed that IAT algorithms with built-in error penalties -if anything- performed slightly better than the other D-measures. Given the similarity in performance of the $D_1$-measure and $D_2$-measure to the $D_{600}$-measure that was demonstrated by Greenwald and colleagues, we assume that the $D_1$-measure and $D_2$-measure can be used in laboratory settings as well.

As a more general issue, we note that the way RTs are used in the context of the IAT differs in an important way from their use in the tradition of Donders (1868), Sternberg (1969), and many others, where they are used to develop a model of the structure of information processing. In this tradition, RTs provide a measure of time duration as a physical property of a mental process, and are usually interpreted on a measurement scale at 'ratio / interval' level. In most of the IAT-literature, however, the aim is not to measure the duration of a process, but rather the strength of an implicit association and this is done indirectly, through its effect on time duration. Therefore, the scale of measurement of implicit associations is probably 'ordinal', as often is the case in psychometrics. When the IAT-effect indeed has to be interpreted on an ordinal measurement scale, this makes it acceptable that the algorithms of D-measures transform RTs in a complex way into an outcome index that is only weakly related to the original 'process duration'. From this perspective, we regard D-measures as algorithms that correct effectively for a number of notorious RT-confounders (errors, global speed), and therefore perform well in measuring individual differences in associations. However, we should be cautious with interpreting parametric statistics on the 'ordinal' D-measures.

### *Conclusion*

To summarize, the present study clearly and convincingly demonstrated that the $D_{600}$-measure is suitable to be used in a laboratory setting in the domain of psychopathology for IATs with a fixed order of category combinations. However, these findings should be further replicated, especially in studies that include outcome measures of more spontaneous kinds of behaviours. In future studies that make use of the IAT, it would be interesting not only to report results of the D-measure, but also the results of alternative IAT algorithms. Hopefully this will give us even more insight into the optimum use of the Implicit Association Test as a measure for automatic associations.

# Appendix 2.A

*IAT Stimulus words*

Me: I, myself, self, my, own

(ik, mezelf, zelf, mijn, eigen)

Others: other, you, they, them, themselves

(ander, jullie, zij, hun, zijzelf)

Anxious: anxious, afraid, nervous, insecure, worried

(angstig, bang, nerveus, onzeker, ongerust)

Calm: calm, balanced, placid, secure, relaxed

(kalm, evenwichtig, rustig, zeker, ontspannen)

Depressed: useless, pessimistic, inadequate, negative, meaningless

(nutteloos, pessimistisch, ongeschikt, negatief, zinloos)

Elated: positive, optimistic, active, valuable, cheerful

(positief, optimistisch, actief, waardevol, opgewekt)

*Note*. Words are translated from Dutch

# 3 Dysfunctional automatic self-associations in anxiety and depression

# Study 1: Group differences

## Abstract

Cognitive theory points to the importance of negative self-schemas in the onset and maintenance of depressive and anxiety disorders. Hereby, it is important to distinguish between automatic and explicit self-schemas, reflecting different cognitive-motivational systems. This study tested whether patients with a current major depressive disorder and/or anxiety disorder are characterized by automatic self-anxious and self-depressed associations and whether these associations are disorder-specific. Patients ($n$ = 2329) and non-clinical controls ($n$ = 652) were tested as part of the Netherlands Study of Depression and Anxiety, a multi-center, longitudinal, cohort study with patients from different health care settings. Patient groups and non-clinical controls (18–65 years of age) were compared with regard to automatic self-anxious and self-depressed associations measured with the Implicit Association Test. Individuals with an anxiety disorder showed enhanced self-anxious associations, whereas individuals with a depression showed enhanced self-depressed associations. Individuals with co-morbid disorders scored high on both automatic self-associations. Although remitted individuals showed weaker automatic self-associations than people with a current disorder, their automatic self-anxious/depressed associations were still significantly stronger than those of the control group. Importantly, automatic self-associations showed predictive validity for the severity of anxious and depressive symptoms over and above explicit self-beliefs. This study represents the first evidence that automatic self-anxious and self-depressed associations are differentially involved in anxiety and depressive disorders. This may help to explain the refractoriness of these disorders and points to the potential importance of automatic self-associations in the development of psychopathological symptoms.

Based on: Glashouwer, K. A., & de Jong, P. J. (2010). Disorder-specific automatic self-associations in depression and anxiety: Results of the Netherlands Study of Depression and Anxiety. *Psychological Medicine, 40,* 1101-1111.

## Introduction

During the past two decades, an increased interest for automatic associations has also spread to the field of psychopathology (e.g., de Houwer, 2002). This kind of research is inspired by recent information-processing models that emphasize the importance to distinguish between more explicit and more automatically activated cognitions. Both types of cognitions are believed to have different functional qualities (e.g., Gawronski & Bodenhausen, 2006). Explicit cognitions are assumed to reflect the outcome of the weighting of propositions and their corresponding 'truth' values (i.e. validation processes), whereas automatic associations are assumed to follow from direct activation of simple associations in memory, independent of their truth value. While explicit cognitions tend to predict more explicit, controlled behaviours, automatic associations seem to play an important role in guiding relatively spontaneous, uncontrollable behaviours (e.g., Spalding & Hardin, 1999; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a), the kind of behaviours that are also critically involved in psychopathology where patients often report symptoms being unpredictable and uncontrollable (e.g. Mayer, Merckelbach & Muris, 2000).

Dysfunctional automatic associations are not, by definition, present in psychological disorders and sometimes automatic associations diverge from their explicit equivalents (e.g., de Jong, van den Hout, Rietbroek & Huijding, 2003; De Raedt, Schacht, Franck & De Houwer, 2006; Brauer, de Jong, Huijding, Laan & ter Kuile, 2009). Moreover, in the cases where dysfunctional automatic associations do co-occur with dysfunctional explicit cognitions, this does not imply that automatic associations are redundant, since they tend to predict different kinds of behaviour (see e.g., Asendorpf et al. 2002; Huijding & de Jong, 2006a). In addition, treatment-induced changes in explicit associations do not necessarily imply similar changes in automatic associations or vice versa (e.g., Huijding & de Jong, 2007, 2009). Starting from this, it seems possible that persistent automatic associations at least partially account for the persistence and/or return of psychopathological symptoms.

Cognitive theory points to the importance of negative schemas with regard to 'the self' in the onset and maintenance of psychopathology (e.g. Clark, Beck & Alford, 1999). In line with this, several studies provided evidence for a relationship between automatic self-associations and various types of psychopathological symptoms, such as symptoms of obsessive compulsive personality disorder (Weertman, Arntz, de Jong & Rinck, 2008) and chronic pain (Grumm, Erbe, von Collani & Nestler, 2008). There is also considerable support for the notion that automatic self-anxious associations are involved in anxiety (Egloff & Schmukle, 2002; Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008). Yet, thus far, studies testing the role of automatic self-anxious associations typically compared a 'pathologi-

cal' group with a healthy control group. It remains, therefore, to be tested whether these self-associations reflect disorder/anxiety-specific automatic associations or should be considered as more general characteristics that are shared by other psychological disorders as well. Following this, we included depressed individuals as a clinical control group and tested whether enhanced automatic self-anxious associations are typically involved in anxiety disorders but not in depression.

Although there is already supportive evidence that automatic self-anxious associations are involved in anxiety and anxious behaviour (Egloff & Schmukle, 2002), little is known about automatic self-depressed associations and the link with depression. Yet, analogous to self-anxious associations in anxiety disorders, self-depressed associations may be especially relevant for guiding relatively spontaneous depressed behaviours, thoughts and feelings and may thus help to explain the uncontrollability and persistence of these symptoms. Therefore, the second goal of this study was to examine whether patients with a current major depressive disorder are specifically characterized by automatic self-depressed associations.

The third goal was to investigate whether dysfunctional automatic self-associations represent relatively stable characteristics that remain unchanged even after recovery of anxiety and/or depressive disorders. Therefore, we examined whether individuals remitted from an anxiety disorder, a major depressive disorder or both were still characterized by enhanced automatic self-depressed/anxious associations. If dysfunctional automatic self-associations are a stable cognitive feature of these disorders, we would expect them to be still present in remitted individuals.

Two Implicit Association Tests (IATs ; Greenwald, McGhee & Schwartz, 1998) were used to obtain measurements of automatic self-anxious and self-depressed associations. Data were collected among patients and non-clinical controls as part of the Netherlands Study of Depression and Anxiety (NESDA; see www.nesda.nl). This is the first study that explores the specificity of automatic self-associations in such a large-scale, clinical sample. We hypothesized that (remitted) depressed patients would be characterized by enhanced automatic self-depressed associations and (remitted) anxious patients by enhanced automatic self-anxious associations.

## Method

This study was carried out in the context of the NESDA (Penninx et al., 2008), a multi-center, longitudinal cohort study designed to examine the long-term course and consequences of anxiety and depressive disorders. This study concerns the baseline measurement

conducted from September 2004 to February 2007. The study protocol was approved centrally by the Ethical Review Board of VU Medical Center Amsterdam and subsequently by local review boards of each participating center/institute.

## *Participants*

Recruitment of respondents took place in the general population, in general practices and in mental health care institutions and included a range of psychopathology: controls without symptoms or disorders; individuals at risk because of prior episodes; sub threshold symptoms or family history; individuals with a current first or recurrent depressive or anxiety disorder. Across the recruitment setting, uniform inclusion and exclusion criteria were used. A general inclusion criterion was an age of 18–65 years. Only two exclusion criteria existed: (1) primary clinical diagnosis of a psychiatric disorder not subject of NESDA, which would largely affect course trajectory: psychotic disorder, obsessive compulsive disorder, bipolar disorder or severe addiction disorder; (2) not being fluent in Dutch. In total, 2981 participants [66.5% female; mean age 41.9 years, standard deviation (SD) 13.0] were included, of which 652 were non-clinical controls without present or past diagnosis. We chose to focus on individuals with a current (during the past month) or past diagnosis of major depressive disorder and/or a current or past anxiety disorder (general anxiety disorder, panic disorder, social phobia and agoraphobia). The different groups and their characteristics can be found in Table 3.1 (see also Penninx et al., 2008).

## *Measures*

**Implicit Association Test.** The IAT is a computerized reaction time task originally designed by Greenwald et al. (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left or right response key. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated (e.g. an anxious person should find it easier to categorize words of *me* and *anxious* with the same button than *me* and *calm*). The category labels are visible in the upper left and right-hand corners of the screen during the whole task (for an example, see https://implicit.harvard.edu/implicit). Following the design of Egloff & Schmukle (2002), two IATs were constructed to measure automatic self-anxious associations and automatic self-depressed associations. For both IATs, target labels were *me* and *other*. Attribute labels were *anxious*and *calm* for

the anxiety IAT and *depressed* and *elated* for the depression IAT. Each category consisted of five stimuli (see Chapter 2, Appendix 2.A). Both IATs consisted of two critical test blocks that were preceded by practice blocks (see Chapter 2, Table 2.1). The order of category combinations was fixed across participants to reduce method variance. This is assumed to enhance the sensitivity of the IAT as a measure of individual differences, which is important in view of the prospective design of the NESDA (cf. Asendorpf et al. 2002; Schnabel, Banse & Asendorpf, 2006; Steffens & König, 2006).

To obtain explicit equivalents of the automatic associations, participants rated all IAT attribute stimuli on a 5-point scale (1 = hardly/not at all, 5 = very much) (i.e. "For each word please indicate to what extent you think it generally applies to you").

Table 3.1

*Means and standard deviations of self-report and automatic measures as a function of group*

## Major Depressive Disorder

| | **current MDD** | **current MDD without history AD** | **remitted MDD** |
|---|---|---|---|
| | ***n* = 283** | ***n* = 176** | ***n* = 330** |
| Age | 41.47 (12.50) | 40.77 ± 12..99 | 42.38 ± 13.12 |
| Gender, % women | 62.54 (.49) | 61.93 (.49) | 67.27 (.47) |
| Education in years | 11.79 (3.14) | 11.60 (.3.23) | 12.78 (3.14) |
| Recruitment setting [a] | 39 : 53 : 8 | 44 : 51 : 5 | 53 : 13 : 34 |
| D-measure anxiety IAT [b] | .24 (.50) | .30 (.52) | .44 (.44) |
| Error rate anxiety IAT | 6.24 (6.57) | 6.79 (7.75) | 5.36 (5.69) |
| D-measure depression IAT [b] | .10 (.36) | .11 (.37) | .32 (.33) |
| Error rate depression IAT | 5.98 (.6.14) | 6.68 (7.22) | 4.74 (4.97) |
| Anxiety EA [b] | .10 ( 1.38) | .24 (1.45) | 1.49 (1.16) |
| Depression EA [b] | .28 (1.59) | .36 (1.65) | 2.08 (1.15) |
| IDS-SR | 31.72 (10.73) | 31.78 (10.64) | 14.46 (9.23) |
| BAI | 15.00 (9.78) | 14.20 (9.56) | 6.55 (5.97) |

*Note*. PC = Primary Care; SMHC = Specialized Mental Health Care; GP = General Population; IAT = Implicit Association Test; EA = Explicit Associations; MDD = Major Depressive Disorder; AD = Anxiety Disorder; IDS-SR = Inventory of Depressive Symptoms self-report; BAI = Beck Anxiety Inventory. [a] % PC : % SMHC : % GP; [b] Positive effects indicate a relatively strong automatic/explicit association between *me* and *calm*/*elated*; [c] Social Phobia *n* = 128; Panic Disorder *n* = 129; Agoraphobia *n* = 63; Generalized Anxiety Disorder *n* = 50; more than one anxiety disorder *n* = 142.

Table 3.1

*continued*

## Anxiety Disorder

| | current AD[c] | current AD without history MDD | remitted AD |
|---|---|---|---|
| | ***n* = 512** | ***n* = 195** | ***n* = 138** |
| Age | 41.97 (12.65) | 41.84 (13.09) | 41.02 (15.25) |
| Gender, % women | 67.78 (.47) | 64.62 (.48) | 69.57 (.46) |
| Education in years | 12.14 (3.21) | 12.06 (3.39) | 12.51 (3.14) |
| Recruitment setting [a] | 54 : 38 : 8 | 56 : 39 : 5 | 63 : 12 : 25 |
| D-measure anxiety IAT [b] | .12 (.52) | .11 (.56) | .38 (.47) |
| Error rate anxiety IAT | 5.88 (5.83) | 5.99 (5.70) | 5.21 (5.05) |
| D-measure depression IAT [b] | .20 (.38) | .19 (.38) | .31 (.34) |
| Error rate depression IAT | 5.21 (.5.07) | 4.64 (4.18) | 5.10 (4.31) |
| Anxiety EA [b] | -.21 (1.37) | -.07 (1.38) | 1.31 (1.14) |
| Depression EA [b] | 1.17 (1.39) | 1.49 (1.32) | 2.23 (1.07) |
| IDS-SR | 23.36 (10.27) | 21.48 (9.93) | 13.73 (8.54) |
| BAI | 15.98 (9.99) | 15.50 (10.15) | 7.56 (5.95) |

## Controls

| non-clinical |
|---|
| ***n* = 648** |
| 41.07 (14.69) |
| 61.42 (.49) |
| 12.80 (3.19) |
| 78 : 0 : 22 |
| .49 (.45) |
| 5.55(.5.19) |
| .40 (.34) |
| 5.28 (5.02) |
| 2.18 (1.03) |
| 2.70 (.84) |
| 8.46 (7.49) |
| 4.03 (4.86) |

## Comorbid

| | current MDD/AD | remitted MDD/AD | current MDD/ history AD | current AD history MDD |
|---|---|---|---|---|
| | ***n* = 487** | ***n* = 326** | ***n* = 107** | ***n* = 317** |
| Age | 41.68 (11.77) | 41.62 (12.39) | 42.62 (12.63) | 42.06 (12.39) |
| Gender, % women | 67.56 (.47) | 75.15 (.43) | 63.55 (.48) | 69.72 (.46) |
| Education in years | 11.09 (3.15) | 12.40 (3.22) | 12.09 (2.97) | 12.19 (.3.10) |
| Recruitment setting [a] | 37 : 58 : 5 | 57 : 16 : 27 | 32 : 56 : 12 | 53 : 37 : 10 |
| D-measure anxiety IAT [b] | .04 (.51) | .31 (.45) | .15 (.44) | .12 (.50) |
| Error rate anxiety IAT | 6.37 (5.91) | 5.19 (4.87) | 5.32 (3.74) | 5.81 (5.92) |
| D-measure depression IAT [b] | .04 (.41) | .21 (.37) | .08 (.34) | .20 (.38) |
| Error rate depression IAT | 6.10 (5.91) | 5.17 (4.47) | 4.83 (3.46) | 5.56 (5.52) |
| Anxiety EA [b] | -1.12 (1.30) | .84 (1.26) | -.12 (1.22) | -.29 (1.36) |
| Depression EA [b] | -.47 (1.50) | 1.64 (1.23) | .15 (1.49) | .97 (1.39) |
| IDS-SR | 37.17 (12.38) | 18.05 (9.73) | 31.62 (10.93) | 24.51 (10.32) |
| BAI | 22.29 (11.52) | 9.39 (7.46) | 16.32 (10.04) | 16.28 (9.89) |

**Diagnostic assessment and other measures.** Depressive and anxiety disorders were determined by means of the lifetime Composite International Diagnostic Interview (CIDI) (WHO version 2.1; Robins et al., 1989), which classifies diagnoses according to DSM-IV criteria (American Psychiatric Association, 2000). Participants with a current disorder suffered from this disorder during the past month. People were considered remitted when they currently no longer met the criteria, but had a depressive or anxiety disorder in the past. 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, 2006) and high validity for depressive and anxiety disorders (Wittchen, Burke, Semler & Pfister, 1989; Wittchen, 1994). In order to conduct the study, over 40 research assistants (psychologists, nurses and residents in psychiatry) were trained during 1 week by the fieldwork coordinator. Interviewer performance was monitored by checking a random selection of about 10% of all taped interviews. In addition, a continuous monitoring system of interviewer variances and interviewer-specific item non-response was maintained through computer analyses in SPSS (SPSS Inc., Chicago, IL, USA).

Severity of depressive symptoms was measured with the 30-item Inventory of Depressive Symptoms Self-Report version (IDS-SR; Rush, Gullion, Basco & Jarrett, 1996). Severity of anxiety symptoms was measured using the 21-item Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988). We used total scale scores of these questionnaires as indices for severity of depressive and anxiety symptoms.

## *Procedure*

The assessment lasted between 3 and 5 h and was conducted on 1 day. During the assessment, first the CIDI, then the IATs and explicit ratings were obtained. In between and afterwards, other measurements were collected, but these are not of interest for the present study (for a detailed description, see Penninx et al., 2008). Each participant completed the anxiety IAT, followed by the depression IAT. After that, they explicitly rated the attribute words that were used in the IATs. Respondents were compensated with a €15,- gift certificate and travel expenses.

## *Data analyses*

**Data reduction.** IAT scores were computed according to the now widely used algorithm proposed by Greenwald, Nosek and Banaji (2003). We report the $D_4$-measure. Reaction times above 10,000 ms were discarded and error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred plus a pen-

alty of 600 ms. For the anxiety IAT, the IAT effect was calculated by subtracting mean reaction times of block 6 from block 3 (practice) and block 7 from block 4 (test). The means of these two effects were divided by their pooled standard deviation based on all responses in blocks 3, 4, 6 and 7. Analogously, the IAT effect was calculated for the depression IAT, based on blocks 9, 10, 12 and 13. Positive IAT effects indicate relatively fast responses when *me* shared the response key with either *calm* or *elated*.[1] For descriptive purposes, mean scores (ms per block per group) are summarized in Appendix 3.A. Split-half reliabilities of the present IATs were good, with Spearman-Brown corrected correlations between test halves of .82 for the depression IAT and .87 for the anxiety IAT (test halves were based on trials 1, 2, 5, 6, 9, 10, etc. v. 3, 4, 7, 8, 11, 12, etc.).

To compute explicit association effects, mean ratings of anxious (depressed) stimuli were subtracted from mean ratings of calm (elated) stimuli. Hence, positive effects indicate strong explicit associations between *me* and *calm* (or *me* and *elated*). The internal consistency of the explicit self-association measures was good, with Cronbach's $\alpha$ .94 for the difference scores of anxious and calm words and .95 of depressed and elated words.

**Statistical analyses.** The anxiety and depression IATs could not be compared directly, because different attribute concepts were used and because the order of IATs was fixed. Therefore, univariate analyses of variance (ANOVA) were run on automatic self-anxious and self-depressed associations with group as between-subject factor. The univariate tests were conducted with $\alpha < .05$. The Bonferroni procedure was used to control for the inflation of type 1 errors arising from testing multiple planned comparisons. For comparison reasons, all tests were repeated with explicit self-anxious and self-depressed associations as dependent variables. Finally, stepwise regression analyses were used to explore whether automatic associations had predictive validity for symptom severity as measured by the BAI and IDS-SR over and above explicit self-beliefs.

## Results

### *Descriptives*

**Missing values.** Due to technical problems, IAT data for 129 participants was missing. Furthermore, ten participants were discarded from all analyses because more than 10% of the IAT trials were below 300 ms (Greenwald et al., 2003), suggesting that they were trying to respond too rapidly. Five participants were discarded because of unusual D-scores (> 5

[1] Please note that this scoring procedure is reversed compared to the published article in Psychological Medicine (Glashouwer & de Jong, 2010). Originally we decided to make the D-measure comparable to symptom measures in which a higher score also indicates a less favourable outcome. However, thanks to an anonymous reviewers' comment on a different study, we decided to reverse the scoring to make them comparable to the general literature and the other studies in this thesis. The only thing that differs between this study and the published paper is the multiplication sign before the D-measure / explicit self-beliefs.

SD divergent from mean), that were explained by a very slow overall response tendency (> 4000 ms) and/or high error rates (> 28.8%). The mean D-scores and standard deviations of the IATs and the explicit associations are reported in Table 3.1.

**Construction of groups.** As can be seen in Table 3.1, groups were constructed based on different combinations of current and past diagnoses of depressive and anxiety disorders. We first constructed groups of participants with a current Major Depressive Disorder (MDD), a current Anxiety Disorder (AD) or both (irrespective of their history) and included a non-clinical control group.

Secondly, we examined whether people who were remitted from MDD, AD or both were still characterized by higher levels of automatic self-anxious/depressive associations. We constructed groups with remitted patients (MDD, AD or both) and compared them to patients with a current diagnosis and controls. To keep the comparisons straightforward, we examined MDD and AD groups separately. Additionally, we compared current and past comorbid groups with controls, and also included participants with a current MDD with a history of AD and similarly, participants with a current AD who had a history of MDD.

**Correlations.** The correlations between automatic and explicit self-associations are shown in Table 3.2. Further exploration for separate groups revealed similar patterns of automatic-explicit correlations.

## *Are anxious patients characterized by automatic self-anxious associations?*

A 4 Group (current MDD, current AD, current AD/MDD, control) ANOVA on automatic self-anxious associations showed a significant main effect for Group ($F(3,1926) = 92.04$, $p < .001$, partial $\eta^2 = .13$). As expected, the anxious group showed significantly stronger automatic self-anxious associations (i.e., a relatively faster response when *me* and *anxious*

Table 3.2

*Correlation matrix of automatic and explicit self-anxious and self-depressed associations over all participants (N = 2837)*

| Measure | 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| 1. IAT anxiety | - | .49* | .37* | .31* |
| 2. IAT depression | | - | .34* | .38* |
| 3. EA anxiety | | | - | .78* |
| 4. EA depression | | | | - |

IAT = Implicit Association Test; EA = Explicit Associations.
*Correlation is significant at the .01 level (2-tailed).

shared the response key) than both the depressed and the control group (anxious vs. depressed: $t(793) = 3.32$, $p = .001$, $d = .24$; anxious vs. controls: $t(1005.69) = 12.79$, $p < .001$, $d = .76$). The comorbid group inclined towards stronger automatic self-anxious associations than the anxious group ($t(997) = 2.23$, $p = .03$, $d = .16$).

### *Are depressed patients characterized by automatic self-depressed associations?*

A similar 4 Group ANOVA on self-depressed associations showed a significant main effect for Group ($F(3,1926) = 98.54$, $p < .001$, partial $\eta^2 = .13$). As expected, the depressed group showed significantly stronger automatic self-depressed associations than the control group ($t(929) = 12.23$, $p < .001$, $d = .86$) and the anxious group ($t(793) = 3.42$, $p = .001$, $d = .27$). The comorbid group again inclined towards stronger automatic self-depressed associations than the depressed group ($t(656.87) = 2.08$, $p = .04$, $d = .16$). However, future research has to show, whether this trend represents a robust phenomenon.

The anxiety disorders (General Anxiety Disorder, Panic Disorder, Social Phobia and Agoraphobia) were compared on both IATs. There was no significant main effect of Group ($p$'s > .1) indicating that automatic self-anxious and automatic self-depressed associations were very similar for the various anxiety disorders.

### *Are remitted patients still characterized by automatic self-anxious/depressed associations?*

**MDD.** A 3 Group (current MDD without history AD, remitted MDD, control) ANOVA on IAT depression showed a significant main effect for Group ($F(2,1151) = 49.64$, $p < .001$, partial $\eta^2 = .08$). Remitted participants showed significantly weaker self-depressed associations than participants with a current depression ($t(504) = 6.41$, $p < .001$, $d = .52$). However, remitted patients still scored significantly stronger self-depressed associations than controls ($t(976) = 3.55$, $p < .001$, $d = .24$).

**AD.** A 3 Group (current AD without history MDD, remitted AD, control) ANOVA on IAT anxiety showed a significant main effect for Group ($F(2,978) = 47.98$, $p < .001$, partial $\eta^2 = .09$). Remitted participants showed significantly weaker automatic self-anxious associations than participants with a current anxiety disorder ($t(322.05) = 4.80$, $p < .001$, $d = .52$). However, remitted patients still showed significantly stronger self-anxious associations than the controls ($t(784) = 2.56$, $p = .01$, $d = .24$).

**Comorbid.** A 5 Group (current AD/MDD, current AD/remitted MDD, current MDD/remitted AD, remitted AD/MDD, control) ANOVA on IAT depression showed a significant

main effect for Group ($F(4,1880) = 70.43$, $p < .001$, partial $\eta^2 = .13$). Remitted MDD/AD participants showed significantly weaker automatic self-depressed associations than participants with current MDD/AD and participants with current MDD that were remitted from AD (remitted AD/MDD vs. current AD/MDD: $t(811) = 5.76$, $p < .001$, $d = .56$; remitted AD/MDD vs. current MDD/remitted AD: $t(431) = 3.04$, $p < .01$, $d = .37$). However, remitted patients still showed significantly self-depressed associations than controls ($t(972) = 8.22$, $p < .001$, $d = .53$). No significant difference was found in automatic self-depressed associations between participants remitted from AD/MDD and participants with current AD that were remitted from MDD ($p = .80$).

A 5 Group (remitted AD/MDD, current AD/MDD, current AD/remitted MDD, current MDD/remitted AD, control) ANOVA on IAT anxiety showed a significant main effect for Group ($F(4,1880) = 72.30$, $p < .001$, partial $\eta^2 = .13$). Remitted participants showed significantly weaker automatic self-anxious associations compared to all groups of participants with current disorders (remitted AD/MDD vs. current AD/MDD: $t(811) = 7.77$, $p < .001$, $d = .56$; remitted AD/MDD vs. current AD/remitted MDD: $t(641) = 5.12$, $p < .001$, $d = .40$; remitted AD/MDD vs. current MDD/remitted AD: $t(431) = 3.17$, $p < .01$, $d = .36$). However, remitted patients still showed significantly stronger self-anxious associations than the controls ($t(972) = 5.72$, $p < .001$, $d = .40$).

### *Do the groups differ similarly on explicit equivalents?*

Similar univariate ANOVAs were run, but this time with explicit self-anxious and self-depressed associations as dependent variables instead of automatic self-associations. In line with the trend for the automatic measurements, the comorbid group showed significantly stornger explicit self-anxious associations than the anxious group and stronger explicit self-depressed associations than the depressed group (*p's* < .001). However we observed a difference for participants remitted from AD/MDD, who had significantly weaker explicit self-depressed associations than the participants with current AD who were remitted from MDD ($t(641) = 6.39$, $p < .001$, $d = 1.09$). Apart from this difference, the analyses revealed a similar pattern of results as with the automatic measures.[2]

### *Are automatic associations predictive for symptom severity?*

First, explicit self-depression associations were entered in a regression model to predict the score on IDS-SR. This was shown to be significant ($F(1,2828) = 3586.20$, $p < .001$, $R^2_{change} = .56$). Then, IAT depression was added to the model and was shown to be predictive for

[2] The full outcome of these analyses can be received on request by the first author.

IDS-SR over and above explicit self-depression associations ($F(1,2827) = 18.12$, $p < .001$, $R^2_{change} = .003$). Second, explicit self-anxious associations were entered in a regression model to predict the score on BAI, which was also shown to be significant ($F(1,2835) = 2343.17$, $p < .001$, $R^2_{change} = .45$). Next, IAT anxiety was predictive for BAI over and above explicit self-anxious associations ($F(1,2834) = 23.51$, $p < .001$, $R^2_{change} = .005$).

## Discussion

This study represents the first research into disorder-specificity of automatic self-associations in anxiety disorders and depression. The main findings can be summarized as follows: (i) Patients with an anxiety disorder showed stronger automatic self-anxious associations than did depressed patients and controls; (ii) similarly, patients with a major depressive disorder showed stronger automatic self-depressed associations than did anxious patients and controls; and (iii) participants with comorbid anxiety and major depressive disorder displayed both strong automatic self-anxious and self-depressed associations. Furthermore, (iv) although people remitted from a disorder showed weaker automatic self-associations than people with a current disorder, their automatic self-anxious/depressed associations were still significantly stronger than those of the control group. Finally, (v) although the effects were small, automatic associations significantly predicted the severity of anxious and depressive symptoms over and above explicit self-beliefs.

In line with current views stressing the potential importance of dysfunctional automatic associations in the aetiology and maintenance of affective disorders (e.g., Beevers, 2005; Haeffel et al., 2007), the present study clearly shows that patients and healthy controls do differ with respect to automatic self-associations. Consistent with the hypothesis that negative self-schemas are important in the onset and maintenance of psychopathology (e.g., Clark et al., 1999) automatic self-anxious and automatic self-depressed associations differentiated between depressed and anxious patients, whereas the comorbid group displayed both types of dysfunctional automatic self-associations. These results further strengthen earlier findings that the IAT can measure more specific associations than simple positive-negative evaluations (cf. Teachman, Gregg & Woody, 2001; Rüsch et al., 2007). However, at the same time, the results suggest that next to disorder-specific factors, common-factors underlie both anxiety and depressive disorders, which is underlined by the high correlations that were found between anxiety and depressive measures. Although the differences between the groups on explicit equivalents were generally in the same direction, the correlations between the automatic and explicit measurements were only moderate. This is consistent with other studies (e.g., Hofmann, Gawronski, Gschwendner,

Le & Schmitt, 2005) and in accordance with the starting point that different memory processes form the basis of explicit and automatic cognitions (Gawronski & Bodenhausen, 2006).

A second goal was to investigate whether dysfunctional automatic self-associations represent a relatively stable characteristic that remains unchanged after recovery of anxiety and/or depressive disorders. Results showed that remitted individuals automatically associated themselves stronger with anxious and/or depressed words than healthy controls. Although the differences were only small to moderate, this pattern of results is consistent with the view that negative self-associations may form a stable cognitive feature for affective disorders. It remains however unclear whether it indeed concerns pre-morbid vulnerability, a 'scar'[3] that remained as a result of an prior episode which may set people at risk for recurrence of symptoms, or both. To arrive at more solid conclusions in this respect, an important next step would be to complement these cross-sectional data with a longitudinal approach to examine the alleged role of dysfunctional automatic associations in the onset and maintenance of anxiety and depressive symptoms.

At the same time, remitted individuals showed weaker automatic self-anxious/depressed associations than individuals with a current disorder. This could indicate that automatic self-associations also relate to the severity of current symptoms[4], which is supported by the fact that remitted individuals show less anxious and depressive symptoms than individuals with a current disorder, but somewhat more symptoms than the control group. However, the present correlational data are silent with regard to the direction of this relationship. Therefore, it remains to be tested whether automatic associations lead to symptoms, or vice versa, or whether automatic associations are merely epiphenomena of a disorder. Furthermore, it would be important to test the predictive validity of automatic associations for the recurrence of anxiety and depressive episodes. It is possible that treatment might differentially influence automatic and explicit cognitions. As a result, unaffected, residual, dysfunctional automatic self-associations may play an important role in the recurrence of spontaneous, uncontrolled depressive and anxiety symptoms (cf. Huijding & de Jong, 2007).

Interestingly, in comparison with patients with a single current anxiety or major de-

[3] To further test the scar hypothesis, we examined (in the remitted group) whether automatic self-depressed associations were related to the number of prior depressive episodes. The results provided no support for the scar hypothesis as the automatic self-depressed associations were not especially pronounced in individuals with relatively many prior depressive episodes.

[4] In addition, we examined whether automatic associations were related to symptom severity in the remitted groups. There were indeed some small, but significant correlations (*r's* varying from .10 to .14). Furthermore, we found that the more time had passed after having suffered from a depressive or anxiety disorder; the more positive were the automatic self-associations (depression: $r = -.14$, $p < .05$; anxiety: $r = -.19$, $p < .05$).

pressive disorder, the current comorbid patients inclined towards stronger automatic self-anxious and self-depressed associations. For explicit self-associations a similar pattern was evident. This pattern of findings is in accordance with current and previous observations that comorbid patients also report relatively severe symptoms (see also Bruce et al., 2005; Hecht, von Zerssen & Wittchen, 1990; Roy-Byrne et al., 2000), and provides further evidence for the link between the strength of automatic self-associations and the severity of symptoms. However, it could also constitute a general vulnerability to develop both depressive and anxiety disorders.

### *Limitations*

Some comments are in order with respect to the limitations of the present study. First, the order of the anxiety IAT and the depression IAT, and the order of the category combinations within both IATs, were fixed. Although this has clear advantages with respect to the enhancement of the sensitivity of the IAT as a measurement of individual differences (cf., Asendorpf et al., 2002; Schnabel et al., 2006; Steffens & König, 2006), this procedure also has some important drawbacks. IAT effects tend to decrease with the number of IATs presented to a participant (Greenwald et al., 2003). Consequently, the present fixed order hampers direct comparison of both IATs. Furthermore, it makes it hard to interpret the absolute value of the IAT outcomes, which means that the negative IAT indices we found not simply imply stronger self-calm/elated associations. The negative indices could also be caused by order effects resulting in the zero point not being an actual 'zero point'. However, it seems that this ambiguity is not critical in the present context, because our focus was primarily on the relative differences in automatic associations between groups. Secondly, it is important to note that the IAT is only one of several instruments that are often used to index automatic associations (for a critical overview, see e.g., De Houwer, 2006) and, although the psychometric properties of the IAT have been well tested during the past decade, the IAT is not without its critics (e.g., Fiedler, Messner & Bluemke, 2006). Finally, the correlations between automatic measures and between explicit self-association measures were higher than between automatic and explicit measures of self-anxious associations and between automatic and explicit measures of self-depressed associations. This may indicate that the method variance is rather high. However, the present pattern of results may also be due to a greater 'conceptual overlap' between automatic measures and between explicit measures.

## *Conclusions and future directions*

The present study produced the first evidence that automatic self-anxious and automatic self-depressed associations are differentially involved in anxiety and/or depressive disorders. These findings point to the potential importance of automatic self-associations in the understanding of underlying cognitive mechanisms of affective disorders. In addition, the present study provides tentative evidence consistent with the notion that enhanced self-anxious and self-depressed associations can be considered as relatively stable features of affective disorders. An important next step would be to complement these cross-sectional data with a longitudinal and/or experimental approach to elucidate further whether automatic self-associations might indeed have differential predictive value for the onset, maintenance and recurrence of anxiety and/or depression. This could generate fresh starting points in order to improve and develop tailored interventions that might contribute to more effective treatment of depression and anxiety.

# Appendix 3.A

*Mean scores per block of IAT reaction times*

| IAT Blocks | MDD | AD | Comorbid | Controls |
|---|---|---|---|---|
| Blocks 3 & 4 (me/anxious) | 1258 (496) | 1199 (421) | 1227 (370) | 1202 (417) |
| Blocks 6 & 7 (me/calm) | 1135 (453) | 1152 (422) | 1222 (447) | 969 (274) |
| Blocks 9 & 10 (me/depressed) | 1103 (383) | 1098 (349) | 1110 (317) | 1071 (307) |
| Blocks 12 & 13 (me/elated) | 1048 (348) | 1010 (318) | 1106 (388) | 894 (228) |

*Note*. The mean reaction times in ms were calculated for the correct responses. The unweighted mean between practice- and test trials is reported. IAT = Implicit Association Test; MDD = Major Depressive Disorder; AD = Anxiety Disorder.

# Study 2: Suicidal ideation

## Abstract

Dysfunctional self-schemas are assumed to play an important role in suicidal ideation. According to recent information-processing models, it is important to differentiate between 'explicit' beliefs and automatic associations. Explicit beliefs stem from the weighting of propositions and their corresponding 'truth' values, while automatic associations reflect more simple associations in memory. Both types of associations are assumed to have different functional properties and both may be involved in suicidal ideation. Thus far, studies into self-schemas and suicidal ideation focused on the more explicit, consciously accessible traces of self-schemas and predominantly relied on self-report questionnaires or interviews. To complement these 'explicit' findings and more directly tap into self-schemas, this study investigated automatic self-associations in a large scale community sample that was part of the Netherlands Study of Depression and Anxiety (NESDA). The results showed that automatic self-associations of depression and anxiety were indeed significantly related to suicidal ideation and past suicide attempt. Moreover, the interactions between automatic self-depressed (anxious) associations and explicit self-depressed (anxious) beliefs explained additional variance over and above explicit self-beliefs. Together these results provide an initial insight into one explanation of why suicidal patients might report difficulties in preventing and managing suicidal thoughts.

Based on: Glashouwer, K. A., de Jong, P. J., Penninx, B. W. J. H., Kerkhof, A. J. F. M., van Dyck, R., & Ormel, J. (2010). Do automatic self-associations relate to suicidal ideation? *Journal of Psychopathology and Behavioral Assessment, 32,* 428-437.

## Introduction

Every year, nearly 1 million people over the world commit suicide (World Health Organization, 2003). With this, suicide is at the top of the list of the ten leading causes of mortality and disease burden in adults between 15 and 59 years of age. One important risk factor associated with suicide is suicidal ideation (e.g., Kessler, Borges & Walters, 1999). Considering the potentially severe outcomes of suicidal ideation, it seems paramount to enhance insight into the underlying processes that may influence suicidal thoughts.

According to cognitive theories, psychopathological symptoms (including suicidal ideation) are the result of dysfunctional schemas that exist in memory (e.g., Beck & Steward, 1989, cited in Weishaar & Beck, 1992; Clark, Beck, & Alford, 1999; Ellis, 2006). Schemas are assumed to be more or less enduring cognitive structures that organize people's thoughts, feelings, and behaviours into stable patterns. It has been argued that individuals can have several different schemas, and that it is possible to switch between them (e.g., Young, 1990). Dysfunctional self-schemas could lead to negative beliefs with respect to 'the self' which in turn may lead to suicidal thoughts. In line with this, several studies already showed that negative self-beliefs were related to suicide risk (e.g., Becker & Grilo, 2007; Cox, Enns & Clara, 2004; Kienhorst, de Wilde, van den Bout & Diekstra 1990; Lewinsohn, Rohde & Seeley, 1994), and suicidal ideation (e.g., Beck & Steward, 1989, cited in Weishaar & Beck, 1992; De Man & Gutiérrez, 2002; Evans, Hawton & Rodham, 2004).

Thus far, studies into self-schemas and suicidal ideation focused on the more explicit, consciously accessible traces of self-schemas and predominantly relied on self-report questionnaires or interviews. However, recent information-processing models propose that it is important to differentiate between this explicit belief level and more automatic memory associations (e.g., Beevers, 2005; Fazio & Towles-Schwen, 1999; Wilson, Lindsey & Schooler, 2000). Explicit beliefs are thought to reflect the outcome of the weighting of propositions and their corresponding 'truth' values (i.e., validation processes; e.g., Gawronski & Bodenhausen, 2006). In contrast, automatic self-associations are assumed to more directly mirror the activation of simple links in memory between 'self' and particular concepts (Gawronski & Bodenhausen, 2006) and likewise may reflect a deeper level of cognitive structure more closely linked to the schemas themselves. The measurement of automatic self-associations is based on behaviour (e.g., performance in reaction-time tasks), and therefore is believed to be less distorted by lack of introspection, social desirability or other demand characteristics (De Houwer, 2002; Greenwald & Farnham, 2000). Thus, while self-report measures are thought to capture explicit, conscious self-beliefs that stem from certain self-schemas, performance-based measures are assumed to more

directly tap into the self-schemas.

Recent studies have already demonstrated a relationship between disorder-specific automatic self-associations and various types of psychopathological symptoms, such as symptoms of obsessive compulsive personality disorder (Weertman, Arntz, de Jong & Rinck, 2008), chronic pain (Grumm, Erbe, von Collani & Nestler, 2008), anxiety (Egloff & Schmukle, 2002; Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008; Glashouwer & de Jong, 2010) and depression (Glashouwer & de Jong, 2010). Most important for the present context, these automatic self-associations showed differential predictive validity for more spontaneous, uncontrollable kind of behaviours such as autonomic responding and nonverbal behaviours (e.g., Asendorpf, Banse & Mücke, 2002; Spalding & Hardin, 1999; Egloff & Schmukle, 2002). On the basis of these findings it has been argued that automatic associations may well play an important role in guiding relatively spontaneous, uncontrollable behaviours that are critically involved in psychopathology (e.g., Huijding & de Jong, 2006a), and may thus at least partially account for the persistence of psychopathological symptoms.

Building on this, we hypothesized that dysfunctional automatic self-associations might also contribute to the onset and maintenance of suicidal ideation, since suicidal patients often report difficulties in controlling suicidal thoughts and preventing them from repetitively entering their awareness. Germane to this, it has already been found that automatic self-injury associations were predictive of suicidal ideation during the past year, past suicide attempt, as well as future suicidal ideation, over and above known risk-factors (e.g., mood disorder, prior suicidal ideation, etc.; Nock & Banaji, 2007).

Since depression is importantly being linked to suicidal ideation (e.g., Ellis, 2006), the first goal of the present study was to test the hypothesis that automatic self-associations of depression relate to suicidal ideation. However, recent research also showed a link between anxiety disorders and suicidal ideation even after correcting for depressive symptoms (Norton, Temple & Pettit, 2008; Sareen et al., 2005). Therefore, the second goal of this study was to examine whether automatic self-associations of anxiety were related to suicidal ideation in addition to automatic self-depressed associations. Data were collected among a large sample of anxious and depressed patients and non-clinical controls as part of the Netherlands Study of Depression and Anxiety (see: www.nesda.nl). We used two adapted versions of the Implicit Association Test (IAT; Greenwald, McGhee & Schwartz, 1998) to assess automatic self-depressed associations and automatic self-anxious associations. We hypothesized that both automatic self-depressed associations as well as automatic self-anxious associations may relate significantly to suicidal ideation. If this indeed

is the case, it could help explain the repetitive character of suicidal thoughts and the difficulty patients experience to control these thoughts.

## Methods

The study was carried out in the context of the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008), a multi-center, longitudinal, cohort study designed to examine the long-term course and consequences of anxiety and depressive disorders. This study concerns the baseline measurement that started in September 2004 and was completed in February 2007. The study protocol was approved centrally by the Ethical Review Board of the VU Medical Center Amsterdam and subsequently by the local review boards of each participating center/institute.

### *Participants*

NESDA has been designed to be representative of those with depressive and anxiety disorders in different health care settings and stages of the developmental history. Therefore, recruitment of respondents took place in the general population, in general practices, and in mental health care institutions and included a range of psychopathology: those with no symptoms or disorders ('controls'), those with prior episodes or at risk because of sub threshold symptoms or family history, and those with a current first or recurrent depressive or anxiety disorder. Across recruitment setting, uniform in- and exclusion criteria were used. A general inclusion criterion was an age of 18 through 65 years. Only two exclusion criteria existed: (1) a primary clinical diagnosis of a psychiatric disorder not subject of NESDA which would largely affect course trajectory: psychotic disorder, obsessive compulsive disorder, bipolar disorder, or severe addiction disorder, and (2) not being fluent in Dutch since language problems would harm the validity and reliability of collected data. The focus of NESDA is on Dysthymia (current: $n$ = 305; life-time: $n$ = 663)[5] , Major Depressive Disorder (MDD; current: $n$ = 1115; life-time: $n$ = 1925), General Anxiety Disorder (current: $n$ = 464; life-time: $n$ = 784), Panic Disorder (current: $n$ = 670; life-time: $n$ = 878), Social Phobia (current: $n$ = 665; life-time: $n$ = 908) and Agoraphobia (current: $n$ = 187; life-time: $n$ = 288). In total, 2981 participants (66.5% female; mean age = 41.9 years, SD = 13.0) were included in the NESDA study of whom 652 were non-clinical controls without any depressive or anxiety disorder (for a more detailed description of the NESDA sample, see Penninx et al., 2008).

[5] The numbers between brackets refer to the number of diagnoses included in the NESDA. Current refers to the 6-months prevalence. The number of life-time diagnoses includes current as well as diagnoses earlier in life. Several participants had more than one current/life-time diagnosis.

### *Measurements*

**Implicit Association Test.** The IAT is a computerized reaction time task originally designed by Greenwald et al. (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left or right response key. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated. The category labels are visible in the upper left and right-hand corners of the screen during the whole task (for an example see https://implicit.harvard.edu/implicit). Following the design of Egloff and Schmukle (2002), two IATs were constructed to measure automatic self-depressed associations and automatic self-anxious associations. For both IATs the target labels were *me* and *other*. The attribute labels were *depressed* and *elated* for the depression IAT and *anxious* and *calm* for the anxiety IAT. Each category consisted of five stimuli (see Chapter 2, Appendix 2.A). Both IATs consisted of two critical test blocks that were preceded by practice blocks (see Chapter 2, Table 2.1). In one test block me and depressed/anxious (and other and elated/calm) shared the same response key, whereas in the other test block me and elated/calm (and other and depressed/anxious) shared the response key. Before the start of a new sorting task, written instructions were presented on screen. After a correct response, the next stimulus was presented after 500 ms. Following an incorrect response, the Dutch word *FOUT!* (wrong) appeared shortly above the stimulus. Meanwhile, the stimulus remained on the screen until the correct response was given. The order of the category combinations was fixed across participants to reduce method variance. This is assumed to enhance the sensitivity of the IAT as a measure of individual differences, which is important in view of the prospective design of the NESDA (cf., Asendorpf et al., 2002; Schnabel, Banse & Asendorpf., 2006; Steffens & König, 2006).

To obtain explicit self-beliefs of depression and anxiety, participants rated all 20 IAT attribute stimuli on a 5-point scale (1 = hardly/not at all, 5 = very much) (i.e., "For each word please indicate to what extent you think it generally applies to you"). To compute the explicit self-beliefs, the mean ratings of the depressed (anxious) IAT-stimuli were subtracted from the mean ratings of the elated (calm) IAT-stimuli. Hence, a positive effect indicates a stronger explicit link between self and elated/calm. The internal consistency of the explicit self-beliefs was good, with Crohnbach's *α's* for four subscales (anxious, calm, depressed, elated) varying from .89 to .91.

**Scale for Suicidal ideation.** The Scale for Suicide Ideation (SSI; Beck, Kovacs & Weiss-

man, 1979) was used to measure suicidal ideation during the past week. The original scale contained 19 items, but items 6-19 are only asked when individuals score positive on the first items. Because the skewness of the SSI tends to be very high, it was decided only to include the first five items as part of the NESDA interview. Each item had three response categories (e.g. "Which feelings did you have during the past week concerning life and death? Did you want to live and how strong was this wish?" Scoring options: 0 - moderate to strong; 1 - weak; 2 - no wish). It appeared that the scores on the SSI were not normally distributed, because the majority of participants was non-suicidal.

**Item 18 of the Inventory of Depressive Symptoms self-report version.** As a second measure of suicidal ideation during the past week, we used item 18 'Thoughts of death and suicide', which is part of the self-report Inventory of Depressive Symptoms (IDS-SR; Rush, Gullion, Basco & Jarrett, 1996). This is a multiple-choice item existing of four response possibilities (0 – "I don't think of suicide or death"; 1 - "I have the feeling my life is empty and I wonder if it is still worth living for"; 2 - "Several times a week I think of suicide or death"; 3 - "Several times a day, I think seriously about suicide or death, or I made suicide plans, or I already made a suicide attempt"). Again the outcome was not normally distributed.

**Suicidal ideation.** Because both measures of suicidal ideation were not normally distributed, it was not possible to use these measures as dependent variables in a normal regression analysis. Therefore, we decided to transform the outcomes of both measurements into one dichotomous variable where we grouped together the participants who scored above zero on both measures of suicidal ideation. Thus, participants who scored positive on one of the five items of the SSI and at the same time on item 18 of the IDS-SR were categorized as 'suicidal'. Participants scoring zero on both measures were categorized as 'non-suicidal'. Participants who had a missing value or that scored only positive on one of the measures were excluded from the analyses. Additionally, participants were asked if they had ever made a serious suicide attempt during their lives. This question was used as a dichotomous variable for past suicide attempt (yes/no).

**Diagnostic assessment.** Depressive and anxiety disorders were determined by means of the lifetime Composite International Diagnostic Interview (CIDI) (WHO version 2.1, Robins et al., 1989), which classifies diagnoses according to the DSM-IV criteria (American Psychiatric Association, 2000). In order to conduct the study, more than 40 research assistants (psychologists, nurses, and residents in psychiatry) were trained during one week by the fieldwork coordinator. All interviews were taped to monitor data quality and interviewer performance.

### *Procedure*

The assessment lasted between three and five hours. During the assessment, first self-report questionnaires, the CIDI and Scale for Suicide Ideation were obtained, and then the IATs and explicit self-beliefs were administered. In between, other measurements were collected, but these are not of interest for the present study (for a detailed description, see Penninx et al., 2008). When the assessment was completed, respondents were compensated with a small incentive (a €15,- gift certificate and travel expenses) for their time and cooperation.

### *Data analyses*

**Data reduction IAT.** IAT scores were computed according to the now widely used algorithm proposed by Greenwald, Nosek, and Banaji (2003). We report the $D_4$-measure. Following these guidelines, all reaction times above 10,000 ms were discarded and error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred plus a penalty of 600 ms. For the anxiety IAT, the IAT effect was calculated by subtracting the mean reaction times of Block 6 from Block 3 (practice) and Block 7 from Block 4 (test). The means of these two effects were divided by their pooled standard deviation based on all responses in Blocks 3, 4, 6 and 7. Analogously, the IAT effect was calculated for the depression IAT, based on Blocks 9, 10, 12 and 13.

Positive IAT effects indicate relatively fast responses when *me* shared the response key with either *calm* or *elated*.[6] The split-half reliabilities of the present IATs was good, with Spearman-Brown corrected correlations between test-halves of .82 for the depression IAT and .87 for the anxiety IAT (test-halves were based on trials 1, 2, 5, 6, 9, 10 etc vs. 3, 4, 7, 8, 11, 12 etc.).

**Statistical analyses.** The distributions of the measures for suicidal ideation were not normal, because non-suicidal individuals were overrepresented. Therefore, binary logistic regression was used to predict the dichotomous variables of suicidal ideation and past suicide attempt. Logistic regression follows the same general principle of linear regression; it is used when the outcome variable is a categorical dichotomy. Relations between the predictors and suicidal behaviour are presented by means of odds ratios, which indicate the increased likelihood of suicidal behaviour given an increase of one unit in the

[6] Please note that this scoring procedure is reversed compared to the published article in Journal of Psychopathology and Behavioral Assessment (Glashouwer, de Jong, Penninx, Kerkhof, van Dyck & Ormel, 2010). Originally we decided to make the D-measure comparable to symptom measures in which a higher score also indicates a less favourable outcome. However, thanks to an anonymous reviewers' comment on a different study, we decided to reverse the scoring to make them comparable to the general literature and the other studies in this thesis. The only thing that differs between this study and the published paper is the multiplication sign before the D-measure / explicit self-beliefs. In addition, the odds ratios in the logistic regression analyses are 'reversed'.

independent variable. The variables were standardized in order to make the odds ratios comparable. Furthermore, bivariate Spearman rank correlation coefficients were calculated between automatic and explicit self-depressed and self-anxious associations, suicidal ideation and suicide attempt. Additionally, although we were aware of the fact that the suicidal ideation and attempt variables were not continuous, partial correlations were reported to get a general idea of the shared variance between variables.

Our primary interest in the present paper was whether automatic self-associations of depression and anxiety relate to suicidal ideation. Therefore, hierarchic logistic regression analysis was performed with a theory-driven order of predictors. Since depression is importantly being linked to suicidal ideation, IAT depression was included in the first block. The second block consisted of IAT anxiety to examine whether automatic self-anxious associations added predictive value over and above automatic self-depressed associations. For the same reasons explicit self-depressed beliefs were added before explicit self-anxious beliefs in blocks 3 and 4. To test whether the relationship between automatic self-depressed (anxiety) associations and suicidal thoughts would be especially strong in people also showing enhanced explicit self-depressed (anxiety) associations, the interaction between automatic self-depressed associations and explicit self-depressed beliefs was added in step 5 and the same interaction for anxious self-associations was added in step 6. All tests were conducted with $\alpha < .05$.

## Results

### *Descriptives*

Due to technical problems, the IAT data and explicit self-beliefs for 129 participants were missing. Furthermore, ten participants were discarded from all analyses because more than 10% of the IAT trials were below 300 ms (Greenwald, Nosek & Banaji, 2003), suggesting that they were trying to respond too rapidly. Five participants were discarded because of unusual D-scores (> 5 SD divergent from mean), that were explained by high error rates (> 28.8%). Additionally, 46 values were missing on the dichotomous measurement of suicidal ideation, because questionnaires were not returned, or because participants did not answer (1 on the Scale for Suicide Ideation; 45 on item 18 of the IDS-SR) and 570 participants were excluded from the analysis because they only scored positive on one of the two suicidal ideation measures. Finally, 17 values were missing on the measure of suicide attempt. Missing information on any of the variables resulted in exclusion of the case from the particular analysis. Both the scores on the SSI and item 18 of the IDS-SR were not normally distributed, because the majority of participants was non-suicidal (SSI:

Skewness = 4.47, SD = .05; Kurtosis = 22.53, SD = .09; item 18 IDS-SR: Skewness = 1.58, SD = .05; Kurtosis = 1.35, SD = .09). Consequently, one combined dichotomous measure of suicidal ideation was constructed, as is explained in the method section. The descriptives of the variables as well as diagnoses of major depressive disorder and anxiety disorders during the past month for groups with and without suicidal ideation are reported in Table 3.3. The correlations between automatic and explicit self-depressed and self-anxious associations, suicidal ideation and attempt are shown in Table 3.4. Partial correlations of the predictors with suicidal ideation and attempt are shown in Table 3.5.

Table 3.3

*Means and standard deviations of variables and number of diagnoses during the past months for suicidal and non-suicidal participants*

| **Measure** | **Suicidal ideation** ***n* = 271** | **No suicidal ideation** ***n* = 1950** |
|---|---|---|
| Gender, % female | 65.7 | 65.7 |
| Age | 41.39 (11.70) | 41.53 (13.34) |
| Educational level in years | 11.66 (3.37) | 12.41 (3.22)* |
| IAT depression, D-measure | .02 (.40) | .29 (.38)* |
| IAT anxiety, D-measure | .07 (.49) | .34 (.50)* |
| EA depression | -.89 (1.49) | 1.93 (1.33)* |
| EA anxiety | -1.14 (1.35) | 1.11 (1.54)* |
| Suicide Attempt, % yes | 29.5 | 6.3* |
| Dysthymia | 89 (32.8%[a]) | 83 (4.3%[a])* |
| Major Depression | 198 (73.1%[a]) | 287 (14.7%[a])* |
| Social Phobia | 115 (42.4%[a]) | 253 (13.0%[a])* |
| Panic Disorder with Agoraphobia | 57 (21%[a]) | 165 (8.5%[a])* |
| Panic Disorder without Agoraphobia | 31 (11.4%[a]) | 72 (3.7%[a])* |
| Agoraphobia | 18 (6.6%[a]) | 81 (4.2%[a]) |
| Generalized Anxiety Disorder | 96 (35.4%[a]) | 161 (8.3%[a])* |
| None of the diagnoses above | 27 (10.0%[a]) | 1278 (65.5%[a])* |

*Note*. IAT = Implicit Association Test; EA = Explicit Associations; * Difference between the two groups is significant at the 0.01 level (2-tailed) [a] % of the suicidal/non-suicidal group that has this diagnosis; note that the percentages do not add up to 100, because individuals often had more than 1 diagnosis.

Table 3.4

*Correlation matrix of automatic and explicit self-anxious and self-depressed associations, suicidal ideation and attempt over all participants (N = 2837)*

| **Measure** | **1.** | **2.** | **3.** | **4.** | **5.** | **6.** |
|---|---|---|---|---|---|---|
| 1. IAT anxiety | - | .49* | .37* | .32* | -.18* | -.11* |
| 2. IAT depression | | - | .35* | .38* | -.22* | -.12* |
| 3. EA anxiety | | | - | .79* | -.40* | -.19* |
| 4. EA depression | | | | - | -.47* | -.23* |
| 5. Suicidal ideation [a] | | | | | - | .26* |
| 6. Suicide attempt [a] | | | | | | - |

*Note*. IAT = Implicit Association Test; EA = Explicit Associations; *Correlation is significant at the .01 level (2-tailed); [a] These are dichotomous variables, '0' stands for non-suicidal/no attempt and '1' stands for suicidal/attempt.

Table 3.5

*Partial correlation matrix of automatic and explicit self-anxious and self-depressed associations with suicidal ideation (n = 2221) and attempt over all participants (n = 2820)*

| **Dependent Variable** | **Variable** | **Variable corrected for** | | | |
|---|---|---|---|---|---|
| | | **1. IAT anxiety** | **2. IAT depression** | **3. EA anxiety** | **4. EA depression** |
| Suicidal ideation | 1. IAT anxiety | - | -.08** | -.03 | - [a] |
| | 2. IAT depression | -.16** | - | - [a] | -.02 |
| | 3. EA anxiety | -.40** | - [a] | - | .01 |
| | 4. EA depression | - [a] | -.53** | -.40** | - |
| Suicide attempt | 1. IAT anxiety | - | -.06** | -.04* | - [a] |
| | 2. IAT depression | -.07** | - | - [a] | -.03 |
| | 3. EA anxiety | -.17** | - [a] | - | -.02 |
| | 4. EA depression | - [a] | -.21** | -.14** | - |

*Note*. IAT = Implicit Association Test; EA = Explicit Associations; **Correlation is significant at the .01 level (2-tailed); *Correlation is significant at the .05 level (2-tailed); [a] Only partial correlations were calculated correcting for the same emotion (depression or anxiety) or the same measurement method (IAT or EA).

Table 3.6

*Summary of Logistic Regression Analysis for Predicting Suicidal Ideation (n = 2221)*

| Variable | Wald | OR | 95 % CI | $\chi^2$ |
|---|---|---|---|---|
| Step 1 | | | | 109.11** |
| IAT depression | 104.08** | .51 | .45 – .58 | |
| Step 2 | | | | 12.16** |
| IAT depression | 50.30** | .59 | .51 – .68 | |
| IAT anxiety | 12.11** | .77 | .66 – .89 | |
| Step 3 | | | | 538.63** |
| IAT depression | 1.01 | .91 | .76 – 1.09 | |
| IAT anxiety | .08 | .98 | .81 – 1.17 | |
| EA depression | 329.52** | .16 | .13 – .20 | |
| Step 4 | | | | 6.15* |
| IAT depression | .99 | .91 | .76 – 1.10 | |
| IAT anxiety | .02 | 1.02 | .84 – 1.22 | |
| EA depression | 172.19** | .20 | .15 – .25 | |
| EA anxiety | 6.17* | .73 | .57 – .94 | |
| Step 5 | | | | 4.05* |
| IAT depression | 4.63* | .74 | .57 – .97 | |
| IAT anxiety | .01 | 1.01 | .84 – 1.21 | |
| EA depression | 166.36** | .18 | .13 – .23 | |
| EA anxiety | 5.52* | .74 | .58 – .95 | |
| Int IATdep x EA dep | 4.14* | .75 | .57 – .99 | |
| Step 6 | | | | 5.40* |
| IAT depression | 2.25 | .81 | .61 – 1.07 | |
| IAT anxiety | 2.55 | .80 | .61 – 1.05 | |
| EA depression | 158.36** | .19 | .15 – .24 | |
| EA anxiety | 8.26** | .68 | .52 – .88 | |
| Int IATdep x EA dep | 1.31 | .84 | .62 – 1.13 | |
| Int IATanx x EA anx | 5.36* | .77 | .62 – .96 | |

*Note*. IAT = Implicit Association Test; EA = Explicit Associations; OR = Odds Ratio; CI = Confidence Interval.
**Correlation is significant at the .01 level (2-tailed). *Correlation is significant at the .05 level (2-tailed).

## *Are automatic self-associations predictive of suicidal ideation and suicide attempt?*

Hierarchic logistic regression analysis showed that automatic self-depressed associations significantly predicted suicidal ideation (Table 3.6) and suicide attempt (Table 3.7). These odds ratios indicate a decreased likelihood of suicidal ideation and past attempt,

Table 3.7

*Summary of Logistic Regression Analysis for Predicting Suicide Attempt (n = 2820)*

| **Variable** | **Wald** | **OR** | **95 % CI** | $\chi^2$ |
|---|---|---|---|---|
| Step 1 | | | | 38.82** |
| IAT depression | 38.89** | .70 | .62 – .78 | |
| Step 2 | | | | 10.35** |
| IAT depression | 14.32** | .78 | .68 – .89 | |
| IAT anxiety | 10.38** | .81 | .71 – .92 | |
| Step 3 | | | | 104.75** |
| IAT depression | .58 | .95 | .83 – 1.09 | |
| IAT anxiety | 2.70 | .89 | .78 – 1.02 | |
| EA depression | 103.02** | .53 | .47 – .60 | |
| Step 4 | | | | .92 |
| IAT depression | .63 | .95 | .82 – 1.09 | |
| IAT anxiety | 1.95 | .91 | .79 – 1.04 | |
| EA depression | 39.46** | .57 | .47 – .68 | |
| EA anxiety | .92 | .91 | .75 – 1.10 | |
| Step 5 | | | | 4.46* |
| IAT depression | 2.74 | .88 | .75 – 1.02 | |
| IAT anxiety | 2.18 | .90 | .78 – 1.04 | |
| EA depression | 44.12** | .54 | .45 – .65 | |
| EA anxiety | .55 | .93 | .77 – 1.13 | |
| Int IATdep x EA dep | 4.37* | .84 | .71 – .99 | |
| Step 6 | | | | .79 |
| IAT depression | 2.14 | .89 | .76 – 1.04 | |
| IAT anxiety | 2.95 | .87 | .75 – 1.02 | |
| EA depression | 42.50** | .55 | .46 – .65 | |
| EA anxiety | .79 | .92 | .76 – 1.11 | |
| Int IATdep x EA dep | 2.42 | .87 | .72 – 1.04 | |
| Int IATanx x EA anx | .79 | .94 | .81 – 1.09 | |

*Note*. IAT = Implicit Association Test; EA = Explicit Associations; OR = Odds Ratio; CI = Confidence Interval. **Correlation is significant at the .01 level (2-tailed). *Correlation is significant at the .05 level (2-tailed).

when people automatically associated themselves stronger with elated words. Furthermore, the results consistently showed that automatic self-anxious associations predicted suicidal ideation and past suicide attempt over and above the automatic self-depressed associations. In both analyses, the effects of the IATs disappeared when explicit self-depressed beliefs were entered into the regression model. Only in the prediction of suicidal

ideation, explicit self-anxious beliefs had predictive validity over and above explicit self-depressed beliefs. The interaction between automatic self-depressed associations and explicit self-depressed beliefs that was added in step 5 showed small, but differential predictive validity for both suicidal ideation and suicide attempt. Only in the prediction of suicidal ideation, the interaction between automatic self-anxious associations and explicit self-anxious beliefs added predictive validity on top of all other predictors. Together, this pattern of results indicates that individuals with depressed (anxious) explicit self-beliefs generally showed a greater probability of having suicidal thoughts than individuals with less depressive (anxious) self-beliefs, whereas this heightened probability of having suicidal thoughts was increased further for individuals who had also depressed (anxious) self-associations on a more automatic level.[7]

## Discussion

The present study was designed as a first step in getting more insight into the relationship between suicidal ideation and automatic self-associations of depression and anxiety. In line with what we expected, the results showed that automatic self-depressed associations were significantly related to suicidal ideation and past suicide attempt. Furthermore, automatic self-anxious associations showed predictive validity for suicidal ideation/attempt in addition to the automatic self-depressed associations. Although the main-effects of automatic self-associations did not explain additional variance over and above explicit self-beliefs, the interactions between automatic self-depressed (anxious) associations and explicit self-depressed (anxious) beliefs did.

It is an important, new observation that traces of dysfunctional self-schemas can be found in suicidal individuals both on an explicit, reflective level, and on a more automatic level (see e.g., Gawronski & Bodenhausen, 2006). The fact that automatic self-anxious associations even showed predictive validity for suicidal ideation/attempt in addition to automatic self-depressed associations is particularly new and in accordance with recent studies showing a relationship between anxiety disorder symptoms and suicidality (Norton et al., 2008; Sareen et al., 2005). Furthermore, the relationship between automatic self-depressed (anxious) associations and suicidal thoughts appeared especially strong in people also showing enhanced explicit self-depressed (anxiety) associations. In other words, the probability of having suicidal thoughts was especially high for individuals who

[7] We repeated the analyses in a subgroup of participants that did not use a benzodiazepine at present to see whether use of medication would influence the results. In general, the patterns of results were the same, except for the interaction effects that were no longer significant. However, this can also be the result of less power due to the smaller group sizes.

had depressed (anxious) self-associations both on an automatic level and an explicit level. Perhaps individuals with relatively positive explicit beliefs about themselves (e.g., "I am elated" or "I am calm") are better able and/or stronger motivated to neutralize or correct the influence of automatic negative self-associations than individuals with more negative explicit self-evaluations (cf. de Jong, van den Hout, Rietbroek & Huijding, 2003). In other words, in individuals with negative self-associations on both automatic and explicit levels this correction might not take place thereby enhancing the risk of eliciting suicidal ideation.

Although the interactions of automatic self-depressed (anxious) associations with explicit self-depressed (anxious) beliefs had only small additional predictive validity and automatic self-associations per se did not uniquely contribute to the prediction of suicidal ideation or attempt over and above their explicit counterparts, this does not imply that automatic self-associations are therefore largely redundant. For the understanding of suicidal ideation, it seems crucial to gain insight into the underlying cognitive structures that are relatively unconscious, especially because suicidal patients often report difficulties in controlling suicidal thoughts, and rapid and unconscious changes in their thinking about suicide. Furthermore, according to information-processing models (e.g., Fazio & Towles-Schwen, 1999; Wilson et al., 2000), automatic and explicit cognitions are assumed to predict different kind of behaviours. Explicit associations (beliefs) tend to predict more explicit, controlled behaviours, whereas automatic associations are most critical for guiding relatively spontaneous behaviours (Asendorpf et al., 2002; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a). There is evidence that this is especially important in circumstances where there is little cognitive capacity left to deliberate about these automatic associations. Accordingly, recent research in the context of alcohol abuse showed that working memory moderates the influence of automatic associations on drinking behaviour (Thush et al., 2008). Only in adolescents with a relatively small working memory capacity, automatic alcohol associations were predictive of drinking behaviour. Analogously, cognitive capacity could also play a role in the context of suicidal ideation. When cognitive capacity is limited, for example by natural ability or by other factors such as life-stress, dysfunctional automatic self-associations might obtain a stronger influence (Beevers, 2005). In these cases, the automatic activation of dysfunctional self-associations could function as a repeating trigger for repetitive negative thoughts of suicide or death, even when more positive beliefs might exist on an explicit level.

In the present study the focus was on automatic self-associations of depression and anxiety. Therefore, it cannot be ruled out that other automatic self-associations may still

be independently related to suicidal ideation. Moreover, it should be acknowledged, that the present approach may have resulted in an underestimation of the importance of automatic self-associations in the context of suicidal behaviour. That is, in the present study the relationship between explicit self-beliefs and suicidal ideation may have been artificially increased because there was methodological overlap between these measures (both were self-report measures). However, the present strong association between suicidal ideation and explicit negative self-beliefs is consistent with earlier findings (e.g., Beck & Steward, 1989, cited in Weishaar & Beck, 1992; De Man & Gutiérrez, 2002; Evans et al., 2004) and seems to underline once more the importance of dysfunctional self-schemas for suicidal ideation.

In conclusion, the present study clearly showed that automatic self-associations are related to suicidal thoughts, which is in line with clinical experience and the theoretical starting point that suicidal thoughts stem from dysfunctional schemas in memory. The finding that this does not only concern automatic self-depressed associations, but that also automatic self-anxious associations are related to suicidal ideation, is especially new. Although automatic self-associations per se did not explain additional variance over and above explicit self-beliefs, the interaction between automatic and explicit self-associations did. This suggests that the probability of having suicidal thoughts was especially high for individuals who had depressive (anxious) self-associations on both an automatic and an explicit level. Future research has to affirm whether this indeed is a robust finding that needs further investigation. Furthermore, it would be important to see whether automatic self-associations have perhaps prognostic value for the course of suicidal ideation and to test whether the predictive validity of automatic associations for suicidal ideation is moderated by cognitive capacity. Together, these results provide an initial insight into the more unconscious parts of cognitive structures that underlie suicidal ideation, which is one plausible explanation of why suicidal patients might report difficulties in preventing and managing suicidal thoughts.

# 4 Dysfunctional automatic associations in social anxiety disorder

# Study 1: Fear of blushing

## Abstract

To explain fear of blushing, it has been proposed that individuals with fear of blushing overestimate the social costs of their blushing. Current information-processing models emphasize the relevance of differentiating between more automatic and more explicit cognitions, as both types of cognitions may independently influence behaviour. The present study tested whether individuals with fear of blushing expect blushing to have more negative social consequences than controls, both on an explicit level and on a more automatic level. Automatic associations between blushing and social costs were assessed in a treatment-seeking sample of individuals with fear of blushing who met DSM-IV criteria for social anxiety disorder ($n$ = 49) and a non-anxious control group ($n$ = 27) using a single-target Implicit Association Test (stIAT). In addition, participants' explicit expectations about the social costs of their blushing were assessed. Individuals with fear of blushing showed stronger associations between blushing and negative outcomes, as indicated by both stIAT and self-report. The findings support the view that automatic and explicit associations between blushing and social costs may both help to enhance our understanding of the cognitive processes that underlie fear of blushing.

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## Introduction

Blushing is a common physiological response assumed to occur in situations in which people suddenly become conscious of themselves (Crozier, 2006). While there is evidence that blushing is functional for interpersonal communication (e.g., Leary, Landel & Patton, 1996; Dijk & de Jong, 2009; Dijk, Koenig, Ketelaar & de Jong, 2011), people often experience their blushing as an undesirable response to the extent that they try to prevent or hide it (Shields, Mallory & Simon, 1990). For some individuals blushing is so unpleasant that they develop a fear of blushing (e.g., Mulkens, Bögels, de Jong & Louwers, 2001). It is increasingly common for individuals with fear of blushing to undergo interventions for their blushing (e.g., surgical procedures; Dijk & de Jong, 2006), which underlines the negative consequences of this condition. Consequently, it seems essential to increase insight into the cognitive processes that may underlie fear of blushing.

Recently, it was proposed that the highly negative evaluation of blushing in individuals with fear of blushing may be due to an overestimation of the social costs of blushing. That is, individuals with fear of blushing may fear being seen by others as incompetent, unlikeable, and unreliable (e.g., Bögels & Reith, 1999; Dijk, de Jong, Müller & Boersma, 2010; Dijk, Voncken & de Jong, 2009). In previous studies, we have used vignettes to assess in individuals with fear of blushing their expectations of displaying a blush in several types of situations: after a social transgression, when being the center of attention, during the disclosure of something personal, and in circumstances in which people usually do not blush (de Jong & Peters, 2005; de Jong et al., 2006; Dijk & de Jong, 2009; Dijk et al., 2010). These studies showed that, compared to individuals with low fear of blushing, individuals with high fear of blushing anticipated heightened social costs of their blushing only in circumstances during which people usually do not blush. In another recent study, individuals were asked about their cognitions concerning blushing without providing a specific context. This study also showed that individuals with fear of blushing expected blushing to be associated with higher social costs than individuals with low fear of blushing (Dijk et al., 2010). Together, these findings seem to indicate that fear of blushing is characterized by an overestimation of the social costs of blushing.

Thus far, research into fear of blushing has mostly relied on self-report questionnaires and therefore has been focused on the consciously accessible traces of blushing cognitions. However, dual process models emphasize the need to distinguish between these so-called explicit beliefs and more automatic levels of information-processing. These models assume that behaviour is not only guided by cognitions of which people are consciously aware, but also by processes that occur more automatically, and often invol-

untary, or even completely outside awareness (e.g., Gawronski & Bodenhausen, 2006). Current cognitive models of anxiety disorders stress the importance of these automatic processes and assume that threatening stimuli will directly activate fear associations in memory. In turn these associations will automatically elicit fear responses such as feelings of anxiety, anxious thoughts and defensive behaviours (Beck & Clark, 1997). Especially in circumstances in which individuals have little opportunity or limited cognitive capacity to reflect on the situation, fear responses occur more spontaneously and are assumed to result from these more automatic associations (Fazio & Towles-Schwen, 1999). In support of this, several studies have shown that automatic associations have predictive validity for relatively spontaneous fear responses (e.g. Spalding & Hardin, 1999; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a). Likewise, automatic associations may also play an important role in fear of blushing. Thus, for a proper understanding of the processes underlying fear of blushing it may be of critical importance to conduct research testing the relevance of more automatically activated associations between blushing and social costs. This will complement previous research that predominantly focused on the more explicit cognitions regarding social costs (de Jong & Peters, 2005; de Jong et al., 2006; Dijk & de Jong, 2009; Dijk et al., 2010).

Therefore, the present study was designed to examine whether individuals with a fear of blushing display stronger automatic associations between blushing and social costs than controls without fear of blushing. To assess the strength of these automatic associations, we used a single-target Implicit Association Test (stIAT) shown to be sensitive to individual differences (e.g., Huijding & de Jong, 2006b; Karpinsky & Steinman, 2006; Thush & Wiers, 2007; Wigboldus, Holland, van Knippenberg, den Hartog & Belles, 2002). The stIAT is used in the present study as a context-independent measure of automatic blushing associations. Therefore as an explicit equivalent of the automatic blushing associations we also included a self-report measure of social costs that was context-independent (cf. Dijk, et al., 2010). Prior studies on the role of anticipated social costs in fear of blushing have predominantly relied on analogue samples. However, there may be quantitative as well as qualitative differences between analogue and clinical samples (Emmelkamp, 1982), and so the present study included a clinical sample. This clinical sample consisted of treatment-seeking individuals with social anxiety disorder and fear of blushing as their primary complaint. This was not only important for the external validity of the present study, but also made it possible to see whether previous findings from analogue samples regarding explicit self-reported social costs of blushing could be replicated in a clinical sample.

## Method

### *Participants and procedure*

The data presented here are part of a larger study on the efficacy of a new psychoeducational intervention for individuals with fear of blushing (Dijk, Buwalda & de Jong, 2012). Participants were 52 individuals who applied for treatment because of their fear of blushing and 27 non-fearful controls. Fearful participants applied for treatment after reading articles about a fear of blushing treatment study that appeared in both local and national media. Control participants were all indirect acquaintances of the staff members of the Department of Clinical Psychology of the University of Groningen who were asked via these staff members to serve as control participants in a study on fear of blushing. They were screened with the blushing scale of the Blushing Trembling Sweating Questionnaire (BTSQ; Bögels & Reith, 1999). Control participants scoring over 50 on the blushing scale of the BTSQ were considered fearful of blushing and therefore excluded ($n = 1$). Mean BTSQ blushing scores for the control group were lower than those of the fearful group (Table 4.1). The two groups were similar with respect to gender, age, and educational level.

Individuals with fear of blushing were included when they met the DSM-IV criteria (American Psychiatric Association, 2000) for social phobia on the Minnesota International Neuropsychiatric Interview-Plus (MINI-Plus, version 5.0.0; Sheehan et al., 1998) and had fear of blushing as the main symptom. The MINI-Plus is a concise structured interview that can be used as a screening instrument for the most important Axis I diagnoses according to the DSM-IV. Five participants with fear of blushing had a comorbid diagnosis: one participant suffered from agoraphobia, one participant from panic disorder, one participant had a current depressive episode, one participant was dysthymic with low suicidality as well as agoraphobic and one participant was afraid of needles. The control group was not screened for additional psychopathological symptoms, because having additional psychopathological symptoms was allowed for all participants. Further inclusion criteria for all participants were being 18 years or older and having a good command of the Dutch language. Exclusion criteria for the individuals with fear of blushing were the presence of other DSM-IV Axis I disorders more prominent than fear of blushing and prior or ongoing Cognitive Behavioural Therapy (CBT). The stIAT was administered after the MINI-plus was finished. It was presented as separate from the psychoeducational intervention and participants could choose not to participate but still undergo the intervention. The test session lasted about 45 minutes and consisted of the stIAT followed by self-report questionnaires, all administered by a computer. By using a fixed order of administration we tried to minimize the influence of the self-reports on the stIAT (cf. Bosson, Swann & Pennebaker, 2000).

Table 4.1

*Means and standard deviations of variables as a function of group*

| Variables | Group | | T-statistics |
|---|---|---|---|
| | NFC | FB | |
| | *n* = 27 | *n* = 49 | |
| Gender, % female | 77.8 | 73.5 | .41 |
| Age | 34.30 (12.68) | 39.43 (11.35) | 1.81 |
| Educational level [a] | 2.67 (.92) | 2.61 (.93) | .25 |
| stIAT blushing, D-measure | -.13 (.47) | -.37 (.42) | 2.29* |
| stIAT, % error trials: overall | 6.62 (6.24) | 8.28 (6.17) | 1.12 |
| stIAT, % error trials: pairing blush-positive | 5.65 (6.48) | 11.77 (10.92) | 3.07** |
| stIAT, % error trials: pairing blush-negative | 5.59 (6.95) | 4.80 (4.23) | 1.90† |
| Others' evaluations [b] | 6.00 (1.62) | 3.23 (1.56) | 7.32** |
| Blushing Questionnaire [c] | 9.16 (6.97) | 73.77 (12.59) | 24.62** |

*Note*. stIAT = single target Implicit Association Test; NFC = non-fearful controls; FB = individuals with fear of blushing; [a] educational level in categories of '1' tot '4', where '1' stands for lower education and '4' for higher education; [b] 9-item subscale of the Conditional Cognition scale; [c] 5-item subscale of the Blushing, Trembling, and Sweating Questionnaire. ** $p < .01$ (2-tailed); * $p < .05$ level (2-tailed); † $p < .10$ (2-tailed).

## *Measurements*

**Fear of blushing.** Severity of fear of blushing was measured with the 'blushing' subscale of the BTSQ. On this 5-item subscale, people answer on a Visual Analogue Scale questions with respect to their fear of blushing (e.g. "How afraid are you that you will be blushing?" Not at all afraid – Very afraid). Mean subscale scores (range 0 -100) were calculated over the 5 items.

**Single target Implicit Association Test (stIAT).** To assess the strength of automatic blushing associations we used a single-target Implicit Association Test (stIAT) originally designed by Wigboldus and colleagues (2002). The stIAT is a computerized reaction time task that measures to what extent a single target category is associated with two attribute categories. Following prior designs (Huijding & de Jong, 2006b; Karpinsky & Steinman, 2006; Thush & Wiers, 2007; Wigboldus et al., 2002), a blushing stIAT was constructed with the target category *blushing* and the attribute categories *positive outcome* and *negative outcome*. Positive and negative social judgments of blushing typically contain attributes from the domains of competence, sociability, likeability, and reliability (e.g., Dijk & de Jong, 2009). Consequently, attributes from these domains were used as stimuli in the present

stIAT. Each category consisted of five stimulus words. These were colour, tomato, blush, glow, and red for *blushing*, charming, fun, social, sincere, and kind for *positive outcome*, and shame, fail, flop, disappointing and disapproval for *negative outcome* (translated from Dutch). Stimulus words from all three categories appeared in randomized order in the middle of a computer screen and participants were instructed to sort them with a left or right response key. The category names stayed visible in the upper left and right-hand corners of the screen during the whole task. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated (e.g., for a person with fear of blushing it is probably easier to categorize words of *blushing* and *negative outcome* with the same button than *blushing* and *positive outcome*). The task consisted of two critical test blocks that were preceded by practice blocks (Table 4.2). In one test block *blushing* and *positive outcome* were mapped on one response key, and *negative outcome* on the other. In the other test block *blushing* and *negative outcome* were mapped on one key and *positive outcome* on the other. To prevent response bias, correct responses of the test blocks were divided equally over the two response keys (cf. Bluemke, Fiedler & Richter, 2009). Before the start of a new sorting task, written instructions were presented on the screen. After a correct response, the next stimulus was presented with a 500 ms delay. After an incorrect response, the Dutch word *FOUT!* (wrong) appeared shortly above the stimulus, and the stimulus remained on the screen until the correct response was given. The order of test blocks was counterbalanced between participants.

**Explicit blushing cognitions.** Participants' explicit expectations about the social costs of their blushing were measured with the others' evaluations subscale of the Conditional Cognition scale (see Dijk et al., 2010). This 9-item subscale contains questions aiming at

Table 4.2

*Arrangement of stIAT blushing*

| **Block** | **Left** | **Right** | **Practice/Test)** |
|---|---|---|---|
| 1 | Negative | Blushing + Positive | Practice |
| # Stimuli | 5 | 5 + 5 | |
| 2 | Negative | Blushing + Positive | Test |
| # Stimuli | 40 | 20 + 20 | |
| 3 | Blushing + Negative | Positive | Practice |
| # Stimuli | 5 + 5 | 5 | |
| 4 | Blushing + Negative | Positive | Test |
| # Stimuli | 20 + 20 | 40 | |

participants' cognitions about others' evaluations when blushing ("when I blush, others will think I am… *competent*, *insecure*, etc."). The questions could be answered on a scale from 0 (applies totally to me) to 10 (does not apply to me at all). Mean subscale scores were calculated over the 9 items; higher scores indicate more positive expectations of blushing.

### *Data analyses*

stIAT effects were computed according to the now widely used algorithm proposed by Greenwald, Nosek, and Banaji (2003) that can also be used for analyzing the stIAT (cf. Karpinsky & Steinman, 2006; Thush & Wiers, 2007). In this paper, we report the so-called $D_4$-measure[8]. Following the algorithm, all reaction times above 10,000 ms were discarded. Error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred, plus a penalty of 600 ms. The stIAT effect was calculated by subtracting the mean reaction times of Block 2 (4) from Block 4 (2). This effect was divided by the pooled standard deviation based on all responses in Blocks 2 and 4. Practice trials were discarded from the analyses, because we considered these trials to be truly practice: each stimulus was presented once to the participant and likewise the responses were not divided equally over the response keys. Negative stIAT effects indicate faster responses when *blushing* shared the response key with *negative outcome*. The bivariate Pearson's correlation coefficient was used to calculate the strength of the association between stIAT and explicit cognitions. T-tests were used to calculate the simple group effects in Table 4.1 and in Figure 4.1 and univariate ANOVAs were used to examine group differences on stIAT blushing and explicit cognitions. The tests were conducted with $\alpha = .05$.

## Results

### *Descriptives*

**Missing values.** Due to technical problems, stIAT data for 2 participants were missing. Furthermore, 1 participant appeared fully inexperienced with the use of computers and took over 20 minutes to complete the stIAT. Consequently, the data of these 3 participants were excluded from all analyses. Therefore, the group with fear of blushing (FB) included 49 participants and the non-fearful control group (NFC) included 27 participants. Group mean scores on the stIAT and other variables are reported in Table 4.1. The split-half reli-

[8] Because the present IAT design did not record the second correct response after a mistake, no built-in error penalty could be used. However, we repeated the analyses with the other available variants of the D-measure ($D^3$, $D^5$, $D^6$), but did not find any differences with respect to the outcomes.

ability of the present stIAT was excellent, with a Spearman-Brown corrected correlation between test-halves of .92 ($D_4$-measure based on odd and even trials). Furthermore, Cronbach's Alpha was calculated over the 9 items of the others' evaluations subscale of the Conditional Cognition scale and over the 5 items of the BTSQ blushing subscale. Both subscales showed good internal consistency (others' evaluations: $\alpha$ = .84; blushing: $\alpha$ = .96).

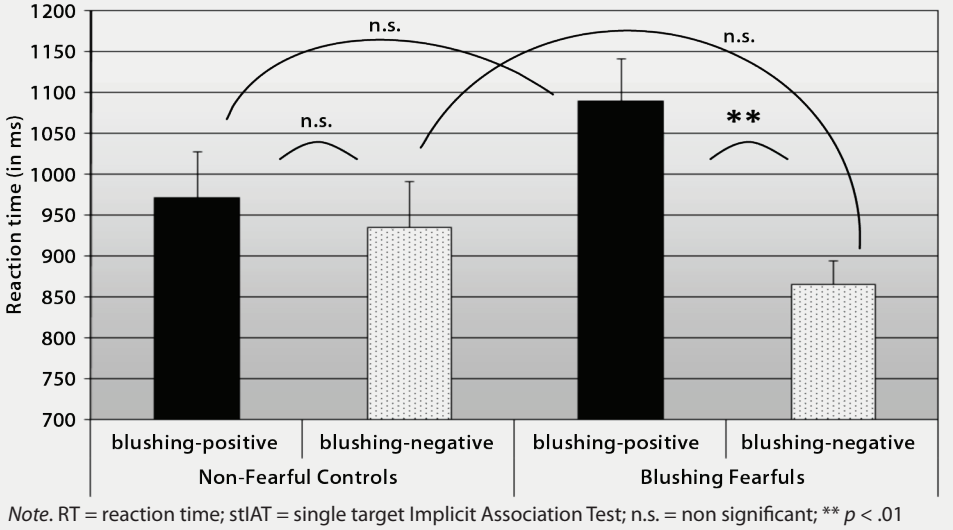
## *Do individuals with a fear of blushing differ in their blushing associations from non-fearful individuals?*

A 2 Group (FB, NFC) x 2 Order (blushing-positive first, blushing-negative first) ANOVA on automatic blushing associations showed a significant main effect for Group ($F(1,72)$ = 5.18, $p < .05$, partial $\eta^2 = .07$). As expected, the fearful group showed significantly stronger automatic negative blushing associations (i.e., a faster response when *blushing* and *negative outcome* shared the same response key) than the control group (see Figure 4.1). The main effect of Order, and the interaction between Order and Group were non-significant ($p$'s > .1).

A 2 Group (FB, NFC) ANOVA on explicit blushing cognitions showed a significant main effect for Group ($F(1,74) = 53.64$, $p < .01$, partial $\eta^2 = .42$) indicating that individuals with fear of blushing had more negative explicit expectations about the social costs of their

Figure 4.1

*Mean RTs of the trials per pairing of the st-IAT blushing: individuals with fear of blushing vs. non-fearful controls*




*Note*. RT = reaction time; stIAT = single target Implicit Association Test; n.s. = non significant; ** $p < .01$ (2-tailed).

blushing than the control group. The stIAT scores and explicit cognitions were shown to be largely unrelated ($r$ = .17, n.s.). This was also evident when correlations were computed for each group separately (fearfuls: $r$ = -.03, n.s.; controls: $r$ = .06, n.s.).

## Discussion

The present study was designed as a first step in getting more insight into the role of automatic associations between blushing and social costs in fear of blushing in a treatment-seeking sample of individuals with fear of blushing. In line with our predictions, the results showed that the fearful group was characterized by stronger automatic associations between blushing and negative social outcomes (and/or weaker automatic associations between blushing and positive outcomes) than the control group. In addition, individuals with fear of blushing had more negative explicit expectations about the social costs of their blushing than the non-fearful control group. Interestingly, explicit cognitions and automatic associations were shown to be largely unrelated, attesting to the relevance of measuring both automatic and explicit blushing associations for fear of blushing.

In an attempt to explain the highly negative evaluation of blushing in individuals with fear of blushing, it has been proposed that fearful individuals overestimate the social costs of their blushing (Dijk et al., 2009). Furthermore, it has been argued that in addition to explicit beliefs, more automatic dysfunctional associations may be critically involved in fears, such as fear of blushing (e.g., Ouimet, Gawronski & Dozois, 2009). In line with the latter, the present study provided evidence that treatment-seeking individuals with fear of blushing show stronger automatic associations between blushing and social costs than non-fearful controls. In addition, the present findings regarding the explicit index of the anticipated social costs of displaying a blush replicated earlier findings from analogue samples (Dijk et al., 2010). Thus, the present results indicate that individuals with fear of blushing are characterized both by explicit negative expectations about the social costs of their blushing and by more automatic associations between blushing and social costs.

While the pattern of results was in the same direction for automatic as well as explicit blushing associations, we did not find a significant correlation between both types of associations. This fits well within recent information-processing models concerning fear that consider explicit and automatic associations as distinct cognitive processes that influence different kinds of behaviours (e.g., Gawronski & Bodenhausen, 2006; Ouimet et al., 2009). In individuals with fear of blushing, automatic associations between blushing and social costs might trigger automatically initiated fear responses such as fearful thoughts and behavioural responses (cf. Strack & Deutch, 2004). Even when individuals with a fear of

blushing have sufficient time and cognitive resources, they will probably not correct these initial dysfunctional associations on a more explicit level, because their explicit beliefs about blushing are similarly negative and dysfunctional. This way automatic and explicit cognitions about blushing may both act to sustain or even enhance the preoccupation with blushing in individuals with fear of blushing (cf. Gawronski & Bodenhausen, 2006). Thus, automatic and explicit blushing associations do not seem to be simply redundant. This signifies the importance of measuring both types of cognitions in order to obtain a more comprehensive understanding of the processes that may underlie fear of blushing.

To further examine the causal influence of automatic blushing associations, it will be important to test whether the experimental manipulation of these automatic associations leads to changes in fear of blushing (cf. Clerkin & Teachman, 2010). Furthermore, the differential predictive validity of automatic versus explicit blushing associations should be tested for actual fearful behaviour in social contexts that might elicit a blushing response (cf. Huijding & de Jong, 2006a). If dysfunctional automatic associations do indeed play a causal role in the generation and/or maintenance of anxiety symptoms, then both explicit and automatic levels of information processing should be addressed in therapy. Following this, it seems important to investigate whether dysfunctional automatic associations decrease or even disappear under the influence of conventional CBT strategies. On the one hand, it seems possible that CBT might change automatic processes, for example via explicit attitude change (cf. Gawronski & Bodenhausen, 2006) or via behavioural experiments or repeated exposure-in-vivo. On the other hand, it could be that residual dysfunctional automatic associations after CBT are involved in the recurrence of symptoms (cf. Glashouwer, de Jong & Penninx, 2011). In the latter case, existing treatments should be adjusted or extended in a way that dysfunctional cognitions are targeted on both explicit and more automatic levels of information processing.

While the present findings suggest that automatic and explicit associations seem to be distinct cognitive processes, the observed group differences for explicit associations were found to be much larger than for automatic blushing associations. However, it cannot be ruled out that the difference in effect sizes is largely attributable to the methodological approach used. Since fear of blushing and explicit blushing associations were both measured via self-report measures, considerable method variance could be shared between explicit blushing associations and fear of blushing that is not shared with the automatic associations, making it a much more stringent test for the automatic associations. Secondly, it should be acknowledged that the categories used in the stIAT were not explicitly labeled in terms of social costs but more generally as negative vs. positive

outcomes. Future research will have to determine whether more specific automatic cost associations are indeed important in fear of blushing, or whether individuals with fear of blushing are characterized by negative automatic associations with blushing in general. Thirdly, the control group in the present design was not assessed for clinical status. Possibly the degree of psychopathological symptoms was low in the present control group, and, therefore, it cannot be ruled out that other clinical groups besides individuals with fear of blushing might also show negative associations with blushing. Lastly, it would be interesting to compare social anxiety patients with and without a fear of blushing to see whether negative blushing associations are specific for individuals with a fear of blushing or a more general characteristic of social phobia. This comparison could also rule out the possibility that socially anxious individuals have a general tendency to judge situations more negatively, irrespective of blushing per se.

To conclude, the present study not only showed that individuals with fear of blushing explicitly indicate that blushing is associated with negative outcomes, it also revealed that at a more automatic level blushing elicits negative automatic associations in this group. Both types of processes appear to be separate constructs that may help to increase our understanding of fear of blushing. These results imply that it might be valuable to include assessments of automatic associations in addition to the more traditional self-report questionnaires, for example as pre and post measures in the evaluation of treatment (e.g., Huijding & de Jong, 2007, 2009; Reinecke, Soltau, Hoyer, Becker & Rinck, 2012; Teachman & Woody, 2003). When conventional treatments do not sufficiently alter dysfunctional automatic associations, it might also be relevant to design new ingredients of CBT that more directly target these automatic associations. As a case in point, it has recently been shown that a computerized association task was effective in reducing social anxiety via modifying participants' dysfunctional automatic associations (Clerkin & Teachman 2010). Together, these findings may help to further increase our understanding of the cognitive processes that underlie fear of blushing.

# Study 2: Social anxiety disorder

## Abstract

Negative automatic associations towards the self and social cues are assumed to play an important role in social anxiety disorder. We tested whether social anxiety disorder patients ($n$ = 45) showed stronger dysfunctional automatic associations than non-clinical controls ($n$ = 45) and panic disorder patients ($n$ = 24) and whether there existed gender differences in this respect. We used a single-target Implicit Association Test and an Implicit Association Test to measure dysfunctional automatic associations with social cues and implicit self-esteem, respectively. Results showed that automatic associations with social cues were more dysfunctional in socially anxious patients than in both control groups, suggesting this might be a specific characteristic of social anxiety disorder. Socially anxious patients showed relatively low implicit self-esteem compared to non-clinical controls, whereas panic disorder patients scored in between both groups. Unexpectedly, we found that lower implicit self-esteem was related to higher severity of social anxiety symptoms in men, whereas no such a relationship was found in women. These findings support the view that automatic negative associations with social cues and lowered implicit self-esteem may both help to enhance our understanding of the cognitive processes that underlie social anxiety disorder.

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## Introduction

Individuals who suffer from social anxiety disorder (SAD) typically experience an intense and persistent fear of social situations in which they are exposed to unfamiliar people or to the possible scrutiny by others (American Psychiatric Association, 2000). According to the cognitive model of social anxiety of Wells and Clark (1997), socially anxious individuals interpret social situations as threatening, because of negative beliefs about their selves and dysfunctional assumptions about their social performance; together with excessively high standards for social performance. Negative self-beliefs typically are unconditional negative statements about the self (e.g., "I'm stupid" or "I'm a failure"), whereas dysfunctional assumptions concerning social performance take the form of conditional beliefs about possible negative consequences of social behaviours (e.g., "if they see my anxiety, then they will think I'm a failure"). In support of the role of these dysfunctional cognitions in SAD, research showed that high socially anxious individuals indeed display more negative self-statements (e.g., Cacioppo, Glass & Merluzzi, 1979; Beidel, Turner & Dancu, 1985; Dodge, Hope, Heimberg & Becker, 1988) and lower levels of self-esteem in social situations (e.g., Bouvard et al., 1999; Tanner, Stopa & De Houwer, 2006) than low socially anxious individuals.

According to the multi-process model of anxiety, not only dysfunctional assumptions and negative self-beliefs (belonging to so-called 'rule-based processes') constitute the cognitive vulnerability to anxiety disorders, but also dysfunctional associative processes play an important role in the development and maintenance of anxiety disorders (Ouimet, Gawronski & Dozois, 2009). In response to anxiety-relevant stimuli, threat-related associations are thought to be directly activated via the spreading of activation from one concept to associated concepts in memory. Subsequently, the input from the associative system is assumed to be used for more explicit, rule-based mental processing (Strack & Deutsch, 2004) which involves the more rational analysis of factual relationships between concepts. Associative and rule-based information processing systems are thought to jointly influence other cognitive processes (e.g., negative interpretive bias) and behaviours (e.g., attention bias for threatening information or avoidance behaviours) that work together in a way to aggravate and/or maintain the anxiety disorder. Based on the model of Wells and Clark (1997) two types of automatic associations seem to be most important for SAD: first of all, negative automatic associations towards the self (i.e. implicit self-esteem) and secondly, associations between social cues and negative outcomes of social performance such as failure or rejection.

In line with the presumed role of automatic associations in anxiety, automatic as-

sociations were found to predict experimentally-provoked anxiety behaviours in unselected student samples in the laboratory (e.g., Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002; Spalding & Hardin, 1999). In addition, three studies focussing on the role of automatic self-anxious associations as generic vulnerability factor in anxiety disorders showed that automatic associations were indeed related to having an anxiety disorder diagnosis as well as to the maintenance and onset of anxiety disorders over time (Glashouwer & de Jong, 2010; Glashouwer, de Jong & Penninx, 2011, 2012). Furthermore, one study showed that socially anxious students were characterized by stronger automatic self-anxious associations than non-anxious students and that these associations seemed to reduce following treatment (Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008). Up to now, only a few studies looked specifically at implicit self-esteem and associations with social cues in SAD and, in addition, these studies mainly relied on analogue samples. Two studies showed that high socially anxious female students indeed were characterized by relatively low implicit self-esteem (de Jong, 2002; Tanner, Stopa & De Houwer, 2006). In addition, recently it was shown that for adolescent girls, but not boys, lower implicit self-esteem was related to more social anxiety symptoms (de Jong, Sportel, de Hullu & Nauta, 2012). Furthermore, there is some evidence supporting the view that also threat-related automatic associations with social cues may be involved in SAD. High socially anxious female students displayed stronger negative automatic associations with social cues than low-anxious participants (de Jong, Pasman, Kindt & van den Hout, 2001). Similar results were found in an adolescent sample, showing that in high socially anxious adolescents social cues automatically elicited relatively strong threat-related associations compared to low socially anxious adolescents (de Hullu, de Jong, Sportel & Nauta, 2012).

Although the available evidence suggests that both dysfunctional automatic associations with respect to the self and social cues seem to be involved in SAD, some important questions still remain unanswered. Prior studies in this field typically compared analogue groups of high socially anxious individuals with low socially anxious individuals. Since there may be quantitative as well as qualitative differences between analogue and clinical samples (Emmelkamp 1982), it seems important to replicate these findings in a clinical sample. In addition, prior studies did not include clinical-control groups, leaving open the question whether differences regarding automatic associations can indeed be attributed to SAD or have to be seen as more general characteristics shared among several anxiety disorders. Furthermore, most studies until now relied on female samples. However, there might be gender differences in the relationship between dysfunctional automatic associations and social anxiety symptoms. Earlier work showed that women are more likely

than men to base their judgments on intuitions and gut impressions (e.g., Pacini & Epstein, 1999). Perhaps this could mean that men typically discard automatic associations as largely irrelevant, whereas women tend to rely more on these automatic associations as a guideline for their behaviour and self-judgments (cf. Pelham et al., 2005). Consequently, automatic associations could have stronger predictive validity in women than in men and one study indeed showed findings in this direction in a group of adolescents (de Jong et al., 2011). Finally, prior studies usually focused on one kind of automatic associations, making it impossible to examine independent contributions of different types of automatic associations for social anxiety symptoms.

The main goal of the present study is to test whether SAD patients show stronger dysfunctional automatic associations (regarding self and social cues) than both clinical and non-clinical controls and whether there exist differences for females and males in this respect. Therefore, we included a clinical sample of treatment-seeking SAD patients, a non-anxious control group and a clinical sample of treatment-seeking panic disorder patients as a clinical control group. We hypothesize that automatic associations with social cues are more dysfunctional in the SAD group than in both control groups. In addition, we expect the SAD group to have a lower implicit self-esteem than controls. For the panic disorder group we have no clear expectations with respect to implicit self-esteem. Furthermore, we expect automatic associations to have stronger predictive validity for social anxiety symptoms in women than in men. Finally, an additional strength of the present study is that it is the first to examine two types of automatic associations in one socially anxious sample, allowing to test whether both types of associations are independently related to social anxiety symptom severity.

## Method

### *Participants*

Patients with social anxiety disorder (SAD) as primary diagnosis ($n$ = 45; 17 women) and patients with panic disorder (PD) as primary diagnosis and no SAD as comorbid disorder ($n$ = 24; 13 women) were recruited among individuals seeking treatment in various ambulant community health care centers in The Netherlands (GGZ Nijmegen: $n$ = 33; Hendriks & Roosenboom: $n$ = 19; GGZ Friesland: $n$ = 15; University Medical Centre Groningen: $n$ = 2). The mean age in the SAD group was 31.47 (SD = 10.57) and in the PD group 37.46 (SD = 14.03). Mean (and median) educational level was intermediate vocational education for both the SAD group and the PD group. All patients met DSM-IV criteria for SAD or PD respectively as assessed with Minnesota International Neuropsychiatric Interview

Plus (M.I.N.I.-Plus; Van Vliet, Leroy & Van Megen, 2000). In the SAD group 33 patients (73%) suffered one or more comorbid disorders, among which were depression or dysthymia (32%), panic disorder (13%) and generalized anxiety disorder (21%). In the PD group 11 patients (46%) suffered from one or more comorbid disorders, among which were depression or dysthymia (50%) and generalized anxiety disorder (22%).

Healthy control participants ($n$ = 45) were recruited through local advertisements and through indirect acquaintances of the staff members and students of the Department of Clinical Psychology of the University of Groningen and Radboud University Nijmegen. They were asked to serve as control participants in a study about anxiety. The non-clinical controls (NCC) matched with the SAD patients on gender, age and level of education and were included after screening on the absence of any DSM-IV axis-I disorder as measured by the M.I.N.I.-Plus, although one of the controls was diagnosed with alcohol dependence. Mean age was 31.16 years (SD = 11.60) and mean (and median) educational level was intermediate vocational education.

All participants included in the study had an estimated IQ of 90 or higher, good comprehension of the Dutch language, showed no signs of current psychosis and did not suffer from dyslexia. The number of participants in the groups differed, because the present report was part of a larger study with a longitudinal design in which we followed the SAD and NCC groups (but not the PD group) over time. Beforehand, we calculated that for group comparison by means of F-tests, power-analysis indicated that at least $n$ = 21 per group was needed, with $\alpha$ = .05 and power = .8 for a large effect (Cohen, 1988, Table 8.3.13). For correlational analyses per group, when looking for large effects, at least $n$ = 23 was needed with $\alpha$ = .05 and power = .8 (Cohen, 1988, Table 3.3.2). Because we expected a drop-out percentage of 50% at most, we decided to double the numbers of the SAD and NCC groups.

## *Materials*

**Minnesota International Neuropsychiatric Interview (M.I.N.I.; Sheenan et al., 1998):** The M.I.N.I. is a brief, structured, diagnostic interview designed to verify axis-I psychopathology according to the DSM. In the present study, a Dutch translation of the M.I.N.I.-Plus was used, adapted for the DSM-IV criteria (Van Vliet et al., 2000).

**Social anxiety measures.** To measure the level of social anxiety symptoms, a Dutch translation of the Social Phobia and Anxiety Inventory (SPAI: Turner, Beidel, Dancu, & Stanley, 1989; Dutch SPAI: Scholing, Bögels, & van Velzen, 1995) was used. The SPAI consists of 32 self-statements on experienced tension/anxiety in various social situations (Social

phobia subscale), and 13 self-statements on experienced tension/anxiety in various non-social situations (Agoraphobia subscale) which can be scored on a scale of 0 (never) to 7 (always). Since we included patients with SAD and patients with panic disorder, we decided to use the subscales separately. Psychometric properties for the Dutch SPAI are good (Bögels & Reith, 1999). Cronbach's Alpha was calculated over the 32 items of the social phobia subscale and the 13 items of the agoraphobia subscale of the SPAI. Both subscales showed excellent internal consistency (social phobia: $\alpha$ = .99; agoraphobia: $\alpha$ = .94). As an additional measure of social anxiety symptoms we used the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987; Oakman, Van Ameringen, Mancini & Farvolden, 2003). The LSAS consists of 24 social and performance situations for which individuals have to rate their fear and avoidance during the past week on a scale of 0 (none/never) to 3 (severe/usually). The LSAS was found to be a reliable, valid measure of social anxiety (Heimberg et al., 1999). Cronbach's Alpha was calculated over the 24 items of the fear subscale and the 24 items of the avoidance subscale of the LSAS. Both subscales showed excellent internal consistency (fear: $\alpha$ = .96; avoidance: $\alpha$ = .95).

**Social single target Implicit Association Test (social stIAT).**To assess the strength of automatic associations with social situations we used a single-target Implicit Association Test (stIAT) originally designed by Wigboldus, Holland, van Knippenberg, den Hartog and Belles (2002). The stIAT is a computerized reaction time task that is designed to measure to what extent a single target category is associated with two attribute categories. Following prior designs (Huijding & de Jong, 2006b; Karpinsky & Steinman, 2006; Wigboldus et al., 2002), a social stIAT was constructed with the target category *social situation* and the attribute categories *positive outcome* and *negative outcome*. Each category consisted of eight stimulus words. These were presentation, diner, party, phone call, chat, speech, meeting and dancing for *social situation*; successful, compliment, sympathy, succeed, friendly, approval, cosiness and fun for *positive outcome*; and disapproval, laugh at, unwanted, rejection, fail, flop, loser, and shame for *negative outcome* (translated from Dutch). The task consisted of four critical test blocks that were preceded by practice blocks (Table 4.3). In two test blocks *social situation* and *positive outcome* were mapped on one response key, and *negative outcome* on the other. In the other test blocks *social situation* and *negative outcome* were mapped on one key and *positive outcome* on the other. To prevent response bias, correct responses of the test blocks were divided equally over the two response keys (cf. Bluemke, Fiedler & Richter, 2009).

**Implicit self-esteem.** To assess implicit self-esteem we used the Implicit Association Test (IAT), a computerized reaction time task originally designed by Greenwald, McGhee

Table 4.3

*Arrangement of social stIAT*

| Block | Left | Right | Practice/Test |
|---|---|---|---|
| 1 | Social situation + Negative outcome | Positive | Practice |
| # Stimuli | 8 + 8 | 8 | |
| 2 | Social situation + Negative outcome | Positive | Test |
| # Stimuli | 16 + 16 | 32 | |
| 3 | Negative | Social situation + Positive | Practice |
| # Stimuli | 4 + 4 | 4 | |
| 4 | Negative | Social situation + Positive | Test |
| # Stimuli | 32 | 16 + 16 | |
| 5 | Social situation + Negative outcome | Positive | Practice |
| # Stimuli | 4 + 4 | 4 | |
| 6 | Social situation + Negative outcome | Positive | Test |
| # Stimuli | 16 + 16 | 32 | |
| 7 | Negative | Social situation + Positive | Practice |
| # Stimuli | 4 | 4 + 4 | |
| 8 | Negative | Social situation + Positive | Test |
| # Stimuli | 32 | 16 + 16 | |

& Schwartz (1998) to measure the relative strengths of automatic associations between two contrasted target categories and two attribute categories. Target categories were *me* and *not-me* and attribute categories were *positive* and *negative*. Each category consisted of six stimuli (cf. de Jong, 2002). These were I, me, mine, own, personal and myself, for *me*; they, their, other, you, another's, and their selves for *not-me*; unstable, bad, failure, passive, stupid, and worthless for *negative*; and stable, good, loved, active, smart and valuable for *positive*. The self-esteem IAT consisted of seven blocks with blocks 4 and 7 being the critical test blocks (see Table 4.4).

For both IAT tasks, stimulus words from all three (social stIAT) or four (self-esteem IAT) categories appeared in randomized order in the middle of a computer screen and participants were instructed to sort them with a left or right response key. The category labels stayed visible in upper left and right-hand corners of the screen during the whole task. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated. Before the start of a new sorting task, written instructions were presented on the screen. After a correct response, the next stimulus was presented with a 500 ms delay. After an

Table 4.4

*Arrangement of self-esteem IAT*

| **Block** | **Left** | **Right** | **Practice/Test** |
|---|---|---|---|
| 1 | Negative | Positive | Practice |
| # Stimuli | 6 | 6 | |
| 2 | Not-me | Me | Practice |
| # Stimuli | 6 | 6 | |
| 3 | Negative + Not-me | Positive + Me | Practice |
| # Stimuli | 12+12 | 12+12 | |
| 4 | Negative + Not-me | Positive + Me | Test |
| # Stimuli | 24+24 | 24+24 | |
| 5 | Me | Not-me | Practice |
| # Stimuli | 6 | 6 | |
| 6 | Negative + Not-me | Positive + Not-me | Practice |
| # Stimuli | 12+12 | 12+12 | |
| 7 | Negative + Not-me | Positive + Not-me | Test |
| # Stimuli | 24+24 | 24+24 | |

incorrect response, a red X (stIAT) or the word 'wrong' (IAT) appeared shortly above the stimulus, and the stimulus remained on the screen until the correct response was given. The order of category combinations within the tasks and order of both IATs was fixed across participants to reduce method variance.

## *Data reduction stIAT and IAT*

stIAT effects and IAT effects were computed according to the now widely used algorithm proposed by Greenwald, Nosek & Banaji (2003) that can also be used for analysing the stIAT (cf. Karpinsky & Steinman 2006; Thush & Wiers 2007). In this paper, we report the so-called $D_4$-measures. Following the algorithm, all reaction times above 10,000 ms were discarded. Error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred, plus a penalty of 600 ms. The stIAT effect was calculated by subtracting the mean reaction times of Block 4 from Block 2 and Block 8 from Block 6. These two difference scores were divided by the pooled standard deviations based on all responses of the particular blocks, after which the unweighted mean of both difference scores was calculated. Practice trials were discarded from the analyses, because we considered these trials to be truly practice: each stimulus was presented once to the participant and likewise the responses were not divided equally over the response keys.

Positive stIAT effects indicate faster responses when social situation shared the response key with positive outcome. The IAT effect was calculated by subtracting mean reaction times of Block 6 from Block 3 (practice) and Block 7 from Block 4 (test). These two differences scores were divided by their pooled standard deviation based on all responses of the particular blocks, after which the unweighted mean of both difference scores was calculated. Positive IAT effects indicate relatively fast responses when me shared the response key with positive. Prior to conducting the planned analyses, data were examined for outliers and excessive error rates following standard IAT analysis procedures (Greenwald et al., 1998). No data had to be omitted as a result of these checks. The split-half reliabilities of the social stIAT and self-esteem IAT were good, with Spearman-Brown corrected correlations between test-halves of .90 for the social stIAT ($D_4$-measure based on odd and even trials) and .89 for the self-esteem IAT ($D_4$-measure based on trials 1, 2, 5, 6, 9, 10 etc. vs. 3, 4, 7, 8, 11, 12 etc.).

### *Procedure*

Assessments lasted around 2 hours and were conducted on 1 day. Each participant completed the social stIAT, followed by the self-esteem IAT. After that, participants completed the questionnaires. Respondents were compensated with a €15,- gift certificate for the assessment and were debriefed afterwards. This report is part of a larger study into the role of cognitive processes in (the treatment of) SAD. Consequently, other measurements were collected as well during the assessments, but these are not of interest for the present study.

## Results

### *Descriptive statistics*

LSAS data was missing for one participant of the socially anxious group. Means and standard deviations for the different groups on the SPAI, the LSAS, the social stIAT, the self-esteem IAT and overall percentage of errors on stIAT/IAT are reported in Table 4.5. As expected, the socially anxious group and the control groups differed markedly on the social anxiety symptom measures during baseline.

### *Group differences on automatic associations*

**Social stIAT.** An overview of mean response times for the different groups for each pairing of the social stIAT is provided in Figure 4.2. A 3 Group (SAD, PD, NCC) x 2 Gender ANOVA on social stIAT showed a significant main effect for Group ($F(2,108) = 3.35$, $p < .05$,

Table 4.5

*Means and standard deviations of variables as a function of group*

| Variables | Group | | |
|---|---|---|---|
| | SAD | NCC | PD |
| SPAI, social phobia score | 125.13 (33.21) | 44.09 (26.03) | 82.47 (39.63) |
| SPAI, agoraphobia score | 27.57 (16.87) | 7.96 (9.00) | 38.96 (14.99) |
| LSAS, fear score | 40.41 (13.33) | 11.44 (7.17) | 23.00 (14.76) |
| LSAS, avoidance score | 31.55 (14.74) | 10.80 (6.74) | 19.38 (13.47) |
| Social stIAT, D-measure | .46 (.37) | .64 (.29) | .66 (.30) |
| Social stIAT, % error trials overall | 6.67 (4.88) | 7.01 (4.73) | 6.58 (3.62) |
| Self-esteem IAT, D-measure | .43 (.45) | .75 (.33) | .63 (.44) |
| Self-esteem IAT, % error trials overall | 7.02 (5.37) | 7.65 (5.79) | 8.30 (7.40) |

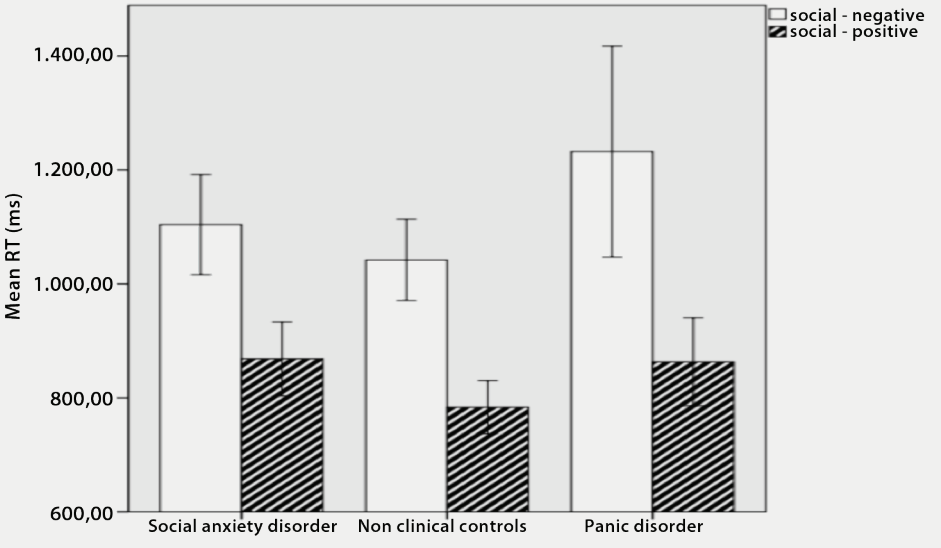
*Note*. stIAT = single target Implicit Association Test; IAT = Implicit Association Test; SPAI = Social Phobia and Anxiety Inventory; LSAS = Liebowitz Social Anxiety Scale; SAD = social anxiety disorder; NCC = non-clinical control; PD = panic disorder.

partial $\eta^2 = .06$). However, the main effect of Gender, and the interaction between Group and Gender were non-significant (*p's* > .4). As expected, planned comparisons with t-tests revealed that the socially anxious group displayed significantly less positive automatic associations with social situations (i.e., a relatively fast response when *social situation* and *negative outcome* shared the same response key) than both the non-anxious control group ($t(82.6) = 2.46$, $p < .05$, $d = .54$) and the panic disorder group ($t(67) = 2.17$, $p < .05$, $d = .53$). The control group and the panic disorder group did not significantly differ on the social stIAT ($t(67) = .26$, n.s.).

**Self-esteem IAT.** An overview of mean response times for the different groups for each pairing of the self-esteem IAT is provided in Figure 4.3. A 3 Group (SAD, PD, NCC) x 2 Gender ANOVA on self-esteem IAT showed a significant main effect for Group ($F(2,108) = 5.35$, $p < .01$, partial $\eta^2 = .09$). However, the main effect of Gender, and the interaction between Group and Gender were non-significant (*p's* > .3). Planned comparisons with t-tests revealed that the socially anxious group displayed significantly less positive implicit self-esteem (i.e., a relatively fast response when *me* and *negative* shared the same response key) than the control group ($t(80.6) = 3.85$, $p < .001$, $d = .86$). The difference between the socially anxious group and the panic disorder group only showed a non-significant trend ($t(67) = 1.74$, $p = .09$, $d = .43$). The control group and the panic disorder group did not significantly differ on implicit self-esteem ($t(67) = 1.30$, $p = .20$, $d = .32$).

Figure 4.2

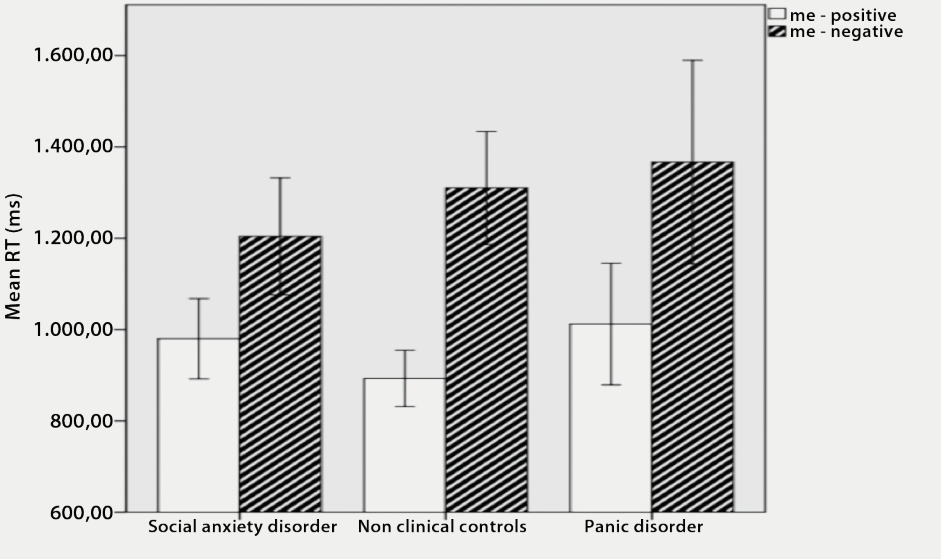
*Mean latencies of the trials per pairing of the social stIAT: social anxiety disorder, non-clinical controls and panic disorder*




*Note*. RT = reaction time; stIAT = single target Implicit Association Test; stIAT latencies include 600 ms penalty for wrong responses; error bars: +/- 2 * SE.

Figure 4.3

*Mean latencies of the trials per pairing of the self-esteem IAT: social anxiety disorder, non-clinical controls and panic disorder*




*Note*. RT = reaction time; IAT = Implicit Association Test; IAT latencies include 600 ms penalty for wrong responses; error bars: +/- 2 * SE.

### *Relationship between automatic associations and social anxiety symptoms (as a function of gender)*

Because the subscales of the LSAS were somewhat skewed to the right, we first performed a square root transformation on both subscales. Correlation coefficients were calculated between stIAT, IAT and the self-report measures of anxiety symptoms (see Table 4.6). Next, we performed forced entry hierarchical regression analyses to explore whether having dysfunctional automatic associations on both the social stIAT and the self-esteem IAT independently contributed to the level of social anxiety symptoms. In addition, we included interaction effects with gender expecting that the regression model would be more sensitive to possible gender differences. Social anxiety symptoms (SPAI) were included as dependent variable and standardized social stIAT, self-esteem IAT and gender as independent variables in the first three steps respectively. In the fourth step, the interactions between standardized social stIAT and self-esteem IAT and the interactions with gender were added to the model (see Table 4.7, upper part). Results show there was only a main effect of self-esteem IAT on social anxiety symptoms, indicating that lower implicit self-esteem was related to higher social anxiety symptom severity. Main effects of social stIAT and gender were non-significant. In addition, we found that the interaction between self-esteem IAT and gender showed predictive validity for severity of symptoms, although the $\Delta R^2$ of step 4 did not reach significance. The interactions between social stIAT and self-esteem IAT and between social stIAT and gender were non-significant.

Because we assumed that the number of parameters in the regression model could have reduced the power of the regression model, we decided to repeat the analysis with only the significant predictors: implicit self-esteem, gender, and the interaction term of implicit self-esteem and gender (see Table 4.7, lower part). Results showed that now the $\Delta R^2$ of adding the interaction effect did reach significance. To interpret these findings, the predicted values for the interaction term are presented in Figure 4.4. Unexpectedly, the figure indicates that lower implicit self-esteem was related to higher social anxiety symptom severity in men, whereas no such a relationship was found in women.

Table 4.6

*Correlation matrix stIAT, IAT, SPAI and LSAS (N =114)*

| Measure | 2. | 3. | 4. | 5. | 6. |
|---|---|---|---|---|---|
| 1. Social stIAT, D-measure | .02 | -.04 | .01 | -.20* | -.08 |
| 2. Self-esteem IAT, D-measure | - | -.38** | -.21* | -.35** | -.32** |
| 3. SPAI, social phobia score | | - | .65** | .94** | .85** |
| 4. SPAI, agoraphobia score | | | - | .58** | .55** |
| 5. LSAS, fear score | | | | - | .88** |
| 6. LSAS, avoidance score | | | | | - |

*Note*. stIAT = single target Implicit Association Test; IAT = Implicit Association Test; SPAI = Social Phobia and Anxiety Inventory; LSAS = Liebowitz Social Anxiety Scale. * $p$ <.05; ** $p$ <.01. Please note that the negative correlations between automatic associations and symptom measures are in line with our expectations, since positive effects for self-associations indicate a relatively stronger automatic association between *me/ social situation* and *positive*.

Table 4.7

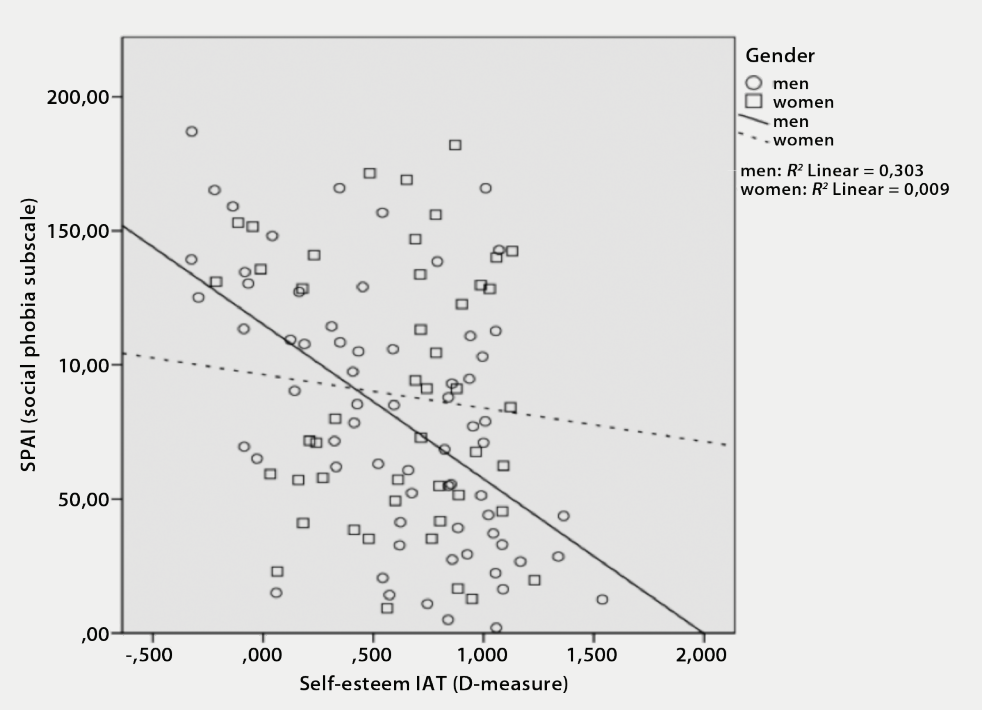
*Summary of hierarchical regression analysis for variables predicting social anxiety measured with the social phobia subscale of the Social Phobia and Anxiety Inventory (SPAI; N =114)*

| Model | Step | Predictor | *B* | SE *B* | *β* | *p* |
|---|---|---|---|---|---|---|
| All predictors included [a] | 1 | social stIAT | -2.11 | 4.55 | -.04 | .644 |
| | 2 | social stIAT | -1.84 | 4.23 | -.04 | .665 |
| | | self-esteem IAT | -18.13 | 4.23 | -.38 | <.001 |
| | 3 | social stIAT | -2.18 | 4.24 | -.05 | .608 |
| | | self-esteem IAT | -18.20 | 4.23 | -.38 | <.001 |
| | | gender | 4.37 | 4.25 | .09 | .306 |
| | 4 | social stIAT | -1.67 | 4.26 | -.05 | .679 |
| | | self-esteem IAT | -16.78 | 4.30 | -.38 | <.001 |
| | | gender | 4.18 | 4.23 | .09 | .326 |
| | | stIAT x IAT interaction | -1.41 | 4.34 | -.03 | .746 |
| | | IAT x gender interaction | 9.39 | 4.45 | .19 | .037 |
| | | stIAT x gender interaction | -1.13 | 4.39 | -.02 | .797 |
| Only significant | 1 | self-esteem IAT | -18.23 | 4.22 | -.38 | <.001 |
| predictors included[b] | | gender | 4.19 | 4.22 | .09 | .322 |
| | 2 | self-esteem IAT | -16.59 | 4.22 | -.34 | <.001 |
| | | gender | 4.11 | 4.15 | .09 | .324 |
| | | IAT x gender interaction | 9.49 | 4.37 | .19 | .032 |

*Note*. stIAT = single target Implicit Association Test; IAT = Implicit Association Test. [a] Step 1: $R^2$ = .002, n.s.; Step 2: Δ $R^2$ = .14, $p$ < .001; Step 3: Δ $R^2$ = .008, n.s.; Step 4: Δ $R^2$ = .035, n.s. [b] Step 1: $R^2$ = .15, $p$ < .001; Step 2: Δ $R^2$ = .035, $p$ = .032.

Figure 4.4

*Interaction effect of implicit self-esteem on symptoms of social anxiety as indexed by the Social Phobia and Anxiety Inventory (SPAI) for men (n = 67) and women (n = 47)*




## Discussion

The present study represents the first design in which dysfunctional automatic associations are studied in a treatment-seeking community sample of SAD patients including both a non-clinical control group and a clinical control group of panic disorder patients. As hypothesized, the results showed that automatic associations with social cues were more dysfunctional in socially anxious patients than in both control groups. In addition, socially anxious patients showed relatively low implicit self-esteem compared to non-clinical controls, whereas panic disorder patients scored in between both groups. Furthermore, implicit self-esteem was shown to be a better independent predictor of anxiety symptoms than automatic associations with social cues. Unexpectedly, we found that lower implicit self-esteem was related to higher severity of social anxiety symptoms in men, whereas no such a relationship was found in women. Finally, implicit self-esteem and automatic associations with social cues were not significantly related to each other and exploratory analyses indicated that having both kinds of dysfunctional associations was not related to heightened symptom severity.

Dysfunctional associative processes are thought to play an important role in the cognitive vulnerability of anxiety disorders (e.g., Ouimet et al., 2009). According to the cognitive model of Wells and Clark (1997), socially anxious individuals interpret social situations as threatening, because they hold negative beliefs about their selves and dysfunctional assumptions about their social performance. Consequently, especially negative automatic associations towards the self and negative associations with social cues seem to be important in SAD. In line with this theoretical starting point, the present study showed that socially anxious individuals were indeed characterized by lowered implicit self-esteem and more negative automatic associations with social cues than non-clinical controls. These outcomes are in line with prior studies that already showed similar differences between analogue groups of high and low socially anxious individuals (de Jong, 2002; de Jong et al., 2001; de Hullu et al., 2011; Tanner et al., 2006). In addition, we also included a clinical control group of panic disorder patients in the present design. Results indicated that dysfunctional automatic associations with social cues seem to be specific for SAD, because panic disorder patients scored comparable to non-clinical controls on this type of associations. Results were somewhat different for implicit self-esteem on which the panic disorder group scored in between patients with social anxiety disorder and non-clinical controls. The latter might indicate that lowered implicit self-esteem can be seen as a more general characteristic of anxiety patients, but at the same time is particularly involved in SAD. Maybe individuals who suffer from an anxiety disorder generally have a relatively low implicit self-esteem, reflecting a vulnerability to experience anxiety over a variety of situations. This would also be in line with the significant relationship that was found between implicit self-esteem and agoraphobia symptoms (SPAI, agoraphobia subscale). Since concerns about the self are a central theme in SAD (Wells & Clark, 1997), it is not surprising that these individuals score even lower on implicit self-esteem. However, it is important to keep in mind that the differences on implicit self-esteem between panic disorder patients and SAD patients (Cohen's $d = .43$) nor between panic disorder patients and non-anxious controls (Cohen's $d = .32$) reached significance. Consequently, the present findings should be replicated before we can draw final conclusions in this respect.

Implicit self-esteem and automatic associations with social cues were not significantly related to each other and exploratory analyses indicated that having both kinds of dysfunctional associations was not related to heightened symptom severity. In addition, implicit self-esteem was the best predictor of social anxiety symptoms, whereas automatic associations with social cues were only related to one of the three indices of social anxiety symptoms (i.e. the fear subscale of the LSAS). However, the magnitude of the latter effect (*r*

= -.20) was similar to a prior study that demonstrated the relationship between automatic associations with social cues and social anxiety symptom severity in adolescents ($\beta$ = -.20; de Hullu et al., 2011). Perhaps these findings point out that we are looking at really different kinds of automatic associations that both play their different roles in SAD. Furthermore, these outcomes might be better understood using dual-process models in which it is postulated that automatic associations primarily influence spontaneous behavioural responses, whereas explicit cognitions guide more controlled behaviours (e.g., Gawronski & Bodenhausen, 2006). It seems plausible that the self-report measures that we used to assess social anxiety symptoms did not effectively capture these relatively spontaneous anxiety behaviours. Maybe automatic associations with social cues are especially important for these direct, spontaneous anxiety reactions in specific social situations, whereas implicit self-esteem might be more related to more global social anxiety symptoms that appear over several situations which were probably captured with the self-report measures that we used. This would explain why the stIAT did not consistently relate to all self-report measures of social anxiety in the present study, whereas the self-esteem IAT did.

A subsidiary goal of the present study was to see whether there are gender differences in the relationship between dysfunctional automatic associations and social anxiety symptoms. When we included gender as a factor in the between-group analyses, we did not find interactions between group and gender on automatic associations. However, when we predicted the strength of anxiety symptoms, we did find a significant interaction between implicit self-esteem and gender. We found that lower implicit self-esteem was related to higher severity of social anxiety symptoms in men, whereas no such a relationship was found in women. This is an unexpected finding, since a prior study that tested the effect of gender in an adolescent group, actually showed opposite findings (de Jong et al., 2011). Here, having lower implicit self-esteem was related to more social anxiety symptoms in adolescent girls, but not in adolescent boys. Furthermore, our findings are in contrast with our hypothesis that automatic associations would have stronger predictive validity in women than in men based on prior work showing that women are more likely than men to base their judgments on intuitions and gut impressions (e.g., Pacini & Epstein, 1999). How should we interpret these findings? Specific sample selection might have contributed to the differences between our findings and the findings of de Jong and colleagues (2011). De Jong and colleagues studied a non-clinical sample of adolescents, whereas we used a clinical sample of adult SAD patients. Possibly, the different findings reflect actual differences in social anxiety between adolescents and adults and/or between non-clinical individuals and patients. Further research will have to elucidate

whether these findings can be replicated suggesting that implicit self-esteem is indeed differentially involved in SAD, or whether the differences that were found between men and women are the result of the specific samples that were selected. Until this issue is clarified, it seems important to pay careful attention to possible gender differences that might exist in underlying cognitive processes of SAD.

It should be acknowledged that we did not include measures of more spontaneous social anxiety behaviours. As discussed, the self-report measures that we used to assess social anxiety symptoms probably might not have effectively captured these relatively spontaneous behaviours. Another disadvantage of this study is that we did not include 'rule-based' equivalents of the automatic associations that rely on more explicit cognitive reasoning. This does not make it possible to investigate whether the present relationships that were found between dysfunctional automatic associations and social anxiety were at least partially due to an overlap with more deliberate, explicit beliefs, nor whether interactions between implicit and explicit associations contribute to social anxiety symptoms (cf. de Jong et al., 2011). Expanding future study designs with other measures of social anxiety as well as explicit equivalents of automatic associations will help to fully understand the possible differential pathways of dysfunctional automatic and/or explicit associations in guiding spontaneous and more controlled symptoms of SAD.

### *Conclusions*

The present outcomes suggest that both dysfunctional automatic associations with social cues as well as lowered implicit self-esteem are related to SAD. Dysfunctional automatic associations with social cues appear to be a specific characteristic of SAD. Lowered implicit self-esteem seems to be a more general characteristic of anxiety disorders that is particularly involved in SAD. For future studies it would be important to further test possible gender differences in the relationship between automatic associations and social anxiety symptoms as well as possible interactions with dysfunctional explicit reasoning. In addition, a crucial next step would be to examine whether experimentally reducing dysfunctional automatic associations (e.g., via classical conditioning procedures; Clerkin & Teachman, 2010), has beneficial effects on symptoms. If so, this would not only elucidate the exact nature of the relationship between automatic associations and SAD, it could also help to improve further interventions for SAD.

# 5 Dysfunctional automatic self-associations over time

# Study 1: Maintenance of anxiety and depression

## Abstract

Dysfunctional self-beliefs are assumed to play an important role in maintaining depression and anxiety. Current dual-process models emphasize the relevance of differentiating between automatic and explicit self-beliefs. Therefore, this study tested the prognostic value of automatic and explicit self-associations for the naturalistic course of depressive and anxiety disorders over two years follow-up. Both self-depressed and self-anxious associations were measured in depressed patients ($n$ = 313), anxious patients ($n$ = 566), and patients with comorbid depressive and anxiety disorders ($n$ = 577) as part of the Netherlands Study of Depression and Anxiety. Outcomes showed that in single predictor models specifically automatic self-anxious associations were related to a reduced chance of remission from anxiety, whereas automatic self-depressed associations were related to a reduced chance of remission from depression. Explicit self-anxious associations and fearful avoidance behaviour showed independent predictive validity for remission from *anxiety*, whereas explicit self-depressed associations and having a double depression showed independent predictive validity for remission from *depression*. These findings are not only consistent with the view that both automatic and explicit dysfunctional self-associations are related to the course of anxiety and depressive disorders, but also suggest that both types of self-beliefs are proper targets for therapeutic interventions.

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## Introduction

Depressive and anxiety disorders represent major problems for public health (e.g., Ormel et al., 2008). An important reason for their high disease burden is that both disorders often display a chronic-intermittent course. Cognitive theories emphasize the role of individuals' maladaptive self-cognitions in the origin and persistence of anxiety and depression (e.g., Beck, Rush, Shaw, & Emery, 1979; Rapee & Heimberg, 1997). Following this, relatively strong negative self-cognitions may make people vulnerable for developing unfavourable course trajectories of depressive and anxiety disorders.

Recent dual-process models (e.g., Gawronski & Bodenhausen, 2006) stress the importance of distinguishing between more explicit self-beliefs and more automatically activated self-associations. Automatic self-associations are assumed to be simple links between self and associated concepts in memory, which can be activated directly in response to relevant stimuli, and are thought to influence more spontaneous behavioural responses towards stimuli (e.g., Huijding & de Jong, 2006). Subsequently, input from the associative system is assumed to be used for more explicit processing (Strack & Deutsch, 2004) where propositions are weighted according to their 'truth' values. These explicit cognitions are thought to guide more controlled behaviours. Considering that anxiety and depressive symptoms include spontaneous as well as more controlled behaviours, both types of self-associations might play a role in the maintenance of anxiety and depression.

There is already considerable evidence indicating that anxious individuals are characterized by relatively strong dysfunctional automatic self-associations (for a review see Roefs et al., 2011). Also more generic automatic self-anxious associations were shown to be specifically enhanced in individuals with anxiety disorders (Glashouwer & de Jong, 2010). Furthermore, there is empirical support for the alleged importance of dysfunctional automatic associations in the persistence of anxiety symptoms. It was shown that stronger automatic catastrophic associations in panic patients were associated with a smaller reduction in anxiety sensitivity following treatment (Schneider & Schulte, 2008), and changes in automatic panic-associations over the course of treatment were correlated with greater symptom reduction (Teachman, Marker & Smith-Janik, 2008). Finally, automatic self-anxious associations were found to predict experimentally-elicited spontaneous anxious behaviours (e.g., Egloff & Schmukle, 2002).

Although earlier results seem to be promising, these studies were conducted over a relatively short time period, and often in the context of treatment, which still leaves undecided whether dysfunctional automatic self-associations are also involved in the naturalistic course of anxiety symptoms over a longer period of time. Furthermore, thus far,

studies testing the role of automatic self-associations typically looked at anxiety-relevant self-associations in relation to anxious behaviours and symptoms. It remains, therefore, to be tested whether automatic self-anxious associations are specifically important for the persistence of anxiety symptoms, or whether they should be considered as more general characteristics that are involved in other psychological disorders as well. Therefore, the present study was designed to investigate further the role of dysfunctional automatic associations in the persistence of anxiety disorders. More specifically, we examined the prognostic value of automatic self-anxious associations for the course of anxiety symptoms over a two-year period and tested the specificity of this relationship by including a clinical control group of depressed individuals.

Although there is already supportive evidence that automatic self-anxious associations are involved in anxiety (e.g., Egloff & Schmukle, 2002; Glashouwer & de Jong, 2010, Glashouwer, de Jong & Penninx, 2011), results regarding automatic self-associations in depression are more mixed. Thus far, most studies focused on global affective self-associations (so-called implicit self-esteem), but did not show consistent differences in implicit self-esteem between depressed and healthy individuals (e.g., De Raedt, Schacht, Franck & De Houwer, 2006; Franck, De Raedt & De Houwer, 2007, 2008; Gemar, Segal, Sagrati & Kennedy, 2001; Valiente et al., 2011). However, there is some evidence indicating that relatively negative automatic self-associations predict depressive behaviour both on short term (Haeffel et al., 2007: study 1) and long term (Franck, De Raedt & De Houwer, 2007; Haeffel et al., 2007: study 2). Yet, analogous to self-anxious associations in anxiety disorders, more disorder-specific self-depressed associations may be especially relevant for guiding relatively spontaneous depressed behaviours, thoughts and feelings, and may thus also be especially relevant for explaining the persistence of these symptoms.

In line with the alleged role of self-depressed associations in depression, previous research already demonstrated that automatic self-depressed associations were enhanced in patients with a major depressive disorder both compared to healthy controls and individuals with an anxiety disorder (Glashouwer & de Jong, 2010). As a logical next step, the second goal of this study was to investigate whether the strength of these automatic self-depressed associations also has prognostic value for the course of depressive symptoms. Anxious individuals served as clinical control group to test the specificity of this relationship.

In summary, although there is already some evidence that automatic self-associations may be involved in both anxiety and depression, little is known about their role in the natural course of depressive and anxiety disorders over a longer period of time.

Furthermore, it is yet unclear whether potential effects of automatic self-associations are disorder-specific or 'trans diagnostic' across anxiety and depression. The current study is the first to examine the prognostic value of automatic self-associations for the natural course of depressive and anxiety disorders. Automatic self-anxious and self-depressed associations were assessed in a large cohort of depressed and anxious individuals, and we tested whether the strengths of these self-associations were predictive for the course of symptoms over two years. As explicit equivalents of automatic self-associations, also self-report measures of self-anxious and self-depressed associations were included. Although previous studies provided evidence indicating that self-reported negative self-views were related to anxious and depressive symptoms and/or behaviours (e.g., Acarturk et al., 2009; Batelaan et al., 2010; Hirsch, Clark, Mathews & Williams, 2003; Ingram, Miranda & Segal, 1998; Tanner, Stopa & De Houwer, 2006), no research exists yet into more specific anxious or depressed self-concepts. We expected that in individuals with anxiety and/or depressive disorders stronger dysfunctional self-associations would be related to relatively unfavourable courses of the disorders.

## Method

### *Study sample*

The present study was carried out in the context of the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008), a multi-center, ongoing cohort study, designed to examine the long-term course and consequences of anxiety and depressive disorders. A total of 2981 persons aged 18 through 65 were included, including healthy controls, individuals at risk because of prior episodes, sub threshold symptoms or family history, and individuals with a current first or recurrent depressive and/or anxiety disorder. The inclusion was restricted to major depressive disorder, dysthymia, general anxiety disorder, panic disorder, social anxiety disorder, and agoraphobia, because these disorders are relatively homogenous in phenotype and are found across different health care settings. Recruitment of respondents took place in the general population, in general practices, and in mental health care institutions. General exclusion criteria were a primary clinical diagnosis of a psychiatric disorder not subject of NESDA which would largely affect course trajectory (i.e., psychotic disorder or bipolar disorder) and not being fluent in Dutch. The present study concerns baseline and 2-year follow-up measurements conducted from September 2004 till April 2009. The study protocol was approved centrally by the Ethical Review Board of VU Medical Center Amsterdam and subsequently by local review boards of each participating center/institute, and all participants provided written informed consent.

After two years, a face-to-face follow-up assessment was conducted with a response of 87.1% ($N = 2596$). Non-response was significantly higher among those with younger age, lower education, non-European ancestry, and depressive disorder, but was not associated with gender or anxiety disorder (Lamers et al., 2011). The presence of depressive and anxiety disorders was established with the Composite International Diagnostic Interview (CIDI; WHO version 2.1) which classifies diagnoses according to DSM-IV criteria (American Psychiatric Association, 2000). For the present analysis, only individuals who were symptomatic during baseline were included. Consequently, the sample was restricted to 1456 participants with a depressive and/or anxiety disorder: 313 with pure depressive disorder, 566 with pure anxiety disorder and 577 with comorbid depressive and anxiety disorders at baseline. Of all depressed individuals 28.2% had both major depressive disorder and dysthymia. Of all anxious individuals 45.1% had more than one anxiety disorder during baseline.

### *Course of depressive and anxiety disorders*

Course was determined using the CIDI interview and the Life Chart Interview (LCI) collected during the 2-year follow-up assessment. The CIDI interview determined presence of DSM-IV classified depressive and anxiety disorders between baseline and 2-year follow-up. For all persons with detected depressive or anxiety symptoms in the CIDI interview, the LCI was completed. Using a calendar method, life events were recalled to refresh memory after which presence of depressive and anxiety symptoms –separately– at each month during this 2-year period was determined (Lyketsos, Nestadt, Cwi & Heithoff, 1994). In addition, for each month with reported symptoms, severity was assessed and symptoms were considered present when at least of mild severity.

As reported in prior work (Penninx et al., 2011), the following course indicators were created: (Time to) remission of anxiety/depressive disorder was defined based on LCI as occurrence and the number of months till the first time-point at which no anxiety/depressive symptoms were reported for three consecutive months (Frank, Prien, Jarrett & Keller, 2002). When individuals had more than one anxiety disorder during baseline, they were considered remitted when no symptoms of any of the anxiety disorders existed for 3 consecutive months. No distinction was made between remission and recovery (Frank et al., 1991) because data did not allow for such precision.

## *Measures*

**Automatic self-associations.** The Implicit Association Test (IAT) is a computerized reaction time task originally designed by Greenwald, McGhee and Schwartz (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left or right response key. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated. For both IATs target labels were *me* and *others*. Following the design of Egloff and Schmukle (2002), an anxiety IAT was constructed with attribute labels *anxious* and *calm*. Analogously, attribute labels were *depressed* and *elated* for the depression IAT. Each category consisted of five stimuli (see Chapter 2, Appendix 2.A). Attribute stimuli of the anxiety IAT were the same self-descriptors as used by Egloff and Schmukle who based their IAT on trait anxiety. Furthermore, we designed a self-depressed IAT in an equivalent way and selected trait self-descriptors of depressed persons that were also used in previous work on attentional bias in (remitted) depression (e.g., McCabe, Gotlib & Martin, 2000). Both IATs consisted of two critical test blocks that were preceded by practice blocks (see Chapter 2, Table 2.1). The order of category combinations was fixed across participants to reduce method variance. The IAT already showed predictive validity for various outcome measures in a wide range of subjects (e.g., Greenwald, Poehlman, Uhlmann & Banaji, 2009) and was found to be one of the best implicit measures in terms of psychometric properties (e.g., Bosson, Swann & Pennebaker, 2000).

**Explicit self-associations.** To obtain explicit self-associations equivalently to the automatic self-associations, participants rated all IAT attribute stimuli on a 5-point scale (1 = hardly/not at all, 5 = very much) (i.e., "For each word please indicate to what extent you think it generally applies to you.").

**Questionnaire data.** Severity of anxiety symptoms at baseline was measured with the 21-item Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988), whereas fearful avoidance behaviour was measured using the 15-item Fear Questionnaire (FQ; Marks & Mathews, 1979). Severity of depressive symptoms was measured with the 30-item Inventory of Depressive Symptoms self-report version (IDS-SR; Rush, Gullion, Basco & Jarrett, 1996). Total scale scores were used for all questionnaires.

## *Procedure*

Baseline and follow-up assessments were similar, lasted between 3 and 5 hours and were

conducted on 1 day. During assessments, other measurements were collected as well, but these are not of interest for the present study (for a detailed description, see Penninx et al., 2008). Each participant completed the anxiety IAT, followed by the depression IAT. After that, participants deliberately rated attribute words that were used in the IATs. Respondents were compensated with a €15, - gift certificate and travel expenses.

## *Data analyses*

**Data reduction.** IAT scores were computed according to the now widely used algorithm proposed by Greenwald, Nosek and Banaji (2003). We report the $D_4$-measure.[9] Reaction times above 10,000 ms were discarded and error trials were replaced with mean reaction times of correct responses in the block in which the error occurred plus a penalty of 600 ms. For the anxiety IAT, the IAT-effect was calculated by subtracting mean reaction times of Block 6 from Block 3 (practice) and Block 7 from Block 4 (test; see Chapter 2, Table 2.1). The means of these two effects were divided by their pooled standard deviation based on all responses in Blocks 3, 4, 6, and 7. Analogously, the IAT-effect was calculated for the depression IAT based on Blocks 9, 10, 12, and 13. Positive IAT-effects indicate relatively fast responses when *me* shared the response key with either *calm* or *elated*.[10] Split-half reliabilities of the present IATs were good, with Spearman-Brown corrected correlations between test-halves of .87 for the depression IAT and .92 for the anxiety IAT.
To compute explicit association effects, mean ratings of anxious (depressed) IAT-stimuli were subtracted from mean ratings of calm (elated) IAT-stimuli. Hence, positive effects indicate relatively strong explicit associations between me and calm (or me and elated). Internal consistencies of explicit self-association measures were excellent (self-anxious: $\alpha$ = .94; self-depressed: $\alpha$ = 95).

**Missing data and construction of groups.** Of the 1456 participants measured at baseline 1209 (83.0%) participated in the 2-year follow-up (median in-between time = 24 months). Due to technical problems, IAT data and explicit self-associations for 48 par-

[9] The $D_4$-measure was chosen out of six variants of the D-measure (Greenwald et al., 2003). Two variants use so-called 'built-in' error penalties; this means that error latencies were included of the correct responses participants made after any error. However, our task version of the IAT did not record the latencies of these second responses; therefore we could not use the $D_1$-measure or the $D_2$-measure. The four D-measures that are left ($D_3$, $D_4$, $D_5$, $D_6$) are very similar. Two measures exclude trials below 400 ms ($D_5$ and $D_6$). However, such an exclusion of trials does not improve the performance to a great extent and therefore we decided to include as much trials/information as possible. Consequently, there remained two options, one measure adding 2 SD to the block mean as error penalty ($D_3$) and another adding 600 ms as error penalty ($D_4$). We decided to use the last one, because we preferred a constant error penalty, instead of a variable one, keeping the data as understandable as possible.

[10] Please note that this scoring procedure is reversed compared to three prior NESDA studies in which the IAT was used (Glashouwer & de Jong, 2010; Glashouwer et al., 2010; van Harmelen et al., 2010). Originally we decided to 'reverse' the D-measure to make it comparable to symptom measures in which higher scores also indicate less favourable outcomes. However, in a recent paper (Glashouwer, de Jong & Penninx, 2011) we decided to change the multiplication sign before the D-measure make it comparable to the general literature.

ticipants were missing. In addition, 22 individuals had missing data on the IDS-SR, 20 on the BAI and 20 on the FQ. Finally, on the criterion variables there were some additional missing values (7 on remission depression; 10 on remission anxiety). Missing data were estimated using multiple imputation. Multiple imputation is one of the state-of-the-art and preferred methods for dealing with missing data (Schafer & Graham, 2002). Missing data was imputed 40 times using PASW Statistics 18.0, based on all predictors that were included in the model. In addition, we included age, gender, education and baseline psychiatric status as predictors in the multiple imputation model, because some of these variables were shown to be associated with drop-out (Lamers et al., 2011). Finally, 8 participants were excluded because more than 10% of the IAT trials were below 300 ms (Greenwald et al., 2003), suggesting that they were trying to respond too rapidly, and 2 participants because of unusual D-scores (> 4 SD divergent from mean). The final sample existed of 1446 participants.

To keep results as straightforward as possible, we conducted analyses concerning course of anxiety in the sample of all individuals with an anxiety disorder during baseline ($n$ = 1136; pure anxiety disorder and comorbid anxiety/depression). Similarly, analyses concerning course of depression were conducted among all individuals with a depressive disorder during baseline ($n$ = 882; pure depressive disorder and comorbid anxiety/depression).

**Statistical analyses.** First, bivariate Spearman correlations were calculated between the continuous predictors, because the BAI and FQ were somewhat skewed to the right. Subsequently, associations between predictors and 2-year course were examined using Cox's proportional hazards analyses. Dependent variables were time till remission (in months) and remission (yes/no). Subjects with duration greater than 24 months were censored at 24 months. Predictors included in the model were anxiety IAT, depression IAT, explicit self-anxious and self-depressed beliefs. In addition, we explored whether effects of self-associations were especially strong for individuals with both explicit and automatic dysfunctional self-associations (e.g., de Jong, Sportel, de Hullu & Nauta, 2012). Therefore, we included the interactions between explicit and automatic self-associations in the models. Furthermore, symptom measures were included as covariates in the statistical models: BAI and FQ for course of anxiety and IDS-SR for course of depression. Moreover, categorical covariates were added to investigate the influence of comorbidity between anxiety and depression and comorbidity within disorders (i.e., having more than one anxiety disorder or having double depression). Finally, for exploratory reasons, we were interested to see whether automatic associations interacted with comorbidity in predicting

course, i.e. whether automatic associations had a stronger influence on course of anxiety and depression for comorbid patients. Some information-processing models postulate that individuals are more likely to act on their automatic associations, when their working memory capacity is occupied (e.g., Fazio & Towles-Schwen, 1999). Prior observations demonstrated that comorbid patients report relatively severe symptoms (e.g., Bruce et al., 2005; Hecht, von Zerssen & Wittchen, 1990; Roy-Byrne et al., 2000). Since higher symptom severity might form a greater load for working memory, we hypothesized a stronger influence of automatic associations for comorbid patients on the course of their symptoms.

## Results

### *Descriptives*

Means and standard deviations of demographics, automatic measures and self-report measures at baseline for the different groups are reported in Table 5.1. Correlations between predictors are shown in Table 5.2.

### *Are automatic self-associations at t0 predictive for the course of anxiety between t0 and t2?*

Single predictor Cox's survival analyses showed that when individuals associated themselves relatively strongly with anxious (anxiety IAT), they had a significantly lower chance for remission, indicating a longer episode duration of the anxiety disorder (Table 5.3). No such an effect was found for automatic self-depressed associations. Additional indicators associated with lower risk for first remission were stronger explicit self-anxious and explicit self-depressed associations, higher severity of anxiety symptoms (BAI), fearful avoidance behaviour (FQ), and having more than one anxiety disorder. When all predictors were simultaneously entered into the model, automatic self-anxious associations no longer showed independent predictive validity. Only stronger explicit self-anxious associations and more fearful avoidance behaviours were found to be independently associated with a decreased chance of remitting from anxiety.

### *Are automatic self-associations at t0 predictive for the course of depression between t0 and t2?*

Single predictor Cox's survival analyses showed that when individuals associated themselves relatively strong with depressed (depression IAT), they had a significantly lower chance for remission, indicating longer episode duration of the depressive disorder (Table 5.4). No such an effect was found for automatic self-anxious associations. Additional indi-

Table 5.1

*Means and standard deviations of demographics, course variables, automatic measures and self-report measures at baseline as a function of group*

| Measure | Pure depression *n* = 310 | Pure anxiety *n* = 564 | Comorbid depression-anxiety *n* = 572 |
|---|---|---|---|
| Gender, % female | 64.2 | 68.1 | 66.6 |
| Age | 41.90 (12.36) | 41.91 (12.68) | 41.59 (11.78) |
| Educational level in years | 11.89 (3.27) | 12.17 (3.22) | 11.03 (3.21) |
| % Remission anxiety | - | 58.1 | 51.4 |
| % Remission depression | 75.0 | - | 69.8 |
| Time to remission anxiety (months) | - | 14.24 (8.93) | 15.13 (8.75) |
| Time to remission depression (months) | 12.28 (8.15) | - | 13.72 (8.02) |
| IAT anxiety, D-measure[a] | .27 (.49) | .13 (.53) | .07 (.52) |
| Mean error rate IAT anxiety | 6.10 (6.62) | 5.77 (5.73) | 6.40 (5.94) |
| IAT depression, D-measure[a] | .13 (.40) | .20 (.40) | .05 (.44) |
| Mean error rate IAT depression | 5.73 (5.80) | 5.18 (4.66) | 5.95 (5.59) |
| Explicit self-anxious[a] | .15 (1.35) | -.19 (1.36) | -1.08 (1.30) |
| Explicit self-depressed[a] | .33 (1.57) | 1.15 (1.38) | -.41 (1.50) |
| Beck Anxiety Inventory | 14.43 (9.56) | 15.86 (9.70) | 22.78 (10.88) |
| Fear Questionnaire | 25.12 (18.08) | 33.53 (18.82) | 41.13 (21.78) |
| Inventory of Depressive Symptomatology | 31.37 (10.61) | 23.57 (9.99) | 37.56 (10.95) |

*Note*. IAT = Implicit Association Test. [a]Positive effects indicate a relatively stronger automatic/explicit association between *me* and *calm/elated*. Please note that the D-measure can take negative as well as positive values.

Table 5.2

*Correlation matrix of predictor variables at baseline in individuals with a depressive disorder and/or anxiety disorder (N =1446)*

| Measure | 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|---|---|---|---|---|---|---|---|
| 1. IAT anxiety | - | .47* | .25* | .17* | -.22* | -.18* | -.18* |
| 2. IAT depression | - | - | .23* | .31* | -.15* | -.18* | -.23* |
| 3. Explicit self-anxious | - | - | - | .63* | -.52* | -.40* | -.53* |
| 4. Explicit self-depressed | - | - | - | - | -.33* | -.33* | -.65* |
| 5. Beck Anxiety Inventory | - | - | - | - | - | .45* | .63* |
| 6. Fear Questionnaire | - | - | - | - | - | - | .40* |
| 7. Inventory of Depressive Symptomatology | - | - | - | - | - | - | - |

*Note*. IAT = Implicit Association Test. *$p < .01$. Please note that the negative correlations between self-associations and symptom measures are in line with the expectation, since positive effects for self-associations indicate a relatively stronger automatic/explicit association between *me* and *calm/elated*.

Table 5.3

*Cox's proportional survival analyses for predicting time to first remission from anxiety disorders between baseline and 2-year follow-up (n =1136)*

| | **Single predictor[a]** | **Multi predictor[a]** |
|---|---|---|
| | **HR (95% CI)** | **HR (95% CI)** |
| IAT self-anx | 1.20 (1.02 – 1.41)* | .97 (.74 – 1.27) |
| IAT self-dep | 1.14 (.95 – 1.38) | .89 (.68 – 1.16) |
| Explicit self-anx | 1.21 (1.12 – 1.29)** | 1.11 (1.02 – 1.21)* |
| Explicit self-dep | 1.15 (1.07 – 1.24)** | 1.06 (.98 – 1.15) |
| Beck Anxiety Inventory | .98 (.97 – 1.00)** | 1.00 (.99 – 1.01) |
| Fear Questionnaire | .99 (.98 – .99)** | .99 (.99 – 1.00)** |
| Comorbidity (anx/dep) | .84 (.67 – 1.06)[b] | 1.08 (.87 – 1.34) |
| Comorbidity (anx/anx) | .73 (.60 – .88)** | .91 (.76 – 1.09) |
| IAT anx x Explicit self-anxious | | .99 (.90 – 1.09) |
| IAT anx x Comorbidity (anx/dep) | | 1.07 (.86 – 1.32) |
| IAT anx x Comorbidity (anx/anx) | | 1.08 (.90 – 1.29) |

*Note*. IAT = Implicit Association Test; anx = anxiety; dep = depression; HR = Hazard Ratio; CI = Confidence Interval. **$p < .01$; *$p < .05$ [a]'Single predictor' means that the predictors were separately included in the regression model, while 'multi predictor' means that all predictors were simultaneously entered into the regression model. [b]Similar results were obtained when we used listwise deletion of missing data with exception of this effect which is no longer significant.

Table 5.4

*Cox's proportional survival analyses for predicting time to first remission from depressive disorders between baseline and 2-year follow-up (n = 882)*

| | **Single predictor[a]** | **Multi predictor[a]** |
|---|---|---|
| | **HR (95% CI)** | **HR (95% CI)** |
| IAT self-dep | 1.23 (1.01 – 1.49)* | 1.04 (.70 – 1.53) |
| IAT self-anx | 1.10 (.93 – 1.30) | .97 (.79 – 1.18) |
| Explicit self-dep | 1.19 (1.11 – 1.27)** | 1.14 (1.05 – 1.23)** |
| Explicit self-anx | 1.14 (1.06 – 1.22)** | .99 (.91 – 1.08) |
| Inventory of Depressive Symptomatology | .98 (.97 – .99)** | .99 (.98 – 1.00) |
| Comorbidity (anx/dep) | .85 (.71 – 1.02) | 1.01 (.84 – 1.22) |
| Comorbidity (dep/dep) | .67 (.55 – .82)** | .76 (.62 – .92)** |
| IAT dep x Explicit self-dep | | 1.02 (.94 – 1.11) |
| IAT dep x Comorbidity (anx/dep) | | .96 (.79 – 1.16) |
| IAT dep x Comorbidity (dep/dep) | | 1.10 (.90 – 1.35) |

*Note*. Similar results were obtained when we used listwise deletion of missing data. IAT = Implicit Association Test; dep = depression; anx = anxiety; HR = Hazard Ratio; CI = Confidence Interval. **$p < .01$; *$p < .05$ [a]'Single predictor' means that the predictors were separately included in the regression model, while 'multi predictor' means that all predictors were simultaneously entered into the regression model.

cators associated with lower risk for first remission were stronger explicit self-depressed and explicit self-anxious associations, higher severity of depressive symptoms (IDS-SR), and having double depression. When all predictors were simultaneously entered into the model, automatic self-depressed associations no longer showed predictive validity. Only stronger explicit self-depressed associations and having double depression were found to be independently associated with a decreased chance of remitting from depression.[11]

## Discussion

This study represents the first research into the prognostic value of automatic self-associations for the natural course of depressive and anxiety disorders over a longer period of time. Moreover, this is the first enterprise investigating the disorder-specificity of automatic self-associations across anxiety and depression longitudinally. Results showed that in the single predictor models specifically automatic self-anxious associations were related to a reduced chance of remission from anxiety, whereas automatic self-depressed associations were related to a reduced chance of remission from depression. These findings support the hypothesis that relatively automatic dysfunctional self-associations are involved in the persistence of depressive and anxiety disorders. When all predictors were simultaneously included in the models only explicit anxious self-beliefs and fearful avoidance behaviour showed independent predictive validity for remission from anxiety. Explicit self-depressed beliefs and double depression were the only independent predictors for remission from depression.

In line with the theoretical starting point of this study (Gawronski & Bodenhausen, 2006; Strack & Deutsch, 2004), outcomes show that indeed automatic self-associations were predictive for the natural course of depressive and anxiety disorders. The relationship between course and automatic associations seems to be disorder-specific, since automatic self-anxious associations showed predictive validity for course of anxiety, whereas automatic self-depressed associations predicted course of depression. This is in line with prior results of this sample showing similar disorder-specific group differences between anxious and depressed patients at baseline (Glashouwer & de Jong, 2011). Yet, this is the first time that the value of automatic self-associations is demonstrated for the natural course of depressive and anxiety symptoms in a longitudinal design. However, the effects of automatic self-associations for course of anxiety and depression did not show addi-

[11] We repeated Cox's survival analyses including only disorder-specific IATs and symptom measures (remission anxiety: IAT anxiety, FQ and BAI; remission depression: IAT depression and IDS-SR). Again, automatic self-associations did not show predictive validity over and above symptom measures of depression and anxiety.

tional predictive validity over and above explicit self-associations and symptom measures of anxiety and depression. In addition, exploratory analyses did not provide support for the hypothesis that automatic self-associations would be especially relevant for predicting course of symptoms in patients with comorbid anxiety and/or depressive disorders.

Although the lack of independent predictive validity of automatic self-associations may seem to suggest that automatic self-associations are largely redundant, this is not necessarily the case. First of all, because the input of the associative system is assumed to be used for more explicit, rule-based mental processing (Strack & Deutsch, 2004), it could well be that effects of automatic associations 'run through' explicit associations. Consequently, entering both in the analysis could have removed statistical significance of automatic associations in predicting future symptoms. More final conclusions regarding the role of automatic self-associations would require experimental manipulation of these associations (cf. Clerkin & Teachman, 2010). In addition, there is considerable method variance shared between self-report measures and outcome measures that is not shared with automatic self-associations, making it a much tougher test for automatic self-associations. Given the alleged importance of automatic associations in guiding relatively automatic behaviours, it would be important for future study designs to also include indices that reflect more spontaneous behavioural aspects of depressive and anxiety disorders (cf. Egloff & Schmukle, 2002; Huijding & de Jong, 2006). Such an approach would allow to test possible differential pathways of dysfunctional automatic and/or explicit associations in guiding spontaneous and more controlled symptoms of anxiety and depression.

When all predictors were included together in the model, explicit self-anxious beliefs and fearful avoidance behaviours showed independent predictive properties for the course of anxiety. Explicit self-depressed beliefs and having a double depression were the only independent predictors for remission from depression. One could argue that the present measures of explicit self-beliefs were no 'official', well-established measurements. However, we had clear reasons to include these particular measures, instead of using more conventional (trait) measures. For comparability, we wanted to include explicit measures that were as similar as possible to the concepts included in the automatic self-associations. Since existing measures of self-concepts are usually questionnaires covering several characteristics, we had to compose specific measures of self-anxious and self-depressed beliefs ourselves. This kind of explicit ratings is often used in psychology research (cf. Hofmann, Gawronski, Gschwendner, Le & Schmitt, 2005), because it is assumed to reduce the divergence of automatic and explicit measures (cf. Payne, Burkley & Stokes, 2008). Finally, the explicit self-beliefs displayed high internal consistencies in our sample and discrimi-

nated clearly between diagnostic groups in a prior study (Glashouwer & de Jong, 2010) indicating that the measure has also adequate psychometric properties.

Although there were good reasons to include these particular measures for explicit self-beliefs, it brings up the question how we should best conceptualize these measures compared to symptom measures and trait characteristics. On the one hand anxious and depressed self-views seem to co-occur with anxiety and depressive symptoms, because they moderately correlated with symptom measures and when explicit self-beliefs and symptom measures were included simultaneously in the statistical model, predictive validity of the BAI and IDS-SR disappeared. On the other hand, explicit self-beliefs showed predictive validity over and above symptom measures. The latter could imply that explicit self-beliefs also partly reflect more stable, trait-like beliefs individuals have regarding themselves, next to temporal self-views which are influenced by or part of momentary symptom severity. This way, explicit self-anxious and self-depressed beliefs might be similar to the concepts of trait anxiety and trait depression.

We are not aware of prior longitudinal studies that demonstrated the influence of trait anxiety/depression on the course of anxiety and depression, although other studies have shown that higher anxiety sensitivity is related to an unfavourable course of panic disorder (e.g., Chavira et al., 2009; Ehlers, 1995; Pérez Benítez et al., 2009; Pollack, Otto, Rosenbaum & Sachs, 1990; Schmidt & Bates, 2003) and higher neuroticism is related to an unfavourable course of depressive disorders (e.g., Brown & Rosellini, 2011; Hayden & Klein, 2001; Rhebergen et al., 2009; Steunbergen, Beekman, Deeg & Kerkhof, 2010) and anxiety disorders (e.g., Chavira et al., 2009; De Beurs, Beekman, Deeg, van Dyck & Tilburg, 2000). While neuroticism refers to a general tendency to experience negative emotional states, explicit self-anxious and self-depressed beliefs might reflect more specific anxious or depressive response tendencies. In line with this idea, the effects of explicit self-beliefs were indeed found to be disorder-specific. Furthermore, anxiety sensitivity and self-anxious beliefs might be related as well, but both constructs also seem to differ in an important respect. When a person has the tendency to respond anxiously to a variety of situations (self-anxious beliefs) it does not automatically imply that this person also fears symptoms related to anxiety (anxiety sensitivity). Clearly, further research will be necessary to test how exactly self-anxious/depressed associations relate to neuroticism and anxiety sensitivity in the context of the course of anxiety disorders and depression.

Finally, it should be acknowledged that the order of the anxiety IAT and the depression IAT, and the order of the category combinations within both IATs, was fixed. Although this has clear advantages with regard to the enhancement of the sensitivity of the IAT as

a measurement of individual differences (cf. Asendorpf, Banse & Mücke, 2002; Steffens & König, 2006), this procedure also has some important drawbacks. IAT-effects tend to decrease with the number of IATs presented to a participant (Greenwald et al., 2003). Consequently, the present fixed order hampers direct comparison of both IATs. Furthermore, it makes it hard to interpret the absolute value of the IAT outcomes, which means that the positive IAT indices that we found do not simply imply stronger self-calm/elated associations. The positive indices could also be caused by order effects resulting in the zero point not being an actual 'zero point'. However, it seems that this ambiguity is not critical in the present context, because our focus was primarily on the predictive validity of automatic associations for course instead of absolute values of automatic associations per se.

### *Conclusions*

The present outcomes demonstrate that automatic dysfunctional self-associations are related to course of anxiety and depressive disorders. However, only explicit self-associations together with other factors (avoidance behaviours and comorbidity) seem to play an independent role in maintaining depressive and anxiety symptoms over time. Hereby, the present results are in line with the theoretical and empirical starting point that negative cognitions with regard to the self form an important underlying mechanism in the maintenance of anxiety and depressive disorders. A crucial next step would be to examine whether experimentally reducing dysfunctional self-associations (e.g., via classical conditioning procedures; Clerkin & Teachman, 2010), has beneficial effects on symptoms. If so, this would not only elucidate the exact nature of the relationship between self-associations and depressive and anxiety disorders, it could also help to improve further preventive interventions for depression and anxiety and 'break through' the persistent and recurrent nature of these disabling disorders.

# Study 2: Onset of anxiety disorders

## Abstract

Negative self-beliefs are assumed to play an important role in the onset of anxiety disorders. Current dual process models emphasize the relevance of differentiating between more automatic and more explicit self-beliefs in this respect. Therefore, this study was designed to test the prognostic value of both explicit and automatic self-anxious associations as a generic vulnerability factor for the onset of anxiety disorders between baseline and 2-year follow-up. To test the disorder-specificity of negative self-associations we also measured self-depressed associations. Self-report measures of depressive symptoms, anxiety symptoms, neuroticism, and fearful avoidance were included as covariates. Healthy controls ($n$ = 593), depressed individuals ($n$ = 238), and individuals remitted from an anxiety disorder ($n$ = 448) were tested as part of the Netherlands Study of Depression and Anxiety (NESDA). Explicit self-anxious associations predicted the onset of anxiety disorders in all groups. Automatic self-anxious associations only showed predictive validity in individuals remitted from an anxiety disorder or in currently depressed individuals. Although explicit self-depressed associations were related to the onset of anxiety disorders as well, automatic self-depressed associations were not. In the (remitted) patient groups only explicit self-anxious associations showed independent predicting value for the onset of anxiety disorders together with self-reported fearful avoidance behaviour. In the healthy controls, only a composite index of negative emotionality (comprising of depressive/anxiety symptoms and neuroticism) showed independent predictive validity. This study provides the first evidence that automatic and explicit self-anxious associations have predictive value for the future onset of anxiety disorders.

Based on: Glashouwer, K. A., de Jong, P. J., & Penninx, B. W. J. H. (2011). Predictive validity of automatic self-associations for the onset of anxiety disorders. *Journal of Abnormal Psychology, 120,* 607-616.

## Introduction

Anxiety disorders represent a major problem for public health. The prevalence, persistence, and recurrence of anxiety disorders form a social (e.g., Buist-Bouwman et al., 2006) and economic (Smit et al., 2006) burden that weighs heavily on the shoulders of patients and society. Therefore, it seems of paramount importance to further enhance insight into factors that contribute to the onset of anxiety disorders. Cognitive theories point to the importance of negative cognitions with regard to 'the self' in the onset and maintenance of psychopathology (e.g., Clark, Beck & Alford, 1999). Following this, it has been proposed that relatively strong negative self-beliefs may set people at risk for developing anxiety disorders (e.g., Egloff & Schmuckle, 2002; Glashouwer & de Jong, 2010).

According to recent dual-process models, it is important to distinguish between more explicit, ruled-based (i.e., explicit) self-beliefs and more automatically activated associations (e.g., Gawronski & Bodenhausen, 2006). Automatic self-associations are assumed to be simple links between self and associated concepts in memory, which can be activated directly in response to relevant stimuli. Thus, when an anxiety-relevant stimulus appears, this is thought to directly activate anxiety-related self-associations via the spreading of activation from one concept to associated concepts. These automatic associations are thought to influence more spontaneous behavioural responses towards threatening stimuli (e.g., Huijding & de Jong, 2006a). Subsequently, the input of the associative system is assumed to be used for more explicit, rule-based mental processing (Strack & Deutsch, 2004) where propositions are weighted according to their 'truth' values (i.e., validation processes; Gawronski & Bodenhausen, 2006). These explicit cognitions are thought to guide more controlled behaviours. Considering that anxiety symptoms include spontaneous as well as controlled behaviours, both explicit and more automatic dysfunctional self-associations might play an important role in the cognitive vulnerability for developing anxious symptoms (Strack & Deutsch, 2004; Ouimet, Gawronski & Dozois, 2009).

In support of the potential role of automatic self-associations in the onset of anxiety disorders, several cross-sectional studies already demonstrated that anxious individuals had stronger dysfunctional automatic self-associations than non-anxious controls (social anxiety: de Jong, 2002; Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008; Tanner, Stopa & De Houwer, 2006; panic disorder: Teachman, 2005; Teachman, Smith-Janik & Saporito, 2007; for an extensive review see: Roefs et al., 2011). Moreover, it was shown that higher strength of automatic catastrophic associations significantly predicted a smaller reduction in anxiety sensitivity in response to cognitive-behavioural therapy (CBT) in panic patients (Schneider & Schulte, 2008). Furthermore, changes in automatic panic associations

over the course of CBT for panic disorder were correlated with greater symptom reduction (Teachman, Marker & Smith-Janik, 2008). Finally, automatic self-anxious associations were found to predict experimentally-provoked spontaneous anxious behaviours (Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002). In line with the latter findings, we hypothesized that specifically automatic self-anxious associations might form a generic vulnerability factor for developing an anxiety disorder. Therefore, as a first step, we demonstrated in a previous study that automatic self-anxious associations indeed were stronger in individuals with an anxiety disorder, not only compared to controls, but also compared to depressed individuals (Glashouwer & de Jong, 2010). The present study forms a logical next research step, in which the prognostic value of automatic self-associations for the aetiology of anxiety disorders is studied in the context of a prospective design.

As part of the Netherlands Study of Depression and Anxiety (NESDA; see www.nesda.nl) we, therefore, assessed automatic self-anxious associations in a large cohort comprising of healthy controls, depressed individuals without a comorbid anxiety disorder, and individuals who were remitted from an anxiety disorder. We tested whether the strength of automatic self-anxious associations during baseline assessment were predictive of the onset of anxiety disorders at two years follow up. In addition, we tested the specificity of automatic self-anxious associations in predicting the onset of anxiety disorders by measuring automatic self-depressed associations supplementary to self-anxious associations. In addition, as explicit equivalents of the automatic self-associations, explicit self-anxious and self-depressed associations were included in the study. Although previous studies provided evidence indicating that self-reported negative self-views were predictive of the onset of anxious symptoms and/or behaviours (e.g., Acarturk, et al., 2009; Batelaan et al., 2010; Hirsch, Clark, Mathews & Williams, 2003; Hirsch, Mathews, Clark, Williams & Morrison, 2006), no research exists yet into more specific anxious or depressive self-concepts. Finally, for exploratory reasons we studied whether the predictive validity of automatic self-associations varied across the onset of the various anxiety disorders that were included in the study (i.e., panic disorder, social phobia, generalized anxiety disorder, and agoraphobia). Our hypothesis was that automatic self-anxious associations would be generally related to the onset of anxiety disorders, irrespective of the type of anxiety disorder or whether it was the first, second or subsequent episode of the disorder.

## Methods

### *Study sample*

The present study was carried out in the context of the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008), a multi-center, ongoing cohort study, designed to examine the long-term course and consequences of anxiety and depressive disorders. A total of 2981 persons aged 18 through 65 were included, including healthy controls, individuals at risk because of prior episodes, sub threshold symptoms or family history, and individuals with a current first or recurrent depressive and/or anxiety disorder. The inclusion was restricted to Major Depressive Disorder, Dysthymia, General Anxiety Disorder, Panic Disorder, Social Phobia, and Agoraphobia, because these disorders are relatively homogenous in phenotype and are found across different health care settings. Recruitment of respondents took place in the general population, in general practices, and in mental health care institutions. General exclusion criteria were a primary clinical diagnosis of a psychiatric disorder not subject of NESDA which would largely affect course trajectory (i.e., psychotic disorder, bipolar disorder, or severe addiction disorder) and not being fluent in Dutch. The present study concerns the baseline and the 2-year follow-up measurements conducted from September 2004 till October 2009. The study protocol was approved centrally by the Ethical Review Board of VU Medical Center Amsterdam and subsequently by local review boards of each participating center/institute, and all participants provided written informed consent.

After two years, a face-to-face follow-up assessment was conducted with a response of 87.1 % ($N$ = 2596). Non-response was significantly higher among those with younger age, lower education, non-European ancestry, and depressive disorder, but was not associated with gender or anxiety disorder. The presence of depressive or anxiety disorders was established with the Composite International Diagnostic Interview (CIDI; WHO version 2.1) which classifies diagnoses according to the DSM-IV criteria (American Psychiatric Association, 2000). The CIDI is used worldwide and WHO field research has found high interrater reliability, (Wittchen et al., 1991) high test-retest reliability, (Wacker, Battegay, Mullejans & Schlosser, 2006) and high validity for depressive and anxiety disorders (Wittchen et al., 1989; Wittchen, 1994). We chose to study the onset of anxiety disorders in three different groups (total $N$ = 1352): healthy controls with no current (during the past month) and prior anxiety disorder or depressive disorder ($n$ = 601); individuals remitted from an anxiety disorder with no current anxiety disorder or depressive disorder ($n$ = 500); individuals with a current major depressive disorder without a current anxiety disorder ($n$ = 251). Of the depressed group, 37% had a prior anxiety disorder. Therefore, the depressed group

was analysed twice, once including all currently depressed individuals independent of a history of anxiety ('broad group' $n = 251$) and once including only the depressed individuals without a history of anxiety ('restricted group' $n = 158$). People were considered remitted when they did not meet the criteria for an anxiety disorder during the past month, but had an anxiety episode in the past.

### *Measures*

**Automatic self-associations.** The Implicit Association Test (IAT) is a computerized reaction time task originally designed by Greenwald, McGhee & Schwartz (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left or right response key. The premise here is that the sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated (e.g., an anxious person should find it easier to categorize words of *me* and *anxious* with the same button than *me* and *calm*). The category labels are visible in the upper left and right-hand corners of the screen during the whole task (for an example see https://implicit.harvard.edu/implicit). For both IATs target labels were *me* and *others*. Following the design of Egloff and Schmukle (2002), an IAT anxiety was constructed with the attribute labels *anxious* and *calm*. Analogously, the attribute labels were *depressed* and *elated* for the depression IAT. Each category consisted of five stimuli (see Chapter 2, Appendix 2.A). The attribute stimuli of the anxiety IAT were the same self-descriptors as used by Egloff & Schmukle (2002) who based their IAT on trait anxiety. We wanted our self-anxious IAT to be highly comparable to these previous findings. Furthermore, we designed a self-depressed IAT in an equivalent way. Therefore, we decided to include self-descriptors that were as little as possible a reflection of depressive symptoms only, e.g. we did not include mood words like sad, unhappy or gloomy. The exact stimuli were selected from trait self-descriptors of depressive persons that were also used in previous work on attentional bias in (remitted) depression of McCabe and Gotlib (e.g., McCabe, Gotlib & Martin, 2000). Both IATs consisted of two critical test blocks that were preceded by practice blocks (see Chapter 2, Table 2.1). The order of category combinations was fixed across participants to reduce method variance. This is assumed to enhance the sensitivity of the IAT as measure of individual differences, which is important in view of the prospective design of this study (cf., Asendorpf et al., 2002; Steffens & König, 2006).

**Explicit self-associations.** To obtain explicit self-associations equivalently to the au-

tomatic self-associations, participants rated all IAT attribute stimuli on a 5-point scale (1 = hardly/not at all, 5 = very much) (i.e., "For each word please indicate to what extent you think it generally applies to you."). In a prior design we tried to create a relative measure of explicit self-associations in a similar structure as the IAT. However, because pilot studies showed that people found it much more difficult to rate to what extent certain attributes applied to 'others' than to 'themselves', and since we were afraid that this confusion could increase measurement errors, we eventually decided to measure only to what extent the attributes applied to themselves. It is rather common to use these kinds of ratings in psychological research (e.g., Hofmann, Gawronski, Gschwendner, Le & Schmitt, 2005) because using this type of rating is assumed to reduce the chance that a divergence of implicit and explicit measures (also in terms of differential predictive validity) only occurs because different type of stimuli are used. Germane to this it was recently shown that the correlation between implicit and explicit measures greatly increased when the measurement procedures were made as similar as possible, by using the same response metric and same type of stimuli in both measurement procedures, with the only remaining difference being that one was a direct and the other an indirect assessment of evaluations (Payne, Burkley & Stokes, 2008).

**Onset of anxiety disorders.** The CIDI interview was used to determine the presence of DSM-IV classified anxiety disorders (General Anxiety Disorder, Panic Disorder, Social Phobia, and Agoraphobia) during the time between baseline assessment and 2-year follow-up. Organic exclusion rules were used in defining hierarchy free diagnoses, i.e. when there was evidence that symptoms were entirely due to an organic (biological) disorder, then – in line with DSM-IV criteria – the diagnosis was not made.

**Questionnaire data.** Several variables were included in the design to correct for possible overlap between self-associations on the one hand and anxiety symptoms, depressive symptoms, and general negative emotionality (neuroticism) on the other hand. Severity of anxiety symptoms at baseline was measured with the 21-item Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988), whereas fearful avoidance behaviour was measured using the 15-item Fear Questionnaire (Marks & Mathews, 1979). To measure neuroticism, we used the 12-item neuroticism subscale of the NEO-Five Factor Inventory (NEO-FFI; Costa & McCrae, 1995). Severity of depressive symptoms was measured with the 30-item Inventory of Depressive Symptoms self-report version (IDS-SR, Rush, Gullion, Basco & Jarrett, 1996). Total scale scores were used for all questionnaires.

### *Procedure*

Baseline and follow-up assessments were similar and lasted between 3 and 5 hours and were conducted on 1 day. During the assessments, other measurements were collected as well, but these are not of interest for the present study (for a detailed description, see Penninx et al., 2008). Each participant completed the anxiety IAT, followed by the depression IAT. After that, participants deliberately rated the attribute words that were used in the IATs. Respondents were compensated with a €15,- gift certificate and travel expenses.

### *Data analyses*

**Data reduction.** IAT scores were computed according to the now widely used algorithm proposed by Greenwald, Nosek and Banaji (2003). We report the $D_4$-measure. Reaction times above 10,000 ms were discarded and error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred plus a penalty of 600 ms. For the anxiety IAT, the IAT effect was calculated by subtracting mean reaction times of Block 6 from Block 3 (practice) and Block 7 from Block 4 (test). The means of these two effects were divided by their pooled standard deviation based on all responses in Blocks 3, 4, 6, and 7. Analogously, the IAT effect was calculated for the depression IAT, based on Blocks 9, 10, 12, and 13. Positive IAT effects indicate relatively fast responses when *me* shared the response key with either *calm* or *elated*.[12] Split-half reliabilities of the present IATs were good, with Spearman-Brown corrected correlations between test-halves of .87 for the depression IAT and .92 for the anxiety IAT (test-halves were based on trials 1, 2, 5, 6, 9, 10 etc. vs. 3, 4, 7, 8, 11, 12 etc.).

To compute explicit association effects, mean ratings of anxious (depressed) IAT-stimuli were subtracted from mean ratings of calm (elated) IAT-stimuli. Hence, positive effects indicate strong explicit associations between *me* and *calm* (or *me* and *elated*). The internal consistency of the explicit self-association measures was excellent, with Cronbach's *α* .94 for the difference scores of anxious and calm words, and .95 of depressed and elated words.

Due to technical problems, the IAT data and explicit self-associations for 61 participants were missing. Furthermore, 3 participants were discarded from the analyses because more than 10% of the IAT trials were below 300 ms (Greenwald et al., 2003), sug-

[12] Please note that this scoring procedure is reversed compared to three prior NESDA publications in which the IAT was used (Glashouwer & de Jong, 2010; Glashouwer et al., 2010; van Harmelen et al., 2010). Originally we decided to 'reverse' the D-measure to make it comparable to e.g., symptom measures in which a higher score also indicates a less favourable outcome. However, thanks to an anonymous reviewer we decided to change the results to make them comparable to the general literature. The only thing that differs between the studies is the multiplication sign before the D-measure/explicit self-beliefs.

gesting that they were trying to respond too rapidly. Finally, 9 individuals were discarded because of missing data on the questionnaires. Consequently, the final study samples consisted of 593 healthy controls, 448 individuals remitted from an anxiety disorder and 238 depressed individuals (of which 150 depressed individuals had no history of anxiety, the 'restricted group').

**Statistical analyses.** First, bivariate Spearman rank order correlation coefficients were calculated between all predictors and the criterion variable, because some of the measures were not normally distributed. Subsequently, single predictor and multi predictor binary logistic regression analyses were used to predict the onset of an anxiety disorder in the different groups. To test whether self-associations added predictive validity over and above anxiety symptoms, fearful avoidance behaviour, neuroticism, and depressive symptoms, these predictors were included in the analyses as well. However, it appeared that anxiety symptoms, neuroticism, and depressive symptoms were highly correlated with each other (correlations ranging from .68 to .78). Therefore, we decided to combine these three variables into a single composite variable in the logistic regression analyses in order to further simplify these analyses. Relationships between the predictors and onset of an anxiety disorder are presented by means of odds ratios, which indicate the increased likelihood of the onset of an anxiety disorder between baseline and 2-year follow-up, given an increase of one unit in the independent variable. In addition, the prediction of the onset of different anxiety disorders was determined in single predictor and multi predictor multinominal logistic regression analyses using no anxiety disorder as the reference group and calculating the risks for the onset of the different anxiety disorders (General Anxiety Disorder, Panic Disorder, Social Phobia, Agoraphobia, more than one anxiety disorder). These analyses were conducted in the three groups together ($N = 1279$), because power was too low for analysing the groups separately. All tests were conducted with $\alpha < .05$. Missing information on any of the variables resulted in exclusion of the case from the particular analysis.

## Results

### *Descriptives*

The descriptives of the variables for the different groups are reported in Table 5.5. The correlations between the predictors and the criterion variable are shown in Table 5.6.

Table 5.5

*Means and standard deviations of the self-report and automatic measures at baseline as a function of group*

| Measure | Healthy controls | Remitted AD (no current AD or MDD) | Depressed (incl. history of AD) | Depressed (excl. history of AD) |
|---|---|---|---|---|
| Total *n* | 593 | 448 | 238 | 150 |
| *n* onset AD : *n* no onset AD | 34 : 559 | 130 : 318 | 77 : 161 | 37 : 113 |
| Gender, % female | 60.7 | 73.7 | 61.3 | 58.7 |
| Age | 40.90 (14.61) | 41.95 (13.29) | 41.66 (12.80) | 40.57 (13.15) |
| Educational level in years | 12.91 (3.21) | 12.49 (3.23) | 11.95 (3.15) | 11.68 (3.20) |
| IAT anxiety, D-measure[a] | .52 (.43) | .36 (.47) | .28 (.49) | .33 (.49) |
| Mean error rate IAT anxiety | 5.49 (5.16) | 5.26 (5.20) | 6.07 (6.10) | 6.44 (7.13) |
| IAT depression, D-measure[a] | .43 (.36) | .26 (.40) | .13 (.40) | .14 (.42) |
| Error rate IAT depression | 5.19 (4.74) | 5.21 (4.60) | 5.76 (5.54) | 6.09 (6.35) |
| Explicit self-anxious[a] | 2.18 (1.03) | .99 (1.26) | .22 (1.34) | .41 (1.38) |
| Explicit self-depressed[a] | 2.70 (.83) | 1.80 (1.24) | .40 (1.58) | .52 (1.59) |
| Beck Anxiety Inventory | 3.95 (4.66) | 8.98 (6.95) | 14.11 (8.87) | 13.16 (8.36) |
| Fear Questionnaire | 12.02 (11.97) | 20.77 (15.45) | 24.73 (17.28) | 22.74 (16.74) |
| Neuroticism | 26.97 (7.36) | 35.53 (7.63) | 40.17 (6.45) | 39.58 (6.65) |
| Inventory of Depressive Symptomatology | 8.33 (7.28) | 16.97 (9.39) | 31.39 (10.45) | 30.91 (10.08) |

*Note*. IAT = Implicit Association Test; AD = Anxiety Disorder; MDD = Major Depressive Disorder. [a] Positive effects indicate a relatively stronger automatic/explicit association between *me* and *calm/elated*.

Table 5.6

*Correlation matrix of predictors at baseline in healthy controls, depressed individuals, and individuals remitted from an anxiety disorder (N =1279)*

| Measure | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. |
|---|---|---|---|---|---|---|---|---|
| 1. IAT anxiety | .48* | .30* | .26* | -.23* | -.19* | -.31* | -.25* | -.15* |
| 2. IAT depression | - | .32* | .34* | -.26* | -.21* | -.34* | -.32* | -.14* |
| 3. Explicit self-anxious | | - | .75* | -.67* | -.53* | -.79* | -.72* | -.36* |
| 4. Explicit self-depressed | | | - | -.56* | -.48* | -.76* | -.71* | -.30* |
| 5. Beck Anxiety Inventory | | | | - | .51* | .68* | .77* | .34* |
| 6. Fear Questionnaire | | | | | - | .57* | .50* | .31* |
| 7. Neuroticism | | | | | | - | .78* | .35* |
| 8. Inventory of Depressive Symptomatology | | | | | | | - | .35* |
| 9. Onset of anxiety disorder | | | | | | | | - |

*Note*. IAT = Implicit Association Test. * $p < .01$ (2-tailed). Please note that the negative correlations between self-associations and symptom measures are in line with the expectation, since positive effects for self-associations indicate a relatively stronger automatic/explicit association between me and calm/elated.

Table 5.7

*Single and multi predictor logistic regression models for predicting onset of anxiety disorders between baseline and 2-year follow-up in healthy controls and individuals remitted from an anxiety disorder*

| | **Healthy controls** | | **Remitted from AD (no current MDD)** | |
|---|---|---|---|---|
| ***n* onset : no onset AD** | **34 : 559** | | **130 : 318** | |
| **Total *n*** | **593** | | **448** | |
| | **Single predictor[a]** | **Multi predictor[a]** | **Single predictor[a]** | **Multi predictor[a]** |
| | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** |
| IAT anxiety | .75 | 1.29 | .64* | .77 |
| | (.34 – 1.64) | (.50 – 2.09) | (.42 – .98) | (.46 – 1.27) |
| IAT depression | .48 | .81 | .90 | 1.40 |
| | (.19 – 1.23) | (.26 – 2.52) | (.54 – 1.49) | (.75 – 2.62) |
| Explicit | .48** | .78 | .65** | .78† |
| self-anxious | (.36 – .64) | (.49 – 1.25) | (.55 – .77) | (.61 – 1.01) |
| Explicit | .53** | 1.31 | .72** | 1.04 |
| self-depressed | (.38 – .74) | (.79 – 2.18) | (.61 – .85) | (.80 – 1.34) |
| Fear Questionnaire | 1.05** | 1.01 | 1.03** | 1.02* |
| | (1.03 – 1.07) | (.99 – 1.04) | (1.02 – 1.05) | (1.01 – 1.04) |
| Composite score[b] | 4.64** | 3.90** | 2.31** | 1.48† |
| | (2.85 – 7.57) | (1.78 – 8.54) | (1.71 – 3.13) | (.96 – 2.26) |

*Note*. IAT = Implicit Association Test; MDD = Major Depressive Disorder; AD = Anxiety Disorder. ** $p < .01$ (2-tailed); * $p < .05$ level (2-tailed); † $p < .10$ (2-tailed). [a] 'Single predictor' means that the predictors were separately included in the regression model, while 'multi predictor' means that all predictors were simultaneously entered into the regression model; [b] Beck Anxiety Inventory, Inventory of Depressive Symptomatology and Neuroticism were combined in one composite score.

## *Are automatic and explicit self-associations at t0 predictive for the onset of an anxiety disorder between t0 and t2?*

Single predictor logistic regression analyses showed that automatic self-anxious associations were significantly associated with the onset of an anxiety disorder in the remitted group and the depressed group, whereas a non-significant trend in the same direction was shown in the more restricted depressed group (Table 5.7 and 5.8). The automatic self-depressed associations showed no significant predictive validity in any of the groups. Furthermore, in all groups, *explicit self-anxious* associations were predictive of the onset of an anxiety disorder between baseline and 2-year follow-up. Additionally, *explicit self-depressed* associations were shown to be significantly associated with the onset of an anxiety disorder in healthy controls, individuals remitted from an anxiety disorder, and depressed individuals, but not in the more restricted depressed group. In all groups, fearful avoidance behaviour (FQ) as well as the composite score of anxious symptoms (BAI),

Table 5.8

*Single and multi predictor logistic regression models for predicting onset of anxiety disorders between baseline and 2-year follow-up in depressed individuals with and without a history of anxiety disorder*

| | **Depressed (incl. history of AD)** | | **Depressed (excl. history of AD)** | |
|---|---|---|---|---|
| ***n* onset : no onset AD** | **77 : 161** | | **37 : 113** | |
| **Total *n*** | **238** | | **150** | |
| | **Single predictor[a]** | **Multi predictor[a]** | **Single predictor[a]** | **Multi predictor[a]** |
| | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** | **Odds ratio (95% CI)** |
| IAT anxiety | .45** | .56 | .52† | .61 |
| | (.25 – .79) | (.28 – 1.12) | (.24 – 1.10) | (.24 – 1.53) |
| IAT depression | .57 | 1.49 | .73 | 1.50 |
| | (.28 – 1.15) | (.59 – 3.73) | (.30 – 1.81) | (.48 – 4.71) |
| Explicit self-anxious | .54** | .60** | .67** | .67† |
| | (.43 – .69) | (.43 – .85) | (.50 – .89) | (.44 – 1.02) |
| Explicit self-depressed | .77** | 1.15 | .87 | 1.20 |
| | (.64 – .92) | (.89 – 1.49) | (.69 – 1.10) | (.85 – 1.68) |
| Fear Questionnaire | 1.04** | 1.03** | 1.04** | 1.03* |
| | (1.02 – 1.05) | (1.01 – 1.05) | (1.01 – 1.06) | (1.00 – 1.06) |
| Composite score[b] | 2.49** | 1.29 | 1.85* | .98 |
| | (1.67 – 3.72) | (.74 – 2.27) | (1.11 – 3.11) | (.46 – 2.08) |

*Note*. IAT = Implicit Association Test; MDD = Major Depressive Disorder; AD = Anxiety Disorder. ** $p < .01$ (2-tailed); * $p < .05$ level (2-tailed); † $p < .10$ (2-tailed). [a] 'Single predictor' means that the predictors were separately included in the regression model, while 'multi predictor' means that all predictors were simultaneously entered into the regression model; [b] Beck Anxiety Inventory, Inventory of Depressive Symptomatology and Neuroticism were combined in one composite score.

neuroticism (NEO-FFI) and depressive symptoms (IDS-SR) were significantly related to the onset of an anxiety disorder.

When all predictors were simultaneously entered into the logistic regression model, automatic self-associations as well as explicit self-depressed associations were no longer significant predictors. However, explicit self-anxious associations significantly predicted the onset of an anxiety disorder in the depressed group, whereas non-significant trends in the same direction were shown in the remitted group and the more restricted depressed group. Furthermore, fearful avoidance behaviour remained a significant predictor in the three (remitted) patient groups. Finally, the composite score was a significant predictor for onset of anxiety disorders in the control group and a non-significant trend in the same direction was shown in the remitted group. All significant effects were in the expected direction. Thus relatively strong self-anxious (depressed) associations were related to greater probabilities of the onset of an anxiety disorder between baseline and 2-year follow-up.[13]

Table 5.9

*Single and multi predictor multinominal regression analyses for predicting onset of different anxiety disorders between baseline and 2-year follow-up using no anxiety disorder as the reference group in healthy controls, depressed individuals en individuals remitted from an anxiety disorder (N = 1279)*

| | **Presence of Social Phobia between t0 - t2** ***n*** **= 65** | | **Presence of Panic Disorder between t0 - t2** ***n*** **= 58** | | **Presence of Agoraphobia between t0 - t2** ***n*** **= 26** | |
|---|---|---|---|---|---|---|
| | **Single predictor[a] Odds ratio (95% CI)** | **Multi predictor[a] Odds ratio (95% CI)** | **Single predictor[a] Odds ratio (95% CI)** | **Multi predictor[a] Odds ratio (95% CI)** | **Single predictor[a] Odds ratio (95% CI)** | **Multi predictor[a] Odds ratio (95% CI)** |
| IAT A | .55* | .94 | .56* | .71 | .26** | .50 |
| | (.32 – .93) | (.51 – 1.74) | (.32 – .97) | (.38 – 1.33) | (.12 – .56) | (.20 – 1.25) |
| IAT D | .43** | .77 | .81 | 1.67 | .21** | .60 |
| | (.23 – .80) | (.36 – 1.63) | (.41 – 1.59) | (.75 – 3.71) | (.08 – .53) | (.19 – 1.84) |
| EA A | .56** | .57** | .58** | .53** | .52** | .66† |
| | (.47 – .68) | (.44 – .74) | (.48 – .70) | (.40 – .70) | (.39 – .68) | (.44 – 1.00) |
| EA D | .68** | 1.02 | .73** | 1.11 | .58** | .84 |
| | (.58 – .79) | (.80 – 1.30) | (.61 – .87) | (.85 – 1.45) | (.46 – .74) | (.59 – 1.21) |

| | **Presence of GAD between t0 - t2** ***n*** **= 35** | | **Presence of two or more ADs between t0 - t2** ***n*** **= 58** | |
|---|---|---|---|---|
| | **Single predictor[a] Odds ratio (95% CI)** | **Multi predictor[a] Odds ratio (95% CI)** | **Single predictor[a] Odds ratio (95% CI)** | **Multi predictor[a] Odds ratio (95% CI)** |
| IAT A | .66 | 1.21 | .27** | .53* |
| | (.32 – 1.35) | (.52 – 3.82) | (.16 – .46) | (.28 – 1.00) |
| IAT D | .51 | 1.20 | .28** | 1.01 |
| | (.22 – 1.16) | (.43 – 3.32) | (.15 – .53) | (.46 – 2.25) |
| EA A | .46** | .53** | .39** | .43** |
| | (.36 – .58) | (.38 – .76) | (.32 – .48) | (.32 – .58) |
| EA D | .54** | .80 | .53** | .94 |
| | (.44 – .66) | (.59 – 1.08) | (.45 – .62) | (.74 – 1.21) |

*Note*. IAT= Implicit Association Test; EA = Explicit Associations; A = Anxiety; D = Depression; AD = Anxiety Disorder; GAD = Generalized Anxiety Disorder. ** $p < .01$ (2-tailed); * $p < .05$ level (2-tailed); † $p < .10$ (2-tailed). [a] 'Single predictor' means that the predictors were separately included in the regression model, while 'multi predictor' means that all predictors were simultaneously entered into the regression model.

[13] In a subsequent step, the interaction between automatic and explicit self-anxious associations and the interactions between self-associations and gender were added to the multivariate models. In this way, it was possible to examine whether the relationship between automatic self-anxious associations and onset of anxiety was especially strong in people also showing enhanced explicit self-anxious associations. Furthermore, we could test whether self-associations had a different effect in women than in men. Since earlier work showed that women are more likely than men to base their judgments on intuitions and gut impressions (e.g., Pacini & Epstein, 1999), we expected that automatic self-association would have a stronger predictive validity in women than in men. However, none of these interaction effects (nor the main effect of gender) added significant predictive validity to the model. For clarity reasons we therefore decided to leave these predictors out of Table 5.7 and 5.8.

### *Does the predictive validity of self-associations differ for different anxiety disorders?*

Explorative single predictor multinominal logistic regression analysis showed that both automatic and explicit self-anxious and self-depressed associations were predictive of the onset of *social phobia*, *agoraphobia*, as well as of *the presence of two or more anxiety disorders* between baseline and follow-up (as compared to no anxiety disorder between baseline and follow-up; Table 5.9). When predicting the onset of *panic disorder* with or without agoraphobia, explicit self-anxious and self-depressed associations as well as automatic self-anxious associations were significantly related to increased probabilities. When predicting the onset of *generalized anxiety disorder*, only the explicit self-associations were significant predictors. In the multi predictor models explicit self-anxious associations remained a significant and stable predictor of the onset of the different anxiety disorders. In addition, automatic self-anxious associations added independent predictive validity for *the presence of two or more anxiety disorders* between baseline and follow-up.

## Discussion

This study represents the first research into the prognostic value of automatic self-associations as a generic vulnerability factor for the onset of anxiety disorders in the context of a prospective design. In line with what we expected, results showed that automatic as well as explicit self-anxious associations were predictive of the onset of anxiety disorders between baseline and 2-year follow-up. Explicit self-anxious associations predicted the onset of anxiety disorders in all groups, whereas automatic self-anxious associations were related to the onset of anxiety disorders in the remitted group and in currently depressed individuals. However, a non-significant trend in the same direction was found in the more restricted depressed group. In addition, explicit self-depressed associations were related to the onset of anxiety disorders as well, but automatic self-depressed associations were not. When all predictors and covariates were simultaneously included in the analyses, fearful avoidance behaviour remained a stable significant predictor in the (remitted) patient groups. Explicit self-anxious associations showed independent predictive validity for the onset of anxiety disorders in the depressed group with similar non-significant trends in the remitted and more restricted depressed groups. Finally, the composite score of anxious symptoms, depressive symptoms and neuroticism was the only independent significant predictor in the control group.

In line with our hypotheses, automatic self-anxious associations were related to the onset of anxiety disorders. However, this effect was shown only in individuals remitted

from an anxiety disorder and in currently depressed individuals, whereas a non-significant trend was shown in the more restricted depressed group, but not in the healthy controls. Perhaps dysfunctional self-associations only establish on a more automatic level (and can thus only have an influence on the generation of symptoms) after someone has already suffered from an (anxiety) disorder. This would be in line with a previous prospective study into post-traumatic stress disorder (PTSD) showing that strong automatic associations between self and vulnerability seemed a consequence rather than a cause of first-onset PTSD symptoms (Engelhard, Huijding, van den Hout & de Jong, 2007). Furthermore, the present findings fit in well with the large number of cross-sectional studies demonstrating stronger dysfunctional automatic self-associations in anxious individuals than in non-anxious controls (Roefs et al., 2011) and with prior NESDA results showing stronger automatic self-anxious associations in remitted individuals than in healthy controls (Glashouwer & de Jong, 2010). Furthermore, the outcomes point to the importance of explicit self-anxious associations in the onset of anxiety disorders, both in (remitted) patients as well as in controls. Evidently, when individuals perceive themselves as anxious on a more conscious, explicit level, it increases their chance of developing an anxiety disorder later in time. This is in line with prior research (e.g., Acarturk, et al., 2009; Batelaan et al., 2010) and with cognitive theories pointing to the importance of negative cognitions with regard to 'the self' in the onset of psychopathology (e.g., Clark et al., 1999). Consequently, the present results might indicate that explicit self-anxious associations could be a pre-morbid vulnerability factor, whereas automatic self-anxious associations might be the residue of prior anxiety (or depressive) episode(s).

It should be acknowledged that in the present study automatic self-anxious associations did not show additional predictive validity over and above explicit self-anxious associations for the onset of anxiety disorders. Yet, this does not necessarily imply that automatic self-anxious associations are not an important mechanism underlying the risk of anxiety onset. First of all, because the input of the associative system is assumed to be used for more explicit, rule-based mental processing (Strack & Deutch, 2004), it could well be that the effect of automatic associations runs through explicit associations. Consequently, entering both in the analysis, can have taken away the statistical significance of automatic associations in predicting future symptoms. To arrive at more final conclusions regarding the role of automatic self associations it would be necessary for future studies to experimentally manipulate the automatic self-anxious associations (cf. Clerkin & Teachman, 2010). Furthermore, there is considerable method variance shared between explicit self-associations and the outcome measure that is not shared with the automatic self-

associations, making it a much tougher test for the automatic self-associations. Given the alleged importance of automatic associations in guiding relatively automatic behaviours, it would be important for future research to also include indices in the design that reflect the more spontaneous behavioural aspects of anxiety disorders (cf. Egloff & Schmukle, 2002; Huijding & de Jong, 2006a).

In addition, we investigated the specificity of self-anxious associations in predicting the onset of anxiety disorders by measuring self-depressed associations supplementary to the self-anxious associations. Automatic self-depressed associations were not significantly related to the onset of anxiety disorders, but explicit self-depressed associations were. When the predictors were simultaneously entered into the regression model, explicit self-depressed associations no longer had any significant predictive validity. Consequently, the findings seem to indicate a superiority of self-anxious associations over self-depressed associations in predicting the onset of anxiety disorders. Therefore, the most straightforward explanation of the predictive validity of explicit self-depressed associations in the single predictor models would be multicollinearity due to some conceptual overlap between anxious and depressive symptomatology. However, we cannot be certain whether the results are disorder-specific and due to self-anxious associations per se rather than a result of negative (self-)associations in general. Consequently, future research will have to further disentangle this issue by including additional types of self-associations in one design or by experimentally manipulating self-anxious associations. Moreover, it would be interesting to look at the role of (automatic) self-associations in the onset of other disorders, such as depression (see also e.g., Haeffel et al., 2007).

Finally, we explored whether the predictive validity of self-associations varied across the onset of various anxiety disorders. Automatic self-anxious associations were significantly related to the onset of all anxiety disorders with exception of generalized anxiety disorder (as compared to no anxiety disorder). Automatic self-depressed associations were related to the onset of social phobia, agoraphobia, as well as to the presence of more than one anxiety disorder in between baseline and 2-year follow-up. Explicit self-associations were related to the onset of all anxiety disorders. However, only explicit self-anxious associations were shown to be a stable predictor when all predictors were simultaneously included in the analyses. Furthermore, automatic self-anxious associations showed independent predictive validity for the onset of more than one anxiety disorder. These results seem to further underline the importance of automatic self-associations for the development of anxiety disorders, irrespective of which specific anxiety disorder it concerns. In addition, the findings again point to the importance of explicit self-anxious associations

in the onset of anxiety disorders.

An important question is how dysfunctional self-associations exactly increase the chance of developing anxious symptoms. Explicit self-anxious associations seemed to be different from (sub threshold) anxiety symptoms, anxious avoidance behaviour, depressive symptoms, or neuroticism, since at least in the depressed group self-associations showed predictive validity for the onset of anxiety disorders over and above these covariates, whereas similar trends were shown in the remitted and more restricted depressed group. In addition, fearful avoidance behaviour also was an independent predictor for the onset of anxiety disorders over time in the (remitted) patient groups. Possibly, individuals' anxious self-views together with their tendency to avoid fearful situations may increase their fear over time. Anxious self-views lead these individuals to expect to be unable to adequately deal with critical situations ('self-fulfilling prophecy'). Since they also have the tendency to avoid fearful situations, they do not receive critical information that can counter their self-views. By acting fearfully repeatedly over time, these individuals might actually become more and more anxious and get entangled in a vicious circle. Subsequently, associations might become installed on a more automatic level and trigger more spontaneous behavioural responses towards threatening stimuli. Accordingly, automatic self-anxious associations may contribute to the maintenance of anxiety symptoms. However, it should be acknowledged that the explicit self-associations overlapped considerably with the included covariates, especially neuroticism. Consequently, at least some of the predictive power of explicit self-associations in the multi predictor model might have been derived from shared variance with negative emotionality and (sub clinical) anxiety and depressive symptoms.

### *Limitations and considerations*

Since the present study was part of a larger research project, not all anxiety disorders could be included in the present sample. Consequently, it remains to be tested whether the present results also can be generalized to other anxiety disorders, such as obsessive compulsive disorder, post-traumatic stress disorder and specific phobia. Moreover, because there was no data establishing the inter-rater reliability of the diagnoses we cannot be entirely sure of these diagnoses. Meanwhile, we do not have any indications that the CIDI assessments were unreliable. In addition, although the attrition of the present study overall was quite limited, it could have somehow influenced the results. For example, baseline depression was shown to be related to drop-out. Possibly, a group with relatively severe symptoms might have been missed in the present study. Consequently, if any ef-

fect would have occurred it is likely to have resulted in an underestimation of the overall association. Additionally, one could argue that the present measures of explicit self-associations were not 'official', well-established measurements. However, it is rather common to use these kinds of ratings in psychological research (e.g., Hofmann et al., 2005) and we consider it reassuring in this respect that the baseline results showed clear distinctions in the expected direction on explicit self-associations between different groups (Glashouwer & de Jong, 2010). Finally, the structure of the explicit measures did not entirely parallel that of the automatic measures, in the sense that the IAT is a relative measure (self vs. others), whereas the explicit ratings were more one-dimensional. This could have had implications for the differential predictive validity of the explicit and automatic measures. Possibly the explicit measures not only show greater predictive validity for the onset of anxiety disorders because of the mentioned methodological overlap, but also because of the one-dimensional nature of the measurement that may have caused less confounding influence of 'other-associations'. However, as explained in the method section, we did not consider it feasible to measure explicit self-associations in a more relative way.

### *Conclusions*

In conclusion, the present study showed that automatic self-anxious associations are related to the onset of anxiety disorders both in individuals remitted from an anxiety disorder and in currently depressed individuals. Furthermore explicit self-anxious associations were predictive for the onset of anxiety disorders in patient groups as well as controls. In depressed individuals, the effect of explicit self-anxious associations remained significant over and above symptom measures and neuroticism. Finally, self-reported fearful avoidance was shown to be a stable independent predictor for the onset of anxiety disorders. These results are in line with the theoretical and empirical starting point that negative cognitions with regard to 'the self' form an important underlying mechanism in the onset of anxiety disorders. An important next step would be to examine whether experimentally reducing self-anxious associations has beneficial effects on anxious symptoms, for example, by means of classical conditioning procedures (e.g., Baccus, Baldwin & Packer, 2004; Clerkin & Teachman, 2010; Dijksterhuis, 2004). If so, this would not only elucidate the exact nature of the relationship between self-anxious associations and anxiety disorders, it could also point to fresh options that may improve further the currently available treatment options for individuals with anxiety disorders.

# 6 Summary and conclusions

Depressive and anxiety disorders are highly prevalent and represent a major problem for public health these days. Having to deal with one of these disorders has an enormous impact on a patient's life with pervasive social and economic consequences. In addition, for many patients symptoms keep returning, even after successful treatment. Consequently, the aim of this thesis was to examine underlying cognitive mechanisms that might contribute to the development, maintenance and relapse of these disabling disorders. This thesis starts from a dual-process model perspective adopting the idea that two kinds of mental processes are operating behind the scenes of people's behaviours: automatic and propositional processes. In response to external or internal triggers, associations are assumed to be activated spontaneously within pre-existing associative networks. Because activation automatically spreads through the associative network, associations can become activated irrespective of whether a person considers them as valid or invalid. When people are highly motivated to perform accurately and when they have sufficient time and cognitive resources, they can engage in propositional reasoning concerned with the validation of propositions. Associative processes are considered to be fast, require little cognitive capacity and are thought to be 'the default mode' of functioning. In general, automatic processes and corresponding behaviours are seen as really adaptive and contributing to our survival value. However, sometimes individuals can be really bothered by their thoughts and automatic behaviours which might be the case in depressive and anxiety disorders. These disorders have clear characteristics that point to automaticity. Consequently, studying automatic associations seems crucial for a better understanding of how anxiety and depressive disorders develop and why these disorders are so persistent once they have evolved.

The results of the present thesis will be discussed following four main questions. I will start with a methodological issue concerning the specific measurement tool that was used, the Implicit Association Test, asking (1) which algorithm performs best in calculating the IAT- effect in a laboratory setting? In addition, to investigate the potential role of automatic associations in the development and maintenance of anxiety and depressive disorders, I will focus on three questions: (2) are dysfunctional automatic self-associations vulnerability factors for developing anxiety and/or depressive disorders? (3) are dysfunctional automatic self-associations maintaining factors for anxiety and/or depressive disorders? and (4) are dysfunctional automatic associations specific characteristics of certain anxiety and/or depressive disorders or shared characteristics across several disorders?

In this final chapter, I will summarize and integrate the outcomes of the empirical studies presented in this thesis guided by the four research questions. In addition, I will touch

upon some issues that came up while conducting the research in this thesis. Finally, I will discuss the implications of this work for clinical practice.

## Summary and integration of empirical findings

### *1) Which algorithm performs best in calculating the IAT-effect in a laboratory setting?*

The main aim of *Chapter 2* was to evaluate the use of the $D_{600}$-measure in a laboratory setting. In the paper, 'Understanding and using the Implicit Association Test: I. An improved scoring algorithm', Greenwald, Nosek and Banaji (2003) already investigated various ways to calculate the IAT-effect. Their study showed that in large datasets collected through the internet the 'D-measures' perform best. However, it was unclear whether their findings also generalize to laboratory settings. In contrast to laboratory settings, internet studies almost completely lack experimental control, which could lower the commitment of participants to the task and by this, create more lapses of attention. Because short periods of inattention probably increase both the average and the variability of RTs, it might be that the superior performance of D-measures (that correct for variability by dividing by the pooled SD) is limited to situations without experimental control. Consequently, we hypothesized that, in a laboratory setting, the $D_{600}$-measure might perform equal or even worse compared to other algorithms.

It is usually very hard to find laboratory data-sets that are sufficiently large to achieve the required power for the purpose of such an enterprise. Luckily, the large-scale laboratory sample of the Netherlands Study of Depression and Anxiety (NESDA; $N = 2981$) provided us with the unique opportunity to test this hypothesis. In contrast with our expectations, the findings replicated prior findings of Greenwald and colleagues (2003) and the $D_{600}$-measure generally showed the best performance. This was not only the case for evaluation criteria that were identified by Greenwald and colleagues (2003) as most important, i.e. correlation with explicit equivalent and correlation with general response speed, but also for additional criteria that were included in the present study being predictive validity and test-retest reliability. An additional aim of the study was to explore six alternative IAT algorithms with the purpose to disentangle which ingredients of IAT algorithms contribute to the best performance. The results point to the conclusion that the success of the $D_{600}$-measure probably stems from the combination of ingredients being both the division by the pooled standard deviation and the inclusion of practice trials (and also the inclusion of error trials, but we did not specifically investigate this in our study). By this combination of ingredients, the $D_{600}$-measure seems to filter out the most meaningful

information, at least for a fixed block IAT design.

**Conclusion and future directions.** All in all, the outcomes lead to the conclusion that the $D_{600}$-measure is suitable for use in a laboratory setting and in within-subjects designs, when using an IAT in which the order of category combination is fixed. However, it seems a rash conclusion to completely discard all other IAT-algorithms on basis of these findings. The evaluation of scoring algorithms of the IAT is a complicated undertaking and several aspects could not be investigated in the present design. Unfortunately, the present design did not contain an outcome measure of spontaneous behaviours that could be used to assess the predictive validity of the various algorithms. It is often assumed that automatic associations are especially relevant for guiding more spontaneous kinds of behaviours (e.g., Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a; Spalding & Hardin, 1999). By using a self-report measure as outcome measure to test predictive validity, we run the risk of letting in the influence of explicit 'strategic processes' (e.g., Rothermund, Wentura & De Houwer, 2005; Wentura & Rothermund, 2007). Consequently, future research should also validate IAT-scoring algorithms against outcome measures of more spontaneous behaviours, preferably behaviours that are known to be driven primarily by automatic, but not by explicit processes. Furthermore, to arrive at more solid conclusions about which ingredients of IAT algorithms contribute to the best performance, future studies should vary more systematically different IAT ingredients. For example, factors such as use of SD correction, trial exclusion criteria, or latency transformations could be crossed systematically in factorial designs to obtain further insight into which combination of ingredients shows the best performance. Finally, we should be cautious considering properties of IAT-effects and IAT algorithms apart from the specific purpose for which the IAT is used. Consequently, replication in various samples, with different contents and study designs seem inevitable to improve our understanding of what the IAT is measuring, under which circumstances and which algorithm reflects these outcomes best.

### *2) Are dysfunctional automatic self-associations vulnerability factors for developing anxiety and/or depressive disorders?*

Current dual-process models of anxiety and depression stress the importance of dysfunctional automatic associations in the aetiology and maintenance of these disorders (e.g., Beevers, 2005; Ouimet, Gawronski & Dozois, 2009). In addition, negative schema's concerning the self are assumed to be involved in the onset and maintenance of psychopathology (e.g., Clark, Beck & Alford, 1999). Following these theoretical models and follow-

ing the design of Egloff and Schmukle (2002), we studied whether automatic self-anxious associations are a shared vulnerability factor across different anxiety disorders that set individuals at risk for developing anxiety disorders. Equivalently, we investigated whether automatic self-depressed associations can be seen as a pre-morbid vulnerability factor for depression.

As a first step, we tested in *Study 1* of *Chapter 3* whether patients with a current major depressive disorder and/or anxiety disorder are characterized by automatic self-anxious and self-depressed associations. We expected individuals suffering from anxiety and depressive disorders to show stronger automatic anxious and depressed self-associations than healthy controls. In line with our hypothesis, the outcomes showed that patients with an anxiety disorder displayed stronger automatic self-anxious associations than controls and that this did not differ between anxiety disorders. This finding is in line with prior cross-sectional studies that demonstrated that anxious individuals showed stronger dysfunctional automatic self-associations than non-anxious controls (Roefs et al., 2011) and with the finding that automatic self-associations were predictive of experimentally-provoked anxiety behaviours in unselected student samples in the laboratory (e.g., Asendorpf et al., 2002; Egloff & Schmukle, 2002; Spalding & Hardin, 1999). Similarly, patients with a major depressive disorder showed stronger automatic self-depressed associations than controls. Although the latter is in line with cognitive models of depression (Beevers, 2005, Clark et al., 1999), it differs from the typical findings in depression so far focusing on implicit self-esteem. Prior studies failed to find consistent evidence for negative implicit self-esteem in depressed patients. Our findings might be indicative for the hypothesis that implicit self-esteem somehow does not capture the automatic self-associations that are important in depression and that specific self-depressed associations may be more relevant for the cognitive vulnerability to depression. Currently, the data are being collected of the 6-year follow-up assessment of the NESDA in which we included a measure of implicit self-esteem. By studying implicit self-esteem in such a large sample of depressed patients, we hope to solve the puzzle whether lowered implicit self-esteem is still involved in depression, or whether specifically self-depressed associations are more relevant for the cognitive vulnerability to depression.

In line with the idea that dysfunctional automatic self-associations are pre-morbid vulnerability factors that set individuals at risk for developing anxiety and depression, we demonstrated that individuals with a depressive and/or anxiety disorder were characterized by dysfunctional automatic self-associations. A possible source for developing dysfunctional automatic associations might be childhood abuse. Germane to this, in a dif-

ferent study outside this thesis, we found that especially emotional abuse and emotional neglect were associated with stronger automatic self-depressed and self-anxious associations (van Harmelen et al., 2010). In addition, automatic dysfunctional associations partially mediated the relationship between emotional maltreatment and depressive/anxiety symptoms, independent of explicit self-beliefs. However, the cross-sectional design did not allow us to determine whether indeed emotional maltreatment leads to vulnerability for developing anxiety and depression via dysfunctional automatic self-associations. More information about the direction of such relationships might be obtained from prospective studies.

Consequently, in *Study 2* of *Chapter 5* we zoomed in further on automatic self-anxious associations as a vulnerability factor for developing anxiety disorders by testing their prognostic value for the onset of anxiety disorders between baseline and 2-year follow-up. If dysfunctional automatic self-anxious associations are indeed a pre-morbid vulnerability factor for anxiety disorders, they should be able to predict which individuals develop an anxiety disorder over time. We studied the onset of anxiety in three groups being healthy controls, depressed individuals, and individuals remitted from an anxiety disorder. Results showed that automatic self-anxious associations were indeed related to the onset of anxiety disorders, but only in individuals remitted from an anxiety disorder and in depressed individuals. Automatic self-anxious associations did not show predictive validity for first onset of anxiety disorders in healthy controls, refuting the hypothesis that automatic self-anxious associations are a pre-morbid vulnerability factor for developing anxiety disorders. These findings might imply that dysfunctional self-associations only establish on a more automatic level after someone has already suffered from an (anxiety) disorder. That is, dysfunctional automatic self-associations might not so much be critical for the first onset of anxiety, but represent a vulnerability factor for the recurrence of anxiety originating from the repeated activation of negative self-schemas during prior anxiety episodes.

In addition, we explored across all three groups whether the predictive validity of automatic self-anxious associations would be evident for the onset of all anxiety disorders or whether the predictive validity of self-anxious association might be restricted to particular anxiety disorders. Automatic self-anxious associations were significantly related to the onset of social anxiety disorder, panic disorder and agoraphobia, but not to generalized anxiety disorder (as compared to no anxiety disorder). Furthermore, automatic self-anxious associations showed independent predictive validity for the onset of more than one anxiety disorder including generalized anxiety disorder. How should we interpret these findings? The outcomes are in line with the idea that automatic self-anxious

associations are a shared vulnerability for anxiety disorders, irrespective of which specific anxiety disorder it concerns. In addition, automatic self-anxious associations seem to be related to the severity of anxiety symptoms, since stronger self-anxious associations independently predicted having more than one anxiety disorder, which is probably linked to having more anxiety symptoms. The finding that automatic self-anxious associations were not significantly related to the onset of generalized anxiety disorder is puzzling and I do not yet have a clear explanation for this outcome. Perhaps the different results stem form a difference in focus between generalized anxiety disorder and the other anxiety disorders. Generalized anxiety disorder seems to be focused on general threat from the outside world, whereas social anxiety disorder and panic disorder deal with more specific threats concerning the self. Clearly, future research will have to replicate these results to determine whether it concerns a robust phenomenon that deserves further interpretation.

**Conclusion and future directions.** We showed that individuals with a depressive and/or anxiety disorder were indeed characterized by dysfunctional automatic self-associations pointing to their possible role in the development of these disorders. However, automatic self-anxious associations did not (longitudinally) predict the first onset of anxiety disorders. Perhaps, dysfunctional self-associations only establish on a more automatic level after someone has already suffered from an (anxiety) disorder. To see whether automatic self-depressed associations might be a pre-morbid vulnerability factor for developing a first depressive episode, we are currently conducting a prospective study into the onset of depression (Kruijt et al., 2012).

### *3) Are dysfunctional automatic self-associations maintaining factors for anxiety and/or depressive disorders?*

The third question which I will discuss is whether we found evidence for the notion that dysfunctional automatic self-associations might be maintaining factors for anxiety and/or depressive disorders. In *Study 1* of *Chapter 5*, we studied the prognostic value of automatic self-associations for the natural course of depressive and anxiety symptoms over a two years period. Results showed that indeed automatic self-anxious associations were related to a reduced chance of remission from anxiety, whereas automatic self-depressed associations were related to a reduced chance of remission from depression. These findings support the hypothesis that relatively dysfunctional automatic associations towards the self are indeed involved in the maintenance of depressive and anxiety disorders over time. If dysfunctional automatic self-associations establish on a more automatic level

after someone has already suffered from anxiety or depression, these dysfunctional automatic biases might continuously trigger negative cognitive and affective responses, thereby creating a negative feedback loop as described by Beevers (2005). This view is also in line with a prospective study into post-traumatic stress disorder (PTSD) showing that strengthened automatic associations between 'self' and 'vulnerability' seemed a consequence rather than a cause of first-onset PTSD symptoms (Engelhard, Huijding, van den Hout & de Jong, 2007).

In addition, it seems likely that even if individuals recover from their anxiety or depressive disorder, they can still be characterized by dysfunctional automatic associations which make remitted individuals susceptible to relapse, the so-called scar hypothesis. This view is supported by our finding that automatic self-anxious associations related to relapse of anxiety in remitted individuals (*Study 2* of *Chapter 5*). In addition, in *Study 1* of *Chapter 3*, we examined whether dysfunctional automatic self-associations remained present after recovery of anxiety and/or depressive disorders. We examined whether individuals remitted from an anxiety disorder, a major depressive disorder or both were still characterized by enhanced automatic self-depressed/self-anxious associations. Results showed that remitted individuals indeed automatically associated themselves more strongly with anxious and/or depressed words than healthy controls, which is consistent with the view that dysfunctional self-associations may be a remaining scar of prior anxiety and depressive disorders.

Especially in the context of depression, it has been hypothesized that the repeated activation of negative self-schemas during a depressive episode results in an associative memory network where the self becomes increasingly linked to negative attributes. Over time, these chronic negative self-views are more easily activated, even by mild stress or mild negative mood states, and may turn into a chronic vulnerability factor for subsequent depressive episodes (e.g., Segal, Williams, Teasdale & Gemar, 1996). To further explore the scar hypothesis, we examined the relationship between the time since the last episode and the strength of automatic associations (see footnote of *Study 1*, *Chapter 3*). The more time had passed after having suffered from a depressive or anxiety disorder; the more positive were the automatic self-associations. In other words, it appeared that the automatic self-associations were least positive in the recently remitted participants. The data therefore suggest that the putative scar is not a fixed phenomenon, but some healing may occur when people are free of the disorder for a longer period of time. In addition, we examined in the remitted depressed group whether automatic self-depressed associations were related to the number of prior depressive episodes. Results showed

that automatic self-depressed associations were not especially pronounced in individuals with relatively many prior depressive episodes suggesting that the scar does not become worse after more frequent depressive episodes. However, it might be that not so much the number of depressive episodes, but the duration of time that individuals suffer from their symptoms is most important. Therefore, in a current study, we are examining whether a longer duration of depressive symptoms between baseline and 2-year follow-up predicts stronger automatic self-depressed associations during follow-up (Elgersma, Glashouwer, Bockting,Penninx & de Jong, 2012).

### *4) Are dysfunctional automatic associations specific characteristics of certain anxiety and/or depressive disorders or shared characteristics across several disorders?*

The last aim of this thesis was to get more insight into the disorder-specificity of dysfunctional automatic associations. The term disorder-specificity can be used in different ways, which complicates answering this fourth question. As a guideline to interpret the present results, I will look at three ways in which automatic associations might be disorder-specific. First, there could be dysfunctional automatic associations that relate to psychopathology in general ('level 1'). For example, global negative associations towards the self (implicit self-esteem) could set individuals at risk for developing psychopathological disorders and/or once someone has suffered from a disorder, this could lower their implicit self-esteem irrespective of which specific disorder it concerns. Secondly, there might be more specific dysfunctional associations that are related to a 'group' of disorders that share similar characteristics ('level 2'). For example, automatic self-anxious associations might contribute to the cognitive vulnerability of anxiety disorders in general and having an anxiety disorder might strengthen automatic associations between 'self' and 'anxious' irrespective of which specific anxiety disorder it concerns. Thirdly, there may be even more specific dysfunctional associations that play a role in particular psychopathological disorders ('level 3'). For instance, negative associations towards spiders might be important in spider phobia, but not in other (anxiety) disorders. In discussing the research outcomes related to this fourth question, I will mention each time which kind of disorder-specificity we might be looking at.

In *Study 1* of *Chapter 3* and in *Chapter 5*, we investigated the role of automatic self-anxious and self-depressed associations as vulnerability factors for anxiety and depressive disorders. Hereby, we are probably looking at 'level 2 of disorder-specificity', because we want to determine whether these dysfunctional associations are specifically related

to 'groups' of disorders that share similar characteristics, in this case anxiety disorders or depressive disorders. Several findings support the hypothesis that automatic self-anxious and self-depressed associations have disorder-specific effects for anxiety and depression, respectively. We found in *Study 1* of *Chapter 3* that patients were not only characterized by more negative automatic self-associations than healthy controls, but automatic self-associations also differed between patients. That is, patients with an anxiety disorder showed stronger automatic self-anxious associations than depressed patients and, similarly, patients with a major depression showed stronger automatic self-depressed associations than anxious patients. Participants with comorbid anxiety and major depressive disorder displayed both relatively strong automatic self-anxious and self-depressed associations. In addition, *Study 1* of *Chapter 5* showed that specifically automatic self-anxious associations were related to a reduced chance of remission from anxiety, whereas automatic self-depressed associations were related to a reduced chance of remission from depression. Finally, *Study 2* of *Chapter 5* showed that specifically automatic self-anxious associations and not automatic self-depressed associations predicted the future onset of anxiety disorders in remitted individuals and currently depressed individuals. In summary, these outcomes suggest that the effects of automatic self-anxious and self-depressed associations represent disorder-specific cognitive vulnerabilities to anxiety and depressive disorders.

However, we also found some evidence for effects of automatic self-associations that were not disorder-specific. Although anxious patients showed strongest automatic self-anxious associations and depressed patients showed strongest automatic self-depressed associations, we also found that patients in general showed more negative self-associations than the healthy control group, irrespective of disorder (*Study 1* of *Chapter 3*). In addition, in *Study 2* of *Chapter 5*, when we explored the predictive validity of automatic self-associations across different anxiety disorders, automatic self-depressed associations were found to be predictive of the onset of social phobia, agoraphobia, and the onset of more than one anxiety disorder. The latter findings suggest that automatic self-associations also contribute at least partially to a general cognitive vulnerability across different psychopathological disorders.

In *Study 1* of *Chapter 4*, we examined automatic blushing-associations in individuals with fear of blushing hypothesizing that dysfunctional blushing associations might contribute to the cognitive vulnerability to fear of blushing (disorder-specificity at 'level 3'). Our study was inspired by the assumption that fearful individuals overestimate the social costs of their blushing (Dijk, Voncken & de Jong, 2009). In line with this assumption, we wondered whether individuals with a fear of blushing associate blushing automatically

with social costs (Ouimet et al., 2009). The results indeed provided evidence that treatment-seeking individuals with fear of blushing showed stronger automatic associations between blushing and social costs than non-fearful controls. In addition, replicating prior findings from analogue samples (Dijk, de Jong, Müller & Boersma, 2010), individuals with fear of blushing showed more negative explicit expectations about the social costs of their blushing. Interestingly, explicit expectations and automatic associations were shown to be largely unrelated, attesting to the relevance of measuring both automatic and explicit blushing associations for fear of blushing. These results indicate that individuals with fear of blushing are indeed characterized by dysfunctional automatic associations with blushing, which is an extension of prior research that already demonstrated specific dysfunctional associations in specific phobia and social anxiety disorder (Roefs et al., 2011). However, we cannot be certain whether dysfunctional automatic blushing-associations are really specific for individuals with fear of blushing, because we did not include a clinical control group in our design.

In *Study 2* of *Chapter 4*, we examined automatic associations simultaneously at two different levels of disorder-specificity in the context of social anxiety disorder. The design of this study builds further on the cognitive model of social anxiety of Wells and Clark (1997), according to which socially anxious individuals interpret social situations as threatening, because of negative beliefs about their selves and dysfunctional assumptions about their social performance. Prior studies already showed that socially anxious individuals were characterized by dysfunctional automatic associations with respect to the self and social cues (de Jong, 2002; de Jong, Pasman, Kindt & van den Hout, 2001; de Hullu, de Jong, Sportel & Nauta, 2011; Tanner, Stopa & De Houwer, 2006). However, these studies compared analogue groups of high socially anxious individuals with low socially anxious individuals and did not include clinical-control groups. Therefore, we decided to study the two types of automatic associations that seem most relevant to social anxiety disorder, implicit self-esteem and automatic associations with social cues, in a clinical sample. Furthermore, we included a clinical control group of panic disorder patients to really 'check' the disorder-specificity of these dysfunctional associations.

We expected that automatic associations with social cues would be more dysfunctional in the socially anxious group than in both control groups (disorder-specificity at 'level 3'). In addition, we expected that the socially anxious group would have lowered implicit self-esteem compared to controls. However, for the panic disorder group we had no clear expectations with respect to implicit self-esteem. Implicit self-esteem might be lowered in the panic disorder group, in which case it would be disorder-specific at 'level 1 or 2', but

it could also be that implicit self-esteem is specifically lowered in social anxiety patients and normal in panic disorder patients (disorder-specificity at 'level 3'). In line with prior findings and with our hypothesis, the results showed that socially anxious individuals were indeed characterized by more negative automatic associations with social cues than both non-clinical controls and panic disorder patients. This indicates that dysfunctional automatic associations with social cues seem to be specific for social anxiety disorder. In addition, socially anxious individuals displayed a lowered implicit self-esteem than non-clinical controls, but the panic disorder group scored in between these groups.

The latter could mean that lowered implicit self-esteem can be seen as a more general characteristic of anxiety patients ('level 2') or psychopathology in general ('level 1'), but at the same time is particularly involved in social anxiety disorder ('level 3'). Maybe individuals who suffer from an anxiety disorder generally have a relatively low implicit self-esteem, reflecting a vulnerability to experience anxiety over a variety of situations. But because concerns about the self are a central theme in social anxiety disorder, it is not surprising that these individuals score even lower on implicit self-esteem. However, it is important to keep in mind that the differences on implicit self-esteem neither between panic disorder patients and social anxiety disorder patients nor between panic disorder patients and non-anxious controls reached statistical significance.

**Conclusion.** In conclusion, several studies in this thesis demonstrated disorder-specificity of dysfunctional automatic associations for anxiety disorders and depressive disorders ('level 2') as well as for social anxiety disorder and fear of blushing in particular ('level 3'). However, the results also suggest that disorder-specificity is not an all-or-nothing affair and some types of dysfunctional automatic associations might simultaneously be involved in different levels of disorder-specificity.

### *Suicidal ideation*

In *Study 2* of *Chapter 3*, we took a step aside from the main questions of this thesis and explored whether automatic self-associations could also help to improve our understanding of suicidal ideation, a psychopathological symptom that occurs both in depressive disorders and in anxiety disorders. We were inspired by the findings that automatic self-associations seem to display predictive validity specifically for more spontaneous, uncontrollable kind of behaviours such as autonomic responding and nonverbal behaviours (e.g., Asendorpf et al., 2002; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a; Spalding & Hardin, 1999). Building on this, we hypothesized that dysfunctional automatic self-associations might also contribute to the onset and maintenance of suicidal ideation, since

suicidal patients often report difficulties in controlling their suicidal thoughts and preventing them from repetitively entering their awareness. Because not only depression, but also anxiety has been linked to suicidal ideation (e.g., Ellis, 2006, Norton, Temple & Pettit, 2008; Sareen et al., 2005), we hypothesized that automatic self-depressed associations as well as automatic self-anxious associations might relate to suicidal ideation. In line with what we expected, the results showed that both kinds of automatic associations were independently related to suicidal ideation and past suicide attempt. Although the main-effects of automatic self-associations did not explain additional variance over and above explicit self-beliefs, the interactions between automatic self-depressed (anxious) associations and explicit self-depressed (anxious) beliefs did. That is, the probability of having suicidal thoughts was especially high for individuals who had depressed (anxious) self-associations both on an automatic level and an explicit level. Perhaps individuals with relatively positive explicit beliefs about themselves (e.g., "I am elated" or "I am calm") are better able and/or stronger motivated to neutralize or correct the influence of automatic negative self-associations than individuals with more negative explicit self-evaluations (cf. de Jong, van den Hout, Rietbroek & Huijding, 2003). In other words, in individuals with negative self-associations on both automatic and explicit levels this correction might not take place, thereby enhancing the risk of eliciting suicidal thoughts. This would be in line with the dual process model of Beevers (2005) that states that even if propositional processes are being triggered, this does not necessarily imply that dysfunctional automatic associations are corrected. Only when the propositional processes lead to an adjustment of the associative bias, the influence of dysfunctional associations on cognitive and affective responses can be disrupted.

## Considerations

### *Conceptualization of explicit self-beliefs*

Measures of explicit beliefs equivalent to the automatic associations were included in several of the study designs. The outcomes showed that group differences on explicit beliefs were typically in similar directions as group differences on automatic associations, i.e. anxious patients showed stronger explicit self-anxious beliefs than depressed patients and controls; depressed patients showed stronger explicit self-depressed beliefs than anxious patients and controls; and individuals with fear of blushing showed more negative explicit beliefs about the social costs of their blushing. In addition, the predictive validity of explicit self-beliefs for suicidal ideation, maintenance of depressive and anxiety disorders and onset of anxiety disorders was generally in the same direction as the predictive valid-

ity of automatic self-associations. However, effects found for explicit beliefs were much larger than for automatic associations, and often automatic associations did not show independent predictive validity for the outcome variables over and above explicit beliefs. I will further discuss the latter issues below.

One could argue that the present measures of explicit self-beliefs were no 'official', well-established measurements. However, we had several reasons to include these particular measures, instead of using existing (trait) measures. We wanted to include measures that were as similar as possible to the concepts that were included in the automatic self-associations to be able to compare the outcomes of both types of measurements. Since existing measures of self-concepts are usually questionnaires that cover a much broader spectrum of characteristics, we had to compose the measures self-anxious and self-depressed beliefs ourselves. This kind of explicit ratings is often used in psychology research (cf., Hoffman, Gawronski, Geschwendner, Le & Schmitt, 2005), because it is assumed to reduce the chance that a divergence of automatic and explicit measures only occurs because different types of stimuli are used. Germane to this, it was shown that the correlation between automatic and explicit measures greatly increased when the measurement procedures were made as similar as possible, by using the same response metric and same type of stimuli in both measurement procedures (Payne, Burkley & Stokes, 2008). Finally, the explicit ratings displayed high internal consistencies in our sample ($\alpha$ = .94/.95) and discriminated clearly between diagnostic groups (*Study 1* of *Chapter 3*), which suggests adequate psychometric properties.

Although there were good reasons to include these particular measures for explicit self-beliefs, it still brings up the question how we should best conceptualize these measures compared to symptom measures of anxiety and depression and compared to trait characteristics that have already been shown to be involved in anxiety and depressive disorders. On the one hand, anxious and depressed self-views seem to overlap with anxiety and depressive symptoms, because they moderately correlate with symptom measures and as soon as explicit self-beliefs were entered together with symptom measures in the statistical models, the predictive validity of the symptom measures disappeared (*Chapter 5*). On the other hand, explicit self-beliefs showed predictive validity over and above these symptom measures. The latter could imply that explicit self-beliefs also partly reflect more stable, trait-like beliefs individuals have regarding themselves, next to temporal self-views that are influenced by/or part of momentary symptom severity. That way, explicit self-anxious and self-depressed beliefs seem to be similar to trait anxiety and trait depression.

We are not aware of prior longitudinal studies that demonstrated the influence of trait

anxiety or trait depression on the course/onset of anxiety and depression. However, other studies have shown that higher anxiety sensitivity is related to an unfavourable course of panic disorder (e.g., Chavira et al., 2009; Ehlers, 1995; Pérez Benítez et al., 2009; Pollack, Otto, Rosenbaum & Sachs, 1990; Schmidt & Bates, 2003) and higher neuroticism is related to an unfavourable course of depressive disorders (e.g., Brown & Rosellini, 2011; Hayden & Klein, 2001; Rhebergen et al., 2009; Steunbergen, Beekman, Deeg & Kerkhof, 2010) and anxiety disorders (e.g., Chavira et al., 2009; De Beurs, Beekman, Deeg, van Dyck & Tilburg, 2000). While neuroticism refers to a general tendency to experience negative emotional states, explicit self-anxious and self-depressed beliefs might reflect more specific anxious or depressive response tendencies. In line with this idea, the effects of explicit self-associations were indeed found to be (at least partially) disorder-specific. In addition, although we found that explicit self-beliefs were strongly related to neuroticism, self-anxious beliefs also showed predictive validity over and above neuroticism for the onset of anxiety disorders. Furthermore, anxiety sensitivity and self-anxious beliefs might be related as well, but both constructs also seem to differ in an important respect. When a person has the tendency to respond anxiously to a variety of situations (self-anxious beliefs) it does not automatically imply that this person also fears the symptoms that are related to anxiety (anxiety sensitivity). Anxiety sensitivity has been included in the NESDA design, which provides the opportunity to investigate its relationship with explicit self-anxious beliefs in future work.

### *Small effects and no independent predictive validity*

One drawback that is repeatedly being raised with respect to the research outcomes of this thesis is that the explained variances of the automatic associations are typically small, especially in comparison to the effect sizes of the explicit beliefs. In addition, reviewers often point out that dysfunctional automatic associations showed no independent predictive validity over and above explicit self-beliefs for the onset and course of anxiety and depression. These observations elicit questions about the clinical meaningfulness of these findings. Since anxiety and depression form a substantial problem for our society, and large numbers of patients still fail to profit sufficiently from existing treatments (e.g., Hansen, Lambert & Forman, 2002), it seems crucial to look for underlying mechanisms that lead to the persistence of these disorders, which was precisely the goal of this thesis. In addition, as I will discuss at the end of this chapter, the research area of which this thesis is part of, might be fruitful in inspiring new ways towards improving current interventions for anxiety and depressive disorders. Although these arguments support the

clinical meaningfulness of these findings, I would not want to pretend that effect sizes or independent predictive validity do not matter. Therefore, I will discuss three issues that might have contributed to the small effects of automatic associations and the absence of independent predictive validity.

**Methodological overlap.** First of all, there may be differences in methodological overlap between independent measures and outcome measures. It seems that the methodology which was used to measure explicit beliefs is much more similar to the methodology of the outcome measures than the methodology which was used to assess automatic associations. The explicit beliefs and the outcome measures of anxiety and depressive symptoms both asked individuals directly for the construct of interest. Likewise, shared factors such as introspective ability or social desirability might have contributed to a greater correlation between both variables. In contrast, the Implicit Associations Test measures the construct of interest indirectly by means of reaction times in a computer task. Taking this into account makes it less surprising that the explicit beliefs show greater predictive validity than the automatic associations.

**Outcome measures.** The latter point brings us to a possible limitation of our study designs. We consistently used clinical interviews to assess the outcome measures of anxiety disorders and depression. However, as explained in the introduction, large parts of anxious and depressive symptoms seem to occur spontaneously, without the ability to control it, or even outside of awareness. Interviews might capture only part of these spontaneous anxious/depressive symptoms. In addition, these spontaneous behaviours will probably co-occur with other symptoms that are captured with the interviews, which might explain why we found a relationship between dysfunctional automatic associations and the outcome measures. However, self-report measures or interviews probably are not an ideal way to measure automatic behaviours. Consequently, most likely we also missed certain aspects of anxiety and depressive symptoms. Since automatic associations specifically seem to display predictive validity for these more spontaneous, uncontrollable kinds of behaviours (e.g., Asendorpf et al., 2002; Egloff & Schmukle, 2002; Huijding & de Jong, 2006a; Spalding & Hardin, 1999), again it is not surprising that the automatic associations showed relatively small effects. Turning it around, it is quite exciting that we were able to show repeatedly that dysfunctional automatic associations related to anxiety and depression, not only at baseline, but also over time. In some analyses they even predicted unique parts of the variance of these disorders.

**Dual-process model.** The third point I would like to mention goes into the fact that automatic associations did not consistently show predictive validity over and above explicit

beliefs. If we look at this finding from a dual process perspective, it actually appears to be quite logical. As extensively discussed in the introduction, associative networks form the basis for propositional processing, which means that these automatic associations provide the input/information used for reasoning. Although sometimes automatic associations are discarded as valid basis for behavioural decision-making, most of the time both systems are thought to work synchronously. Consequently, it could well be that the effects of automatic associations 'run through' explicit beliefs, and, entering both in the analyses can have taken away the statistical significance of automatic associations in predicting anxiety and depressive symptoms. However, it should be noted that it is not so self-evident that people who report particular cognitions at an explicit level are also characterized by conceptually similar automatic associations. As has been mentioned, when it comes to self-esteem in depression, there is ample evidence that people suffering from depression show low explicit self-esteem on self-report measures as the RSES, whereas the majority of studies did not find evidence for lowered self-esteem in depression at the implicit level (De Raedt, Schacht, Franck & De Houwer, 2006; Franck, De Raedt & De Houwer, 2007, 2008; Gemar, Segal, Sagrati & Kennedy, 2001; Valiente et al., 2011). In a similar vein, research in the context of sexual pain demonstrated that women with dyspareunia showed similarly positive automatic associations with sexual intercourse as healthy controls, whereas at the explicit level these women showed a negative appreciation (Brauer, de Jong, Huijding, Laan & ter Kuile, 2009), suggesting that for this type of complaints explicit beliefs are more important than automatically elicited affective associations. From such a perspective, the present thesis represents an important step in evaluating the potential importance of automatic associations in anxiety and depressive disorders.

Despite these methodological and theoretical issues, it could still be that automatic associations co-exist with having anxiety or depressive symptoms without having any causal influence on them. In other words, dysfunctional automatic associations might be an artefact of symptom severity and likewise, do not have any clinical meaningfulness. In line with such view, remitted individuals scored in between currently symptomatic people and healthy controls both on automatic self-associations and symptom severity. In addition, relationships between automatic self-associations and onset/course variables disappeared when symptom measures were included in the statistical models. Although these outcomes could imply that automatic self-associations fluctuate with the symptom levels, this is not the only possible explanation. Other studies already showed that automatic self-associations might be causally related to anxiety symptoms. For instance, a study measuring automatic self-associations several times over the course of treatment

for panic disorder showed that positive changes in automatic associations were found to predict greater symptom reduction, but not the other way around (Teachman, Marker & Smith-Janik, 2008). In addition, recently, an experimental study demonstrated that training implicit associations in the context of social anxiety had an influence on anxious behaviour (Clerkin & Teachman, 2010). In that study high socially anxious students were trained in a computer task in which their own photographs were paired with pictures of others' positive facial expressions. The outcomes showed that compared to two control trainings, the individuals who received the positive training had less negative associations between 'self' and 'rejected'. Moreover, the positive training group more often completed an impromptu speech. Both studies are in contrast with the idea that dysfunctional automatic associations are mere epiphenomena of symptom severity.

### *What is automaticity?*

After 150 pages talking about automatic associations, it might seem a bit late to ask what automaticity actually is. The reason to discuss it now, is that the nature of automaticity contains a highly complex issue, which is primarily beyond the scope of this thesis. Nevertheless, I will highlight some points that seem especially relevant for this area of research.

The Implicit Association Test belongs to a class of measurement instruments that is often referred to as 'implicit measures'. However, in the literature, there exists quite some conceptual vagueness with respect to the use of the term *implicit*, as has extensively been discussed by Jan De Houwer (e.g., 2006). First of all, De Houwer points out that it is important to distinguish between a measurement procedure and the outcomes of the procedure. When referring to a measurement procedure, it does not make much sense to use the word implicit. It seems more logical to use the terms *indirect* or *direct*. Direct reflects a measurement procedure in which individuals are enquired directly about the construct of interest, whereas indirect measures derive the construct of interest from behaviour. De Houwer (2005) describes the name letter effect (Nuttin, 1985) as a nice example of this distinction. In this task participants have to rate to what extent they like each letter of the alphabet. If a researcher would be interested in attitudes towards letters, this measurement procedure could be considered direct. However, researchers typically use this task as a measure for self-esteem by comparing ratings of the letters of someone's name with ratings of other letters of the alphabet, in which case the procedure can be classified as an indirect measure.

According to De Houwer (2006), the term implicit should be solely reserved for the outcomes of measurement procedures. However, he also brings forward that this might

not be the best term, because it narrows the scope too much to whether or not outcomes are (un)conscious, whereas the constructs we are interested in might be multidimensional in nature. Instead, the term *automatic* might be more appropriate to cover a broader set of features besides awareness, such as unintentional, efficient or fast (De Houwer, Teige-Mocigemba, Spruyt & Moors, 2009). These different features of automaticity might not necessarily co-occur, that is, a process/cognition/behaviour could be automatic in one sense, but not in the other. From this point of view, the term 'adaptive unconscious' introduced in the first paragraph of the introduction might have been somewhat misleading, since it suggests that automatic processes are typically taking place outside awareness, which is only one potential aspect of automaticity.

What happens if we apply these classifications to the Implicit Association Test? It seems that the measurement procedure that is used in the IAT is indirect, because we are not interested in individuals' reaction times sec, but use the reaction times to infer the construct of interest, i.e. automatic associations. It is more complicated to determine whether the outcomes of the IAT can be considered automatic, since not much research has been aimed at unravelling which features of automaticity are reflected by IAT-effects (De Houwer et al., 2009). The little research that has been conducted on this topic suggests that at least part of the participants is aware of what is being measured with an IAT and how it is measured. In addition, it seems that individuals to a certain extent can consciously control the outcomes of an IAT, although this is much harder than for many traditional measures. Furthermore, it was shown that predictive validity of self-report measures, but not of IATs, was reduced when research topics were socially sensitive (Greenwald, Poehlman, Uhlmann & Banaji, 2009) which suggests that compared to self-report measures, the IAT might measure outcomes more independent of whether individuals have the explicit goal or intention to reveal these outcomes. Finally, it seems important to mention that more research has been addressed towards the broader question which underlying cognitive processes contribute to IAT-effects. These studies show that an IAT-effect is certainly not a 'process pure' measure in the sense that 100% of the IAT-effect is determined by automatic associations of interest; and multiple, qualitatively different processes might contribute to IAT-effects (e.g., Conrey, Sherman, Gawronski, Hugenberg & Groom, 2005; Fiedler, Messner & Bluemke, 2006).

**Conclusion and future directions.** It carries too far to give a detailed account of the (methodological) issues surrounding automaticity and the validity of the IAT. Two issues seem to be most crucial for a full appreciation of the outcomes of this thesis. Firstly, automaticity is not a unitary construct, but refers to a broad set of features which do not neces-

sarily co-occur. Up to now, it is still largely unknown which features of automaticity apply to the outcomes of the IAT, which seems an important topic for future research in this area. Secondly, the IAT is, just like other measures, not 'process pure' and different processes are likely to contribute to IAT-effects. Therefore, future research could focus more thoroughly on disentangling specific components that make up an IAT-effect, for instance, by statistically decomposing and/or experimentally manipulating key cognitive components involved in the IAT-effect (cf. Conrey et al., 2005; Klauer, Voss, Schmitz & Teige-Mocigemba, 2007). Relatedly, it seems important to examine how these components are reflected in different IAT algorithms (e.g., Schmitz, Teige-Mocigemba, Voss & Klauer, 2012). Such studies not only will shed a light on which automatic features characterize the IAT-effect and/or which cognitive processes underlie the IAT-effect, but more importantly, will lead towards a better understanding of the outcome measures that we want to predict with the IAT.

## Clinical implications

Last, but not least, I will discuss the clinical implications of these findings. If dysfunctional automatic associations are important underlying factors for the cognitive vulnerability to depression and anxiety disorders, what would this mean for clinical practice?

### *Malleability of automatic associations due to treatment*

As a starting point, one might wonder to what extent existing interventions for anxiety disorders and depression already successfully target dysfunctional automatic processes. Up to now, only a few studies have been conducted focussing on this question, mainly in the context of cognitive behavioural therapy (CBT). The first study on this topic showed that negative associations towards spiders decreased after CBT (Teachman & Woody, 2003). However, this study did not include a waitlist control group, making it impossible to distinguish between treatment effects and learning effects. Two studies that did include a waitlist control group showed similar decreases in dysfunctional associations from pretest to post-test for the treatment group and the control group (Huijding & de Jong, 2007, 2009), suggesting that the reduction in automatic spider-associations was the result of learning effects rather than treatment effects. However, recently, another controlled study found opposite results and showed that automatic spider-associations did normalize under the influence of CBT (Reinecke, Soltau, Hoyer, Becker & Rinck, 2012). Although the contrasting findings of these controlled studies seem puzzling, some methodological differences between the studies might account for the different outcomes. First of all, partici-

pants in the studies of Huijding and de Jong (2007, 2009) received one session individual CBT, whereas the study of Reinecke and colleagues (2012) administered three sessions of CBT in small groups with homework assignments in between the sessions. In addition, post-assessment took place immediately after treatment in the studies of Huijding and de Jong, but one to three days after treatment in the study of Reinecke and colleagues. It might be that the adjustment of dysfunctional automatic associations in spider phobia takes some time to consolidate, in which case the study design of Reinecke and colleagues had a greater chance of finding changes in automatic associations. In the context of social anxiety, only one study has been conducted showing that automatic self-anxious associations seemed to reduce in socially anxious students following 4 sessions of group-based CBT (Gamer, Schmukle, Luka-Krausgrill & Egloff, 2008). However, again this study did not include a waitlist control group. Finally, in the context of panic disorder, it was shown that dysfunctional automatic self-associations changed over the course of CBT and positive changes in automatic self-associations predicted greater symptom reduction, but not the other way around (Teachman, Marker & Smith-Janik, 2008).

The effect of medication on associative biases is largely unknown. As far as I know, only one study has been conducted in the context of suicidal ideation in depressed individuals testing the influence of a single dose of intravenous ketamine on automatic associations towards *escape* and *death* (Price, Nock, Charney & Mathew, 2009). Results showed that associations towards escape seemed to decrease after the ketamine infusion (associations with *stay* were strengthened), but no significant effects were found on death associations. These results give the impression that automatic biases might be changed following drug treatment. However, considering the small sample size of this study ($n$ = 10) and the absence of a control group, this might be a premature conclusion and more research in this area is necessary.

In summary, although the evidence is still rather small, existing CBT interventions might be able to change dysfunctional automatic associations. However, repeated practicing and homework assignments seem crucial elements of treatment to facilitate the adjustment of associative biases. These first results seem promising, although we still have to see whether the findings generalize to other anxiety disorders and depressive disorders, as well as to other types of dysfunctional automatic associations. Consequently, it would be a great step forward if future randomized controlled trials for anxiety and depression more and more include measurements that tap into associative networks, next to the self-report measurements that are typically used. In addition, (successfully) treated patients should be followed-up over time to see whether residual dysfunctional automatic associa-

tions after treatment are predictive for the recurrence of symptoms.

### *Implications for current interventions*

Although we still know little about the influence of CBT on dysfunctional automatic associations, some implications seem important to take into account in interventions for anxiety and depression. As already described in the introduction, associative networks become activated irrespective of whether these associations are considered as valid or invalid (Gawronski & Bodenhausen, 2006). In addition, associative networks exist of simple links between concepts without relationships between them are specified. This might have implications for the use of negations in cognitive therapy. When formulating and practicing alternative thoughts (also referred to as 'rational' or 'helpful' thoughts), it is important to pay attention to the possible effects of these thoughts on the associative system. Thoughts like "I'm not worthless" or "I'm not a loser" might actually strengthen associations between *me* and *worthless* and *me* and *loser*, because these concepts become activated in the associative network. Instead, it might be more effective practicing thoughts like "I'm a valuable person", so *me* becomes linked to *valuable*. First evidence that negation indeed can have negative effects comes from a study in the context of spider phobia (Ouimet, Barber & Radomsky, 2012). Ouimet and colleagues tested in an experimental paradigm whether repeated negation of spider-fear associations (e.g., "a spider is not dangerous") would lead to an attention bias for spider stimuli and higher levels of spider fear. One group of undergraduates was trained to repeatedly press a button to say "yes" to inconsistent stimulus-emotion pairings (e.g., spider + calm; reappraise condition). The other group was trained to press a button to say "no" to consistent stimulus-emotion pairings (e.g., spider + scary; negate condition). Although no significant group differences were found for attention bias, the study demonstrated that participants in the negate condition showed higher levels of self-reported spider fear than participants in the reappraise condition. These findings provide support for the recommendation to encourage patients to reappraise objects, situations and their selves in positive ways rather than negating existing maladaptive associations.

There may be other side-effects of certain CBT interventions on associative biases. For example, talking about pros and cons of dysfunctional cognitions, which is typically part of cognitive therapy, might increase the accessibility of negative associations. In line with this hypothesis, a recent study into prevention of social anxiety in adolescents showed that the adolescents that received preventive CBT displayed stronger dysfunctional automatic associations afterwards than adolescents in the control group, which did not

receive preventive treatment (de Hullu, Sportel, de Jong & Nauta, 2012). A third group that received a computer training in which merely positive material was presented did show an improvement of dysfunctional automatic associations. On the one hand, these findings seem to promote a focus on positive/healthy information to decrease automatic biases. On the other hand, it has also been argued that positive/healthy information can only become ingrained in dysfunctional associative networks, when this network is really activated, for example by exposure towards negative/feared information. It might be that exactly exposure teaches depressed and anxious individuals how to deal with difficulties and negative feelings and to develop more functional (automatic) associations. This latter view is for instance being adopted in exposure-based cognitive therapy for chronic depression (EBCT; e.g., Hayes et al., 2007). In EBCT, after a phase of *stress management*, patients are encouraged to activate their depressive network of negative thoughts, affect and behaviours in specific exercises during the *exposure activation* phase. The goal of this phase is to explore and question the negative beliefs, generate additional, inconsistent information and increase inconsistency and dissonance, in other words, the depressive network is given a 'shaking'. In the final *consolidation and positive growth* phase, new, more adaptive associations are strengthened to compete with the old, maladaptive patterns.

Although strengthening corrective propositional processes in cognitive therapy might help to 'escape' from bad influences of negative associative biases (cf. Beevers, 2005), we should take into account the possibility that sometimes patients might not be characterized by dysfunctional automatic associations. In such situations, it could even be unfavourable to train reflective processes. Some studies showed that relying too much on the reflective system can alienate individuals from natural, adequate ways of reacting. For example, in social psychology it was shown that merely thinking about a decision does not necessarily improve the quality of the decision, such as picking the most tasteful strawberry jam (Wilson & Schooler, 1991), or choosing the best place to live (e.g., Nordgren, Bos & Dijksterhuis, 2011). With respect to psychopathology, this could imply that 'too much thinking' can undermine healthy automatic processes, such as automatic self-favouring tendencies, which actually help individuals to cope with negative experiences. Once more, this underlines the importance of carefully testing whether patients are indeed characterized by dysfunctional automatic associations. In addition, it stresses the necessity to investigate which interventions might be effective for whom instead of searching for one remedy that 'fits all'.

Finally, it seems important to pay attention to the conditions under which reflective and associative systems operate. Working memory capacity seems an important modera-

tor that determines whether individuals are able to use their reflective system (e.g., Fazio & Towles-Schwen, 1999). When working memory is occupied, individuals are more likely to act on their automatic associations. In support of this idea, in the context of addiction it was demonstrated that positive-arousal associations predicted alcohol use after one month more strongly in students with lower levels of working memory capacity (Thush et al., 2008). Consequently, it might be effective to help patients to train their working memory functions (e.g., Siegle, Ghinassi & Thase, 2007) as well as to learn them to recognize under which circumstances their working memory is likely to be 'occupied' which increases their vulnerability to dysfunctional automatic associations in these situations.

### *Expanding current interventions*

Finally, it seems worthwhile to develop new interventions that more directly target dysfunctional automatic associations. Some studies using classical conditioning procedures showed that enhancing positive self-associations might have a positive influence on behaviours (Baccus, Baldwin & Packer, 2004; Dijksterhuis, 2004) and symptoms (Clerkin & Teachman, 2011; see further above under *Considerations*). Such interventions that focus directly on the adjustment of automatic biases could form a welcome expansion of current treatments for anxiety and depression. These kinds of tasks are relatively easy to deliver and patients can even practice at home, which makes it possible to include such interventions in e-mental healthcare. An additional advantage of these computer tasks is that it is a relatively cheap treatment which is a positive feature considering the high prevalence of anxiety and depressive disorders and the accompanying challenge for our healthcare system (Kazdin & Blace, 2011). When incorporated in existing interventions such as CBT, cognitive bias modification tasks may help to lower the threshold to engage in real-life exercises. There also might be a role for these tasks as relapse prevention tool, when administered on a regular basis after treatment. However, either way, we should remain careful that individuals do not receive interventions that produce only minimal effects, since this increases the risk of demoralisation with possible poor treatment prognosis. From this perspective, at present cognitive bias modification tasks generally do not seem to be suitable (yet) as standalone treatments, although this of course might change in the (near) future. Hopefully, this line of interventions/research will prove to be a fruitful step forward towards a better understanding of anxiety and depressive disorders, and a further improvement of existing treatment options for these persistent, recurring, and highly invalidating disorders.

# Samenvatting

Deze samenvatting is deels gebaseerd op:

Glashouwer, K. A., & de Jong, P. J. (2009). Het 'onbewuste' in de psychopathologie. *Tijdschrift voor Neuropsychiatrie en Gedragsneurologie, 8*, 37-40.
Glashouwer, K. A., & de Jong, P. J. (2008). Impliciete persoonlijkheidstrekken en psychopathologie. Achtergrond, huidige inzichten en perspectieven. *Gedragstherapie, 41*, 120-134.

De volgende situatie doet zich voor. Op een internationaal congres staat een spreker op het punt een presentatie te geven voor zijn vakgenoten. Op het moment dat hij het podium betreedt, lijkt het alsof zijn lichaam plotseling zijn eigen gang gaat: zijn handen worden klam, een misselijk gevoel komt opzetten, zijn hartslag gaat omhoog en wanneer hij wat water in een glas wil schenken, merkt hij dat zijn handen lichtelijk trillen. Gelukkig weet hij zich te vermannen, hij richt de aandacht op het publiek in de zaal en op wat hij hen wil vertellen. Na een paar minuten merkt hij dat de spanning is gezakt.

Een dergelijke situatie zal voor velen herkenbaar zijn. Toch is er hier iets geks aan de hand. Want hoewel de spreker zich waarschijnlijk goed heeft voorbereid op de presentatie en weet dat hij het tot een goed einde zal brengen, ervaart hij een nerveus gevoel en angstige reacties zonder dat hij dat eigenlijk wil. In de praktijk blijken de meeste mensen gelukkig in staat om vervolgens zulke gevoelens en lichamelijke sensaties te negeren en de presentatie te voltooien. Voor sommigen is dit echter moeilijk, zo niet onmogelijk. Iemand met een sociale angststoornis zou bijvoorbeeld een presentatie doorstaan met een intense angst, of zoveel mogelijk proberen dergelijke situaties te vermijden.

## Automatische associaties in angst en depressie

Hoe kunnen we de reacties van deze spreker verklaren? Wellicht spelen automatische geheugenprocessen hier een onderliggende rol. Automatische associaties worden geactiveerd zonder dat iemand daar direct invloed op heeft kunnen oefenen. Het woord associatie verwijst naar het verband tussen twee concepten in het geheugen. Zo associëren de meeste mensen het concept *bakker* met *brood* en iemand met sociale angst zou *feestje* kunnen associëren met *uitlachen* of *afgang*. Een associatie wordt automatisch genoemd wanneer deze direct actief wordt in een bepaalde situatie, zonder dat iemand dit kan tegenhouden. Automatische associaties verschillen van gedachten die worden gevormd door de afweging van verschillende stellingen. Bij een dergelijke afweging is het van belang of iemand deze stellingen als waar of onwaar beschouwt. Dat laatste is een belangrijk verschil tussen expliciete gedachten en automatische associaties. Automatische associaties worden namelijk direct geactiveerd, of iemand nu wel of niet denkt dat deze associaties kloppen. Expliciete gedachten worden verondersteld vooral invloed uit te oefenen op meer gecontroleerde gedragingen, terwijl automatische associaties leiden tot meer spontaan, ongecontroleerd gedrag, zoals fysiologische reacties of non-verbaal gedrag (zie bijv. Fazio & Towles-Schwen, 1999; Gawronski & Bodenhausen, 2006). Omdat het hierbij als het ware om twee routes gaat van cognities naar gedrag, wordt deze theorie ook wel 'het duale proces model' genoemd. Als we terug gaan naar het voorbeeld, dan zou

het kunnen zijn dat deze spreker automatisch het geven van de presentatie associeerde met dreiging, maar dat hij deze associaties kon negeren door expliciete gedachten als "ik heb de presentatie goed voorbereid" of "ik ben in staat deze presentatie te geven".

Anders dan in dit voorbeeld zijn spontane gedragingen over het algemeen heel handig en dragen ze bij aan je overlevingskansen. Als er op straat een auto op je afrijdt, is het maar goed dat je direct aan de kant springt en hier niet eerst diep over na hoeft te denken. Jammer genoeg kan het in sommige gevallen juist onhandig en zelfs vervelend zijn om automatisch op een bepaalde manier te reageren. Dit laatste zou wel eens aan de hand kunnen zijn bij angst en depressie. Vaak weten angstige en depressieve patiënten wel dat de werkelijkheid eigenlijk niet zo somber, dreigend of onverdraaglijk is als het lijkt. Toch komen die sombere, angstige of wanhopige gevoelens en gedragingen steeds maar weer opzetten. Het zou dus kunnen dat disfunctionele automatische associaties een belangrijke rol spelen in angststoornissen en depressie. Het bestuderen van deze disfunctionele automatische associaties kan daarom helpen beter te begrijpen hoe depressie en angststoornissen ontstaan en waarom deze stoornissen zo hardnekkig zijn.

Eerder onderzoek heeft al laten zien dat angstige mensen vaak sterkere disfunctionele associaties hebben dan niet-angstige mensen (zie: Roefs et al., 2011 voor een overzicht). Verder is gebleken dat sterkere automatische associaties tussen *ik* en *angstig* voorspellend waren voor meer spontane angstgedragingen, bijvoorbeeld nerveuze bewegingen of oogknipperen tijdens het geven van een toespraak (Egloff & Schmukle, 2002). Hierop voortbordurend dachten we dat deze automatische zelf-angst associaties mensen mogelijk kwetsbaar maken voor het krijgen en houden van een angststoornis, en vergelijkbaar, dat automatische zelf-depressie associaties het risico vergroten dat mensen een depressie ontwikkelen en houden (met zelf-depressie associaties bedoelen we dat je jezelf direct koppelt aan kenmerken als 'nutteloos', 'ongeschikt' of 'negatief'). Tot nu toe is er vooral onderzoek naar dit onderwerp gedaan onder studenten en nog maar weinig onder patiënten, laat staan onder patiënten met verschillende stoornissen tegelijkertijd. Hierdoor weten we niet zeker of de verschillen die gevonden werden nu speciaal bij een bepaalde angst- of depressieve stoornis horen, of een meer algemeen kenmerk zijn van psychopathologie. Ten slotte is er nog maar weinig onderzoek gedaan naar de voorspellende waarde van disfunctionele automatische associaties over de loop van de tijd, met als gevolg dat we niet weten of ze ook voorspellen wie er een stoornis ontwikkelt en, wanneer mensen al angstig of depressief zijn, met wie het beter of slechter gaat na verloop van tijd.

## Doel van het proefschrift

Het doel van dit proefschrift was om meer inzicht te krijgen in de cognitieve kwetsbaarheid voor angst- en depressieve stoornissen, waarbij we specifiek keken naar de rol van disfunctionele automatische associaties. De resultaten van dit proefschrift zullen besproken worden aan de hand van vier vragen. Dit betekent dat sommige hoofdstukken wat door elkaar aan bod zullen komen. Allereerst hadden we een methodologische vraag die ging over het meetinstrument dat we hebben gebruikt om disfunctionele automatische associaties te meten, de Impliciete Associatie Test (IAT: zie hieronder voor een uitleg). De eerste vraag luidde (1) op welke manier kan een IAT-effect het beste worden berekend wanneer we mensen meten in een laboratorium? Vervolgens hadden we drie meer inhoudelijke vragen: (2) Dragen disfunctionele automatische zelf-associaties bij aan het ontstaan van angststoornissen en/of depressieve stoornissen? (3) Dragen disfunctionele automatische zelf-associaties bij aan de instandhouding van angststoornissen en/of depressieve stoornissen? En (4) zijn disfunctionele automatische associaties specifiek voor bepaalde angststoornissen en/of depressieve stoornissen?

De meeste studies in dit proefschrift maken gebruik van gegevens die verzameld zijn in de Nederlandse Studie naar Depressie en Angst (NESDA; zie www.nesda.nl). Dit is een grootschalig onderzoek waarin 2981 angstige, depressieve en gezonde mensen uit verschillende delen van Nederland worden gevolgd in de loop van de tijd. Binnen de NESDA werd ook de Impliciete Associatie Test meegenomen om automatische zelf-angst associaties en automatische zelf-depressie associaties te meten. De studies uit hoofdstuk 4 zijn gebaseerd op andere gegevens. De gegevens van studie 1 zijn verzameld in het kader van een behandeling voor bloosangst. De gegevens van studie 2 zijn verzameld onder sociaal angstige patiënten die zich aanmeldden voor een sociale angstbehandeling bij verschillende GGZ-instellingen.

## Impliciete Associatie Test

Er zijn in de afgelopen decennia verschillende meetinstrumenten ontwikkeld om automatische associaties te meten (voor een overzicht zie: Fazio & Olson, 2003). In dit proefstuk stond echter één meetinstrument centraal, de Impliciete Associatie Test (IAT). De IAT is ongeveer tien jaar geleden ontwikkeld door Greenwald, McGhee en Schwartz (1998). Deze computertaak wordt verondersteld de relatieve associatiesterkte te meten tussen twee doel categorieën (bijvoorbeeld *ik* vs. *ander*) en twee eigenschappen (bijvoorbeeld *angstig* vs. *kalm*). De proefpersoon krijgt de opdracht om woorden uit deze vier categorieën te sorteren met behulp van twee knoppen: twee categorieën met de ene knop (bijv. *ik* en

*angstig*) en de twee andere categorieën met de andere knop (bijv. *ander* en *kalm*). Vervolgens wisselen de categoriecombinaties (bijv. *ik* en *kalm* op de ene knop / *ander* en *angstig* op de andere knop). De achterliggende gedachte is dat het gemakkelijker is om woorden uit twee categorieën met dezelfde knop te sorteren wanneer deze twee categorieën sterk met elkaar geassocieerd zijn. Dat betekent dat mensen in een dergelijk geval sneller reageren. Door de gemiddelde reactietijden van de blokken met verschillende categoriecombinaties van elkaar af te trekken kan men het zogenaamde IAT-effect berekenen. Dit effect geeft dus aan hoe sterk de verschillende categorieën relatief gezien met elkaar zijn geassocieerd. Dus als je sneller reageert wanneer *ik* en *angst* (en *ander* en *kalm*) de knop delen, dan wanneer *ik* en *kalm* (en *ander* en *angstig*) de knop delen, dan noemen we dat een sterkere automatische zelf-angst associatie.

### *1) Op welke manier kan een IAT-effect het beste worden berekend wanneer we mensen meten in een laboratorium?*

Het belangrijkste doel van *hoofdstuk 2* was om vast te stellen hoe we het beste een IAT-effect kunnen berekenen wanneer we de IAT gebruiken in een laboratoriumomgeving. Met laboratorium bedoelen we in dit geval een testruimte waarin mensen gemeten worden, waarbij de omstandigheden voor alle deelnemers aan het onderzoek zoveel mogelijk gelijk gehouden worden. Eerder onderzoek (Greenwald, Nosek & Banaji, 2003) heeft al laten zien dat zogenaamde D-maten het goed doen om een IAT-effect te bereken, wanneer deze worden gebruikt voor gegevens die via het internet zijn verzameld. We wilden daarom graag weten of we dezelfde resultaten zouden vinden voor de IAT in een laboratoriumomgeving. Wanneer deelnemers thuis met de IAT worden gemeten, zou dit kunnen betekenen dat ze minder geconcentreerd zijn en sneller afgeleid worden. Hierdoor zouden ze langzamer kunnen reageren of meer wisselingen in hun reactietijden laten zien. Dit zou de reden kunnen zijn dat D-maten het juist goed doen, omdat deze D-maten het IAT-effect delen door de standaarddeviatie van alle reactietijden en daardoor corrigeren voor zulke prestatiewisselingen. Op grond hiervan verwachtten we dat D-maten het misschien minder goed zouden doen in een omgeving waarin deelnemers geconcentreerder kunnen werken, zoals in een laboratorium.

Normaal gesproken is het moeilijk om genoeg deelnemers te kunnen vinden om dit goed te kunnen testen. Gelukkig beschikten we over de grootschalige laboratorium steekproef van de NESDA. In tegenstelling tot onze verwachting bleek de $D_{600}$ (één van de D-maten) het beste uit de bus te komen. Dit suggereert dat de $D_{600}$ goed gebruikt kan worden om IAT-effecten te berekenen in laboratoriumsteekproeven. Tenminste wanneer

een zelfde soort opzet van het onderzoek wordt gebruikt als in de NESDA. Het is daarom wat voorbarig om op grond van deze uitkomsten alle andere berekeningen voor een IAT-effect overboord te gooien. Eerst zal nog verder bekeken moeten worden of D-maten het ook beter doen in het voorspellen van meer spontaan gedrag. Het duale proces model veronderstelt immers dat automatische associaties vooral belangrijk zijn voor dit type gedragingen. Helaas hadden we geen meetinstrument in onze studie om hiernaar te kijken.

### *2) Dragen disfunctionele automatische zelf-associaties bij aan het ontstaan van angststoornissen en/of depressieve stoornissen?*

Huidige duale procesmodellen van angst en depressie (Beevers, 2005; Ouimet, Gawronski & Dozois, 2009) benadrukken de rol van disfunctionele automatische associaties in het ontstaan en voortduren van deze stoornissen. Vooral ongunstige associaties die mensen hebben met zichzelf zouden wel eens een belangrijke rol kunnen spelen in dit opzicht (zie bijv. Clark, Beck & Alford, 1999; Rapee & Heimberg, 1997). In de *eerste studie van hoofdstuk 3* hebben we daarom onderzocht of automatische zelf-associaties inderdaad samenhangen met angst en depressie. We hebben gekeken of juist mensen met een depressie sterkere automatische zelf-depressie associaties hebben en mensen met angststoornissen sterkere automatische zelf-angst associaties hebben. Dit bleek inderdaad het geval. Verder bleek dat de mensen die hersteld waren van eerdere angstige of depressieve klachten nog steeds negatievere automatische zelf-associaties hadden dan gezonde mensen, maar minder negatief dan mensen met huidige klachten. Zulke negatieve associaties zorgen er mogelijk voor dat mensen kwetsbaarder zijn voor de terugkeer van klachten, ondanks dat ze wel hersteld zijn. Dit onderzoek laat voor het eerst zien dat angstige en depressieve mensen zich inderdaad kenmerken door automatische zelf-angst en zelf-depressie associaties. Dit zou erop kunnen wijzen dat deze disfunctionele automatische associaties inderdaad een rol spelen in het opnieuw ontstaan van deze stoornissen.

Om hier meer zicht op te krijgen, hebben we in de *tweede studie van hoofdstuk 5* gekeken of automatische zelf-angst associaties voorspellen wie er gedurende de twee jaar erna een angststoornis ontwikkelen. Hiervoor onderzochten we drie verschillende groepen, namelijk een gezonde controlegroep, mensen met een depressie en mensen die hersteld waren van een angststoornis. Onze verwachting was dat mensen met sterkere zelf-angst associaties meer kans hebben om een angststoornis te krijgen. Automatische zelf-angst associaties bleken inderdaad te voorspellen wie van de herstelde mensen en wie van de depressieve mensen een grotere kans hadden om een angststoornis te ontwikkelen. Een dergelijk verband vonden we echter niet in de groep gezonde mensen.

Daarmee spreken deze resultaten tegen dat automatische zelf-angst associaties een premorbide kwetsbaarheidsfactor zijn voor het ontstaan van angststoornissen. Het lijkt er eerder op dat mensen sterkere associaties krijgen tussen zelf en angstig doordat ze al een (angst)stoornis hebben gehad. Deze disfunctionele associatieve netwerken zouden vervolgens kunnen bijdragen aan de hardnekkigheid van deze stoornissen en/of ervoor kunnen zorgen dat mensen na herstel weer terugvallen. Het zou natuurlijk interessant zijn om op dezelfde manier te kijken naar het ontstaan van depressie, zodat we dit kunnen vergelijken met de resultaten van deze studie. Dit is iets wat we binnen de NESDA kunnen onderzoeken en waar we op dit moment nog mee bezig zijn.

### *3) Dragen disfunctionele automatische zelf-associaties bij aan de instandhouding van angststoornissen en/of depressieve stoornissen?*

Voortbouwend op de vorige vraag, waren we benieuwd of disfunctionele automatische zelf-associaties ervoor zorgen dat angststoornissen en depressieve stoornissen in stand gehouden worden. Daarom onderzochten we in de *eerste studie van hoofdstuk 5* of automatische zelf-angst en zelf-depressie associaties voorspellend zijn voor het natuurlijk beloop van angst en depressie twee jaar later. Het bleek dat sterkere automatische associaties tussen zelf en angstig samenhingen met een kleinere kans om te herstellen van een angststoornis, terwijl automatische zelf-depressie associaties gerelateerd waren aan een kleinere kans om te herstellen van een depressieve stoornis. Deze uitkomsten sluiten aan bij de theorie dat disfunctionele automatische associaties een rol spelen in de instandhouding van angst en depressie. Zoals ook genoemd bij het bespreken van de vorige vraag komen disfunctionele associaties mogelijk in het geheugen vast te liggen terwijl mensen een angststoornis of een depressie hebben. Deze disfunctionele associaties lokken mogelijk negatieve gedragingen, gedachten en gevoelens uit, welke op hun beurt de negatieve associaties weer verder versterken. Op deze manier zou er een negatieve spiraal kunnen ontstaan die het steeds moeilijker maakt om de angst- en depressieve klachten te stoppen (zie bijv. Beevers, 2005).

### *4) Zijn disfunctionele automatische associaties specifiek voor bepaalde angststoornissen en/of depressieve stoornissen?*

Ten slotte waren we benieuwd of automatische associaties specifiek zijn voor bepaalde stoornissen, of dat ze samenhangen met psychopathologie in het algemeen. In de beantwoording van vraag 2 en 3 kwam al naar voren dat de effecten van automatische zelf-associaties voor een belangrijk deel stoornis-specifiek waren. Mensen met een de-

pressie vertoonden sterkere automatische zelf-depressie associaties en mensen met angstklachten lieten sterkere automatische zelf-angst associaties zien. Daarnaast hingen specifiek zelf-angst associaties samen met het ontstaan van angst en met een kleinere kans om te herstellen van een angststoornis, terwijl specifiek automatische zelf-depressie associaties gerelateerd waren aan een kleinere kans om te herstellen van een depressieve stoornis. We vonden echter ook dat mensen met een angst- en depressieve stoornis in het algemeen negatievere zelf-associaties hadden dan gezonde controles, wat stoornis-specificiteit tegenspreekt.

In *hoofdstuk 4* hebben we nog verder gekeken naar stoornis-specificiteit van automatische associaties. In de *eerste studie* waren we benieuwd of mensen die bang zijn om te blozen sterkere associaties hebben tussen *blozen* en *negatieve sociale uitkomsten*, zoals 'falen' of 'afgaan'. Dit was geïnspireerd op de theorie dat mensen met bloosangst de negatieve sociale gevolgen van hun eigen blozen overschatten (Dijk, Voncken & de Jong, 2009). In overeenstemming met dit uitgangspunt vonden we dat mensen met bloosangst inderdaad negatievere automatische associaties hadden met blozen dan mensen zonder bloosangst. Ook wanneer we vroegen naar hun expliciete gedachten, bleken ze negatievere sociale reacties op hun blozen te verwachten. Deze uitkomsten sluiten aan bij eerder onderzoek dat specifieke automatische associaties liet zien in sociale angst en specifieke fobie (Roefs et al., 2011). In dit onderzoek hadden we alleen geen klinische controlegroep. Daardoor kunnen we niet zeker weten of negatieve bloos-associaties echt specifiek zijn voor mensen met bloosangst.

In de *tweede studie van hoofdstuk 4* hebben we gekeken naar twee soorten automatische associaties die kenmerkend zouden kunnen zijn voor mensen met sociale angst, namelijk negatieve/positieve zelf-associaties (wordt vaak *impliciete zelfwaardering* genoemd) en associaties met sociale situaties, zoals 'feestje' of 'vergadering'. De opzet van dit onderzoek bouwt daarmee voort op de theorie van sociale angst van Wells en Clark (1997) die veronderstelt dat sociaal angstige mensen sociale situaties als bedreigend ervaren, omdat ze negatieve ideeën hebben over zichzelf en over hun sociale vaardigheden. In dit onderzoek vergeleken we niet alleen sociaal angstige patiënten met niet-angstige deelnemers, maar ook met mensen met een paniekstoornis. We vonden dat negatieve automatische associaties met sociale situaties specifiek waren voor sociale angst. Het bleek namelijk dat de patiënten met sociale angst sterkere associaties tussen sociale situaties en negatieve uitkomsten hadden dan niet-angstige deelnemers en paniekpatiënten. Verder hadden sociaal angstige patiënten een lagere impliciete zelfwaardering dan niet-angstige deelnemers. De patiënten met een paniekstoornis zaten qua impliciete

zelfwaardering tussen de niet-angstige groep en de sociale angst groep in. Deze laatste uitkomsten zouden kunnen betekenen dat een verlaagde impliciete zelfwaardering een meer algemeen kenmerk is van angststoornissen. Misschien zijn mensen met een angststoornis over het algemeen minder zeker van zichzelf. Het lijkt erop dat de impliciete zelfwaardering extra verlaagd is in sociale angst, wat niet verbazingwekkend is, omdat sociaal angstige mensen zich per definitie veel zorg maken over zichzelf en hoe ze overkomen op anderen.

Concluderend kunnen we stellen dat verschillende onderzoeken in dit proefschrift stoornis-specifieke effecten hebben laten zien voor angst- en depressieve stoornissen. Tegelijkertijd wordt het duidelijk dat stoornis-specificiteit niet een kwestie is van alles of niets en dat sommige automatische associaties wellicht deels stoornis-specifieke invloed hebben en deels effecten laten zien die stoornis overstijgend zijn.

## Suïcidale gedachten

De *tweede studie van hoofdstuk 3* stond min of meer los van de andere onderzoeksvragen in dit proefschrift. We waren namelijk benieuwd of disfunctionele automatische zelf-associaties ook samenhangen met suïcidale gedachten, een symptoom waar veel mensen met depressieve en angststoornissen last van hebben. Dit onderzoek was geïnspireerd op de bevinding dat automatische zelf-associaties vooral voorspellend zijn voor spontaan en oncontroleerbaar gedrag (bijv. Asendorpf, Banse & Mücke, 2002; Egloff & Schmukle, 2002). Suïcidale patiënten geven vaak aan dat ze hun suïcidale gedachten maar moeilijk kunnen controleren en stoppen. Ongunstige automatische gedachten over het zelf zouden daarom wel eens een belangrijke rol kunnen spelen bij het ontstaan en voortduren van suïcidale gedachten en gedrag. Deze studie was een eerste stap om te onderzoeken of automatische zelf-associaties inderdaad samenhangen met suïcidaliteit. Automatische zelf-associaties bleken inderdaad negatiever te zijn bij mensen die aan suïcide dachten dan bij mensen zonder deze gedachten. Ook mensen die in het verleden een suïcidepoging hadden gedaan, bleken negatievere automatische zelf-associaties te hebben. Verder bleek de kans op suïcidale gedachten extra groot voor de mensen met zowel automatische als meer expliciete negatieve zelf-associaties. Dit zou kunnen betekenen dat mensen die zichzelf ook expliciet als negatief zien het moeilijker vinden om hun negatieve automatische associaties bij te stellen. Dit onderzoek laat voor het eerst zien dat suïcidale mensen zich inderdaad kenmerken door negatievere automatische zelf-associaties.

## Klinische implicaties

Alle onderzoeken in dit proefschrift waren correlationeel van aard, wat betekent dat we wel hebben gekeken of we verbanden konden vinden, maar dat we op grond van deze uitkomsten niet kunnen vaststellen of disfunctionele automatische associaties ook echt de oorzaak zijn van depressie en angstklachten. Hiervoor is eerst verder onderzoek nodig waarin automatische associaties experimenteel beïnvloed worden, zodat bekeken kan worden wat voor effecten dit heeft op angstige en depressieve klachten. Maar áls disfunctionele automatische associaties inderdaad een oorzakelijke rol spelen in depressie en angst, wat zou dit dan voor gevolgen hebben voor de behandeling van deze stoornissen?

Veel van de bestaande psychologische behandelingen zijn gericht op het praten over klachten of vervelende gedachten, of het veranderen van gedragingen. Wat er onder invloed van deze behandelingen gebeurt met de automatische associaties is nog grotendeels onbekend. Onderzoek naar de effecten van cognitieve gedragstherapie op automatische associaties levert verschillende uitkomsten op. Een recente studie suggereert dat disfunctionele automatische associaties normaliseren onder invloed van cognitieve gedragstherapie (Reinecke, Soltau, Hoyer, Becker & Rinck, 2012), maar ander onderzoek laat zien dat dit niet zo is (Huijding & de Jong, 2007, 2009). Een probleem van dit type onderzoek is dat er vaak geen wachtlijst controlegroep wordt meegenomen, waardoor je niet zeker weet of veranderingen in automatische associaties door de behandeling komen of door herhaaldelijk meten. Over de invloed van medicatie op automatische associaties is nog nauwelijks iets bekend.

Als disfunctionele automatische associaties een oorzakelijke rol spelen in depressie en angst, dan lijkt het logisch om ervan uit te gaan dat de invloed van disfunctionele automatische associaties eerst onschadelijk gemaakt moet worden, wil iemand kunnen herstellen van deze stoornissen. Er zijn in ieder geval drie manieren waarop de negatieve invloed van automatische associaties verminderd zou kunnen worden. In de eerste plaats zouden expliciete gedachten de ongunstige invloed van automatische associaties te kunnen bijstellen, mits deze expliciete gedachten inderdaad 'gezond' zijn. Cognitieve therapie heeft bijvoorbeeld als doel om gezondere expliciete gedachten te leren ontwikkelen. Hiermee zou cognitieve therapie indirect de invloed van disfunctionele automatische associaties kunnen stoppen. Ten tweede, wanneer expliciete gedachten eenmaal gezond zijn, is het van belang om de kans te vergroten dat deze gedachten ook kunnen optreden. Expliciete afwegingen maken kost moeite, tijd en motivatie. Wanneer mensen dus over voldoende cognitieve capaciteit beschikken en gemotiveerd zijn, helpt dat om gezonde expliciete afwegingsprocessen te bevorderen. Medicatie zou wellicht een gunstige inv-

loed kunnen hebben op motivatie en cognitieve capaciteit. Wanneer mensen zich immers beter gaan voelen en minder in beslag worden genomen door hun klachten, hebben ze meer gelegenheid om expliciete afwegingen te maken. Ook wordt er gewerkt aan nieuwe interventies die specifiek gericht zijn op het trainen van werkgeheugenfuncties (bijvoorbeeld in depressie: Siegle, Ghinassi & Thase, 2007).

Ten derde blijven er echter momenten bestaan dat motivatie of cognitieve capaciteit niet toereikend zijn en dat automatische associaties toch het gedrag beïnvloeden. Daarom wordt er tevens onderzoek gedaan naar de directe bewerking van automatische associaties en de gevolgen van deze bewerking. Een voorbeeld hiervan zijn studies naar de invloed van evaluatieve conditionering op automatische zelf-associaties (Baccus, Baldwin & Packer, 2004; Dijksterhuis, 2004). Bij evaluatieve conditionering wordt een object (geconditioneerde stimulus) steeds gekoppeld aan een positieve of negatieve stimulus (ongeconditioneerde stimulus), waardoor het object op den duur deze waarde overneemt. In deze studies werden in een computertaak steeds ik-woorden gekoppeld aan positieve eigenschappen. Dit bleek de automatische zelf-associaties van de proefpersonen positief te beïnvloeden. Bovendien bleek dat positieve automatische zelf-associaties als gevolg van de conditioneringstaak mensen minder gevoelig maakten voor negatieve feedback. Een soortgelijke interventie werd onderzocht in de context van sociale angst. Sociaal angstige studenten bij wie foto's van henzelf steeds gekoppeld werden aan positieve gezichtsuitdrukkingen hadden minder sterke associaties tussen *ik* en *afgewezen*. Ook lukte het hen om langer een onverwachte spreekbeurt vol te houden (Clerkin & Teachman, 2011).

Momenteel wordt nog volop onderzocht wat de invloed is van disfunctionele automatische associaties op angstige en depressieve klachten en of trainingen direct gericht op deze associaties ook bruikbaar zijn voor de klinische praktijk. Hopelijk brengen deze studies de komende jaren meer duidelijkheid over de vraag hoe mensen ervoor kunnen zorgen dat ze niet langer nodeloos geleid worden door hun ongewenste automatische associaties.

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# C

# Curriculum Vitae

Klaske Glashouwer was born on the 26th of March 1982 in Kootstertille, the Netherlands. She finished secondary school at Christelijk Gymnasium Beyers Naudé in Leeuwarden in 2000. In the same year, she started studying religious studies at the University of Groningen. However, she quitted this course and after a gap year she switched to psychology at the Radboud University in Nijmegen, with major in Clinical Psychology. In April 2007, she received her Masters degree cum laude. Klaske started her PhD studies in June 2007 at the Department of Clinical Psychology of the University of Groningen, where she carried out the research in this dissertation. Besides research, she has an interest in clinical work and in January 2009 she started the clinical training to become healthcare psychologist. From January 2009 till December 2010 she worked as psychologist at GGZ Friesland in Leeuwarden with adults that suffer from anxiety disorders. From January 2011 till February 2012 she worked as psychologist at the Centre for eating disorders of Accare, child and adolescent psychiatry. Currently, she is working both as healthcare psychologist and researcher in the field of eating disorders at Accare and the University of Groningen.

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