BLURRING AVERSIVE MEMORY

Exploring a novel route to fear reduction

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Exploring a novel route to fear reduction

VERVAGING VAN AKELIGE HERINNERINGEN

Onderzoek naar een nieuwe weg tot angstreductie
(met een samenvatting in het Nederlands)

Proefschrift

ter verkrijging van de graad van doctor aan de Universiteit Utrecht op gezag van de rector magnificus, prof. dr. G.J. van der Zwaan, ingevolge het besluit van het college voor promoties in het openbaar te verdedigen op vrijdag 5 juni 2015 de middags te 12.45 uur

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It's not what you look at that matters, it's what you see.
—HENRY DAVID THOREAU

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Chapter 1

Introduction

Blurring Aversive Memory

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Decades of research on pathological fear have resulted in strong theory of its etiology and maintaining factors. In a nutshell, it is thought that clinical fear originates when the individual has come to associate an innocuous stimulus (e.g., a dog) with an aversive event (e.g., a dog bite). Subsequent avoidance behaviors are held to facilitate the maintenance of fearful responding (e.g., Bouton, Mineka, & Barlow, 2001). Today, the treatment of choice for anxiety-related disorders is cognitive behavioral therapy (CBT; e.g., Hofmann & Smits, 2008). CBT is firmly rooted in basic science. However, about a third of patients do not benefit from the treatment and relapse rates are high (e.g., Barlow, Allen, & Choate, 2004). Additional research is thus urgently needed.

One way to improve treatment is to examine *why* certain therapies are effective. Interestingly, such treatments may not be primarily based on fundamental research. A prime example and main topic of the present thesis is Eye Movement Desensitization and Reprocessing (EMDR), one of the recommended treatments for posttraumatic stress disorder (PTSD). EMDR has drawn much scientific attention, not least because of a unique and allegedly key procedure. In EMDR, patients are instructed to make horizontal eye movements while holding a distressing memory in mind. This procedure typically results in a decrease in the vividness of the memory and in subjective distress associated with the memory. Although it is effective, EMDR has met with skepticism for a long time: its effectiveness – and in particular how the eye movements contribute to its effectiveness – was not explained by a sound theory. Recent years, however, have seen an increase in experimental preclinical research, which has resulted in a robust account. These fresh insights have also revealed a novel route to the reduction of learned fear in anxiety-related disorders other than PTSD. The primary aim of this thesis is to explore this novel route.

The introduction continues with a presentation of models of human fear, followed by current practices and challenges in the treatment of anxiety-related disorders. Subsequently, an overview of EMDR's history is provided and it will be elucidated how new insights in this field may inform the treatment of anxiety-related disorders. The introduction concludes with an outline of the thesis.

Models of human fear

Fear is an adaptive emotion that is evoked by stimuli associated with threat. When someone comes at you with a knife, fear elicits a fight-or-flight response (i.e., increased activation of the sympathetic nervous system that mobilizes the body). This response is adaptive, because it minimizes the risk of harm. Conditioning theory postulates that fearful responding originates from a learning experience in which a previously neutral stimulus (e.g., a knife) is paired with an unconditionally aversive stimulus (abbreviated as UCS or US; e.g., physical injury). As a result, the formerly neutral stimulus (now referred to as *conditional* or *conditioned* stimulus; CS [these terms are used interchangeably in the literature, e.g. Gantt, 1966, and in this thesis]) comes to elicit a conditional fear response (CR; Watson & Rayner, 1920). Fear thus primarily serves a protective function.

Fear becomes "maladaptive" (i.e., pathological or clinical) when it generalizes to objectively harmless stimuli or situations and comes to interfere with normal function or causes suffering. For instance, a patient with panic disorder may believe that a pounding heart signals the occurrence of a heart attack, and therefore avoids physical exercise. Likewise, a patient with obsessive-compulsive disorder may think that having intrusive thoughts about hurting his kids means that someday he will perform a violent act, and therefore spends minimal time with his children. As a final example, a patient with social phobia may avoid social situations because she fears that acting in an embarrassing way will result in negative evaluation or rejection.

Like adaptive fear, clinical fear is thought to result from associative learning. For example, 90% of patients with panic disorder report that a first panic attack had triggered the onset of their disorder (81.3% had a direct experience and 8.7% had a vicarious experience; Öst & Hugdahl, 1983). The classical conditioning model explains that the pairing of a CS (e.g., a crowded place) and a UCS (e.g., a panic attack) results in the CS becoming a dangerous stimulus itself, which *automatically* elicits a fear CR (Watson & Rayner, 1920). However, this model cannot account for several clinical observations (Davey, 1997). First, following a traumatic event only a minority of individuals develops clinical problems. For example, a large survey showed that 9.2% of trauma-exposed individuals developed PTSD (Breslau et al., 1998). Likewise, a majority of Dutch Army troops deployed to Iraq reported to have experienced at least one potentially traumatic event, yet 3.5% developed deployment-related PTSD (Engelhard et al., 2007). Conversely, many patients with an anxiety-related disorder do *not* recall a traumatic experience (e.g., Rachman, 1977). Apparently, CS-UCS pairing is not a requisite or sufficient condition for the expression of conditional responding. Another criticism leveled at

the early model relates to the *equipotentiality* premise, which holds that all stimuli have equal potential to become a conditional stimulus (e.g., Rachman, 1977; Seligman, 1971). This premise is at odds with the observation that phobias typically comprise a limited set of stimuli (e.g., heights, water, insects, snakes, the dark) and that "only rarely, if ever, do we have (…) electric-outlet phobias or hammer phobias, even though these things are likely to be associated with trauma in our world" (Seligman, 1971, p. 312).

Contemporary conditioning theory

Contemporary conditioning theory does account for these clinical phenomena (Davey, 1997). Whereas the original model holds that the CR is *directly* elicited by the CS, the contemporary model explains that the CR is *mediated* by activation of the memory-encoded CS-UCS association (see Figure 1). In other words, fear learning is not conceptualized as stimulus-response (S-R or CS-CR) learning, but rather as stimulus-stimulus (S-S or CS-UCS) learning. Following fear learning the CS activates the CS-UCS association, which triggers UCS expectancy and produces the CR. This current model has many implications that can be briefly summarized in two categories. First, all factors that are of influence on the strength of the CS-UCS association should affect CR intensity. Turning back to the example of an assault with a knife, only a minority of people has been attacked, yet the majority would respond with fear when someone suddenly approaches with a knife. Modern conditioning theory accounts for this seeming discrepancy by assuming that the CS-UCS association can be learned not only through direct experience, but also through observation, socially/culturally transmitted information, and inference. Why then, do we come across knife phobias far less often compared to, for example, snake phobias? After all, we have all learned at some point in our lives that knifes (like snakes) can be dangerous. One account explains that knifes may also have been associated with other non-aversive outcomes (e.g., cutting vegetables) and therefore do not signal danger per se. Indeed, research shows that stimuli that have been encountered in absence of aversive consequences are much more difficult to become associated with threat, a phenomenon referred to as *latent inhibition* (Davey, 1989). Existing beliefs or expectations about the CS-UCS association may thus interfere with new learning. Nevertheless, biological *preparedness* may also account for the violation of the equipotentiality premise. Evolution may have selected certain stimuli to get more easily associated with threat (Öhman & Mineka, 2001; Seligman, 1971).

A second set of implications relates to the subjective evaluation of the UCS. Given that the CR is mediated through activation of the UCS cognitive representation, any factor that

changes existing knowledge about the UCS should impact the strength of the fear response. Thus, if post conditioning information renders a more negative evaluation of the UCS, coined UCS *inflation*, then CR intensity should increase. Likewise, reassessing the UCS less negatively, called UCS *deflation*, should decrease CR intensity (Davey, 1997). Consequently, this model can account for several criticisms directed at the early model. First, some cases of pathological fear that lack a trauma history can be explained by a combination of *sensory preconditioning*, i.e. the pairing of a CS and a non-aversive UCS, and subsequent UCS *inflation*. For example, Davey, de Jong, and Tallis (1993) describe how a bank employee was threatened with a gun by a robber and returned to work the next day without any residual fear symptoms. Only 10 days later, after being informed by the police that the attacker had already taken the lives of other people, he began to develop severe post-traumatic stress symptoms. UCS *deflation* may account for the observation that many individuals do *not* develop clinical problems following trauma exposure. People differ in the use of threat devaluing coping strategies such as positive reappraisal or faith in social support, and the use of such strategies correlates with measures of psychopathology (e.g., Davey, Burgess, & Rashes, 1995).

Contemporary conditioning theory thus provides a comprehensive account of the etiology of anxiety disorders that can explain several clinical observations. The next section will discuss how research and theory have informed the treatment of anxiety disorders.

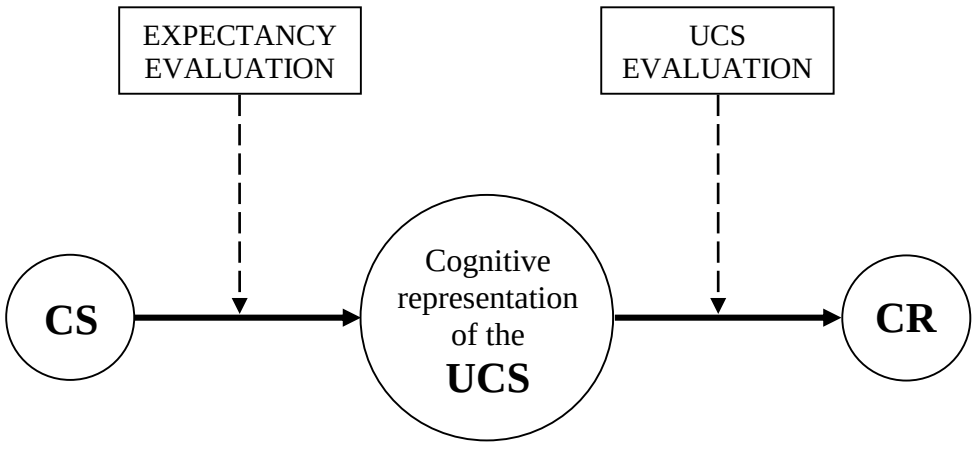

Fig. 1 Schematic representation of contemporary conditioning theory (based on Davey, 1997).

Practices and challenges in the treatment of anxiety disorders

Cognitive behavioral therapy (CBT) is the gold standard treatment for anxiety disorders (e.g., Hofmann & Smits, 2008; Olatunji, Cisler, & Deacon, 2010). CBT comprises of cognitive techniques (e.g., challenging catastrophic beliefs/appraisals) and behavioral techniques (e.g., exposure to feared stimuli/situations) that aim to modify maladaptive responses. Exposure approaches are central to CBT for all anxiety disorders (Arch & Craske, 2009). Importantly, CBT is characterized by the fact that its intervention strategies are derived from scientifically established procedures or principles (e.g., Arch & Craske, 2009; Hermans, Eelen, & Orlemans, 2007). Thus, progress in science has typically preceded the modifications of CBT. The following paragraphs will discuss the major developments of the exposure component.

More than half a century ago, Wolpe (1958) developed a treatment technique called *systematic desensitization*. Inspired by learning theory he noted that "since neurotic behavior demonstrably originates in learning, it is only to be expected that its elimination will be a matter of unlearning" (Wolpe, 1958, p. ix). Unlearning of fearful responding was accomplished using *reciprocal inhibition* (also known as *counter-conditioning*), which refers to the principle that incompatible responses cannot exist together. That is, when competing responses are triggered simultaneously, the dominant response will reciprocally inhibit the antagonistic response. Accordingly, this treatment of fear involved (1) identification of fear-evoking stimuli, (2) relaxation training (e.g., applying deep muscle relaxation, a response that is incompatible with excessive fear), and (3) (imaginal) exposure to feared stimuli while maintaining deep muscle relaxation. Although effective, the additive effect of the relaxation component soon became a topic of debate. A more parsimonious reading of the effects is that non-reinforced CS-exposures (i.e., in absence of the UCS) cause CR decrement (Pavlov, 1927). Basically, this implies that the relaxation component is redundant. Indeed, in a research review, Levin & Gross (1985) concluded that relaxation primarily facilitates the imagination of feared scenes, and may therefore be dispensable for phobic individuals with good imaging ability. Further in line with the 'exposure explanation' of systematic desensitization, *flooding* (i.e., non-graduated exposure with minimal escape possibilities) and *implosion* (i.e., exaggerated imaginal exposure) also appeared successful in the treatment of anxiety-related disorders (Abramowitz, Deacon, & Whiteside, 2012). Based on these insights, contemporary exposure therapy is devoid of the relaxation component.

Further refinement of the exposure procedure has been based on the theoretical model that explains the underlying mechanisms of exposure-based fear reduction. For a long time,

Emotional Processing Theory was the leading account (Foa & Kozak, 1986). In short, this theory purports that exposure to a feared stimulus activates a "fear structure", i.e. the memory-encoded network of associations between the feared stimulus (e.g., an elevator), its meaning (e.g., 'I will lose my mind'), and responses (e.g., fear and avoidance). During exposure, the individual acquires information that is incompatible with the fear structure (e.g., 'I can enter an elevator without going crazy'), and this mismatch will incite corrective learning. Specifically, it will cause the formation of a "non-fear structure" that replaces (Foa & Kozak, 1986) or inhibits (Foa & McNally, 1996) the original fear structure. As a result, *habituation* takes place, which basically means that fear diminishes. Accordingly, the model predicts that treatment success strongly depends on (1) activation of the fear structure and (2) within-session habituation (i.e. immediate fear reduction; e.g., Craske et al., 2008). Recent research, however, has shown that none of these variables are consistently predictive of treatment outcome (e.g., Baker et al., 2010; Meuret, Seidel, Rosenfield, Hofmann, & Rosenfield, 2012). These findings thus call into question whether the reported level of fear during treatment is the right index of corrective learning (Craske et al., 2008). The question remains, then: What *is* the working mechanism of exposure treatment? And how can we use this information to improve the treatment of fear?

Today, there is much support for an *inhibitory learning* account (e.g., Bouton, 1993, 2002, 2004; Craske et al., 2008; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Vervliet, Craske, & Hermans, 2013). The key premise of this model is that non-reinforced CS-exposure does not lead to "unlearning" of the CS-UCS association, but rather causes new learning that is characterized by the formation of an inhibitory CS-UCS association (also referred to as the CS-noUCS association or *extinction memory*). Retrieval of this new association upon CS exposure will not trigger UCS expectancy, and therefore the CR will fail to appear. An important prediction of this theory is that long-term retention of extinguished fear critically depends on the ability to retrieve the extinction memory. Many experiments, using several laboratory procedures, have provided evidence for this hypothesis (for a review, see Vervliet et al., 2013). First, the mere passage of time following CR-extinction typically results in a (partial) return of fear, called *spontaneous recovery*. This finding strongly suggests that the conditioning memory, although inhibited, is still intact. Also, CS presentations outside the extinction context produce so-called *renewal of fear*. Furthermore, exposure to an unsignaled UCS, which arguably retrieves the condition memory from long-term memory, causes *reinstatement* of the CR. Finally, after CR-extinction, the original CS, as compared to a novel CS, more easily reacquires a CR when paired with the UCS again, a phenomenon coined *rapid reacquisition*.

The inhibitory learning account has important clinical implications. First, it predicts that UCS expectancy violation, rather than immediate fear reduction, is a crucial index of corrective learning (e.g., Craske et al., 2008). When a patient reports a reduction in fear, but still does not believe that he or she is safe, then corrective learning has not taken place and fear may easily return. Therefore, the primary focus of exposure-based therapies should be "what do you need to learn" rather than "stay in the situation until fear declines" (Craske et al., 2014, p. 12). Second, extinction retention is a function of the ability to retrieve the extinction memory (Craske et al., 2008), which depends not only on factors such as context and time, but also on the relative strength of the conditioning and extinction memory, and on conditions that facilitate the retrieval of extinction memory.

In sum, exposure-based therapy for the treatment of anxiety-related disorders is anchored in basic science and continues to be informed by new insights into its underlying mechanisms. However, current treatment still has its shortcomings. The next section will outline the major challenge we face and how we might conquer it.

CBT: A short-term solution to a long-term problem?

CBT is quite effective: about 50-80% of patients show clinical improvement (Barlow, Allen, & Choate, 2004). However, many patients relapse. For example, Fava and colleagues (2001) investigated the treatment of 200 patients for panic disorder with agoraphobia, which comprised behavioral exposure homework. After six months, 165 patients (82.5%) had completed treatment, and 136 of them were panic-free for at least 1 month post treatment (68% of the original sample; 82.4% of the final sample). Results further showed that 31 out of 132 patients who were available for follow-up assessments (23%) had a relapse of full blown panic disorder at some time during the 2-14 year follow-up. Other investigations have shown that return of fear may be as high as 50% (Rachman, 1989). It may thus not be surprising that **relapse prevention** has become a major focus of research (e.g., Vervliet et al., 2013).

In the past 10-15 years many relapse prevention strategies have been put to test, inspired especially by the work of Bouton and colleagues (e.g., 1993; 2002; 2004). Based on the assumption that renewed conditional responding follows from a failure to retrieve the extinction memory, most of the procedures that have been invented aim to strengthen its consolidation or facilitate its retrieval (for a review, see Vervliet et al., 2013). For example, conducting exposure in multiple contexts may increase the likelihood of retrieving the extinction memory in a novel context. Indeed, studies have shown that exposure in three contexts compared to one context abolishes fear renewal in an unselected sample (Bandarian Balooch, Neumann, & Boschen,

2012) and in spider phobic students (Vansteenwegen, Vervliet, Iberico, Baeyens, Van den Bergh, & Hermans, 2007). However, other studies did not find such an effect (Neumann, Lipp, & Cory, 2007) or only showed partial attenuation (Bandarian Balooch & Neumann, 2011). Another promising strategy is the use of a 'reminder cue' during the exposure session that serves as a later reminder of the procedure and thereby facilitates the retrieval of extinction memory (e.g., Dibbets, Havermans, & Arntz, 2008; Dibbets & Maes, 2011; Vansteenwegen, Vervliet, Hermans, Beckers, Baeyens, & Eelen, 2006). However, a study testing individuals highly fearful of public speaking showed only a weak effect of this procedure (Culver, Stoyanova, & Craske, 2011, Study 2) that was not replicated (Culver et al., 2011, Study 3). Moreover, the reminder cue itself may come to signal safety, which poses the risk of renewed responding when the cue is absent (Vervliet et al., 2013).

There are more strategies under investigation, such as substantially increasing the number of exposures, exposure to multiple CSs at the same time, and the administration of drugs that enhance learning (e.g., D-cycloserine), but they all have one weakness in common: "even a strong extinction memory leaves the original fear memory intact and thereby presents the continuous risk for a return of fear" (Vervliet et al., 2013, p. 238). For that reason it may be fruitful to explore methods that change the original *conditioning memory* (i.e. the excitatory CS-UCS association). If, somehow, the conditioning memory can be weakened, then its retrieval should be hampered or its activation should produce a less intense CR.

One way to change the conditioning memory is to interfere with its initial consolidation. Research has shown that the consolidation of learned information into long-term memory takes quite some time (i.e., several hours; Nader & Hardt, 2009), and interference with this process may affect how the information is stored. Accordingly, it has been hypothesized that fear extinction (via CS-only exposures) immediately after fear learning (i.e., repeated CS-UCS pairings) disrupts the consolidation of the conditioning memory and is therefore associated with diminished (renewed) conditional responding. However, the experimental studies that have tested this hypothesis are inconclusive. Immediate relative to delayed fear extinction has been shown to reduce relapse (Norrholm et al., 2008), but also to increase relapse (Huff, Hernandez, Blanding, & LaBar, 2009), and to have no effect at all (Schiller et al., 2008). In addition, the acquisition of fear and the treatment of fear are typically separated by a long period of time. This calls into question the clinical applicability of the procedure. A promising and seemingly more feasible approach is to interfere with the *reconsolidation* of conditioning memory. This idea is based on the observation that once retrieved from long-term memory, a memory enters a labile state in which it can be 'updated', and whereupon it restabilizes (i.e., reconsolidates,

Nader & Hardt, 2009). Therefore, interference with the reconsolidation of the conditioning memory may reduce the intensity of renewed responding. In support of this hypothesis, Kindt and colleagues have shown that administration of propranolol (a drug that has been shown to disrupt memory reconsolidation) around the time of memory retrieval results in less return of fear (e.g., Kindt, Soeter, & Vervliet, 2009; Sevenster, Beckers, & Kindt, 2012; Soeter & Kindt, 2010, 2012). This finding arguably reflects that a weakening of the conditioning memory facilitates the retrieval of the extinction memory. However, the effect does not seem to be robust (for meta-analyses, see Lonergan, Olivera-Figueroa, Pitman, & Brunet, 2013).

To summarize, return of fear after extinction is a common clinical problem that needs to be solved. A potentially fruitful pathway to counter this problem, which is rooted in the contemporary inhibitory learning theory, is to weaken the conditioning memory. Recent research has shown that this might be accomplished by drug administration. Theoretically, however, there may be another way to weaken the conditioning memory that does *not* involve a drug manipulation. The aim of the current thesis is to examine this novel perspective.

A novel perspective

As stated earlier, contemporary conditioning theory explains that the intensity of fearful responding not only depends on the memorized CS-UCS contingency, but also on the anticipated intensity of the UCS. That is, "without the perception of negative consequences, an extremely likely event would not create fear" (Nelson, Lickel, Sy, Dixon, & Deacon, 2010, p. 214). Accordingly, reevaluating the UCS less negatively (UCS deflation) should diminish CR intensity. Experimental studies have investigated this hypothesis. For example, Hosoba, Iwanaga, and Seiwa (2001) paired a neutral CS with an aversive 100 dB white noise UCS. In a subsequent phase, they presented the UCS alone at stepwisely decreasing intensity (100-90-80-70-60 dB). Finally, they represented the CS and examined CR intensity. Compared to a control group that received UCS only presentations at equal intensity (i.e., five times at 100 dB), the experimental group showed the expected reduction in conditional responding. Importantly, this experiment shows that UCS deflation and subsequent changes in the CR can occur independently of any changes in the CS-UCS association. It follows that fear can be reduced via a non-associative pathway that likely does not involve the formation of an inhibitory CS-UCS association (cf. Davey, 1997). If return of fear results from a failure to activate the extinction memory, and if fear reduction following UCS deflation does *not* depend on the formation and activation of the extinction memory, then fear reduction acquired via UCS deflation should be associated with less return of fear.

A relevant question at this point is: What UCS deflation processes are known and is there any potential with regard to their clinical implication? If not, then investigating whether UCS deflation attenuates return of fear may not be worthwhile. Davey (1997) describes several processes that can be divided into two categories. The first category comprises of methods that involve actual exposure to the UCS. Specifically, the UCS can be presented repeatedly, which may cause habituation (Davey & McKenna, 1983), or at stepwisely decreasing intensity (Hosoba et al., 2001; Schultz, Balderston, Geiger, & Helmstetter, 2013), which rapidly reduces its perceived aversiveness. These procedures successfully diminish conditional responding. However, their clinical application is limited.

A second category covers UCS deflation processes that do not require the physical presence of the UCS. For example, the use of protective coping strategies might result in UCS deflation. Davey and colleagues (1995) found that being optimistic, having faith in social support, and applying positive reappraisal (among other strategies), were positively correlated with psychological health and negatively correlated with psychopathology. However, the causal relations between these constructs are unclear. Alternatively, verbal transmission of information about the UCS may, in theory, decrease the subjective aversiveness of the UCS. For example, a therapist may explain that spiders usually do not bite and if they do, a spider bite in the Netherlands is hardly ever lethal. However, convincing a patient through verbal information seems extremely difficult. Probably, phobic patients prioritize information that confirms their beliefs or need 'stronger evidence' (i.e., actual experiences, Davey, 1997).

This analysis suggests that UCS deflation techniques that have been explored thus far are unsatisfactory. However, as elucidated in the remainder of this introduction, there *is* a potentially useful UCS deflation technique that has not yet received much empirical attention. Specifically, it involves a technique that capitalizes on mental imagery and is used in EMDR therapy for PTSD. The exploration of this imagery-based technique nicely coincides with the growing interest in the role of imagery in emotion and emotional disorders (e.g., Hackmann & Holmes, 2004; Holmes, Arntz, & Smucker, 2007; Holmes & Mathews, 2010; Lee & Kwon, 2013). For example, it has been shown that imagery, relative to verbal processing, has a significantly greater impact on the elicitation of anxiety (Holmes & Mathews, 2005). Below, the history of EMDR is briefly outlined and new insights in the underlying mechanisms of EMDR are discussed. Finally, it will be explained how the most controversial component of EMDR may help to attenuate fear renewal.

Eye Movement Desensitization and Reprocessing

EMDR is a treatment for PTSD that was founded by Shapiro about 25 years ago (e.g., Shapiro, 1989). Unlike CBT, EMDR was not primarily based on scientifically established procedures. Instead, the efficacy of its central component was discovered serendipitously. During a walk in the park, Shapiro noticed that (spontaneous) eye movement during recall led to the desensitization and disappearance of recurrent, disturbing thoughts (see Engelhard, 2012). Based on this personal experience Shapiro developed EMDR therapy to treat trauma victims. In this therapy, the patient is asked to recollect the trauma memory, and then to select and visualize the most disturbing image of that memory along with accompanying emotions and a related negative cognition. Concurrently the therapist elicits horizontal eye movements by having the client follow the back and forth motion of the therapist's index finger.

Perhaps surprisingly to many, EMDR for PTSD appeared highly effective. A first study showed that EMDR, compared to a waitlist control condition, led to significant reductions in complaints (i.e., avoidance, intrusions, somatization), anxiety (in general and while thinking about the traumatic event), and negative self-evaluation (Wilson, Becker, & Tinker, 1995). A few years later, a first meta-analysis demonstrated that EMDR was not just better than 'no treatment', but as effective as behavior therapy, the very best treatment at that time (Van Etten & Taylor, 1998). Subsequent meta-analyses (e.g., Bradley, Greene, Russ, Dutra, & Westen, 2005), including a very stringent one (Bisson et al., 2007), reached the same conclusion. Today, EMDR is classified as 'evidence based' and (alongside CBT) recommended as the treatment of choice for PTSD (e.g., NICE, 2005; Trimbos, 2011).

Despite its serendipitous discovery and extraordinary appearance, it can thus be concluded that EMDR has lived up to its claims as an efficacious treatment. In short: EMDR works. For a long time, however, many scientists and professionals were highly skeptical (e.g., Herbert et al., 2000; McNally, 1999; Perkins & Rouanzoin, 2002). Their criticisms were not least fueled by the vague theoretical rationale for the eye movement component and disagreement about the added value of the eye movements (see Engelhard, 2012). Shapiro speculated that the eye movements incite a 'biochemical rebalancing of the nervous system' and 'shifting of information that is dysfunctionally locked in the nervous system' (1995). Some scholars qualified these premises as "sketchy neuro-biological theorizing" or merely "metaphorical" (Allen & Lewis, 1996; Muris & Merckelbach, 1999; Van den Hout, Muris, Salemink, & Kindt, 2001). In addition, some reviews concluded that the eye movements were not essential for therapeutic achievement (e.g., Lohr, Tolin, & Lilienfeld, 1999), whereas others

emphasized the need for adequate empirical research on this matter (Perkins & Rouanzoin, 2002). Not surprisingly, then, it came into question whether EMDR may simply comprise an exposure therapy (e.g., Rogers & Silver, 2002; note that, like in the case of systematic desensitization therapy [Wolpe, 1958], explaining EMDR in terms of imaginal exposure would also provide a more parsimonious account of its beneficial effects).

In an attempt to resolve this EMDR debate much research has been conducted in recent years. First, a meta-analysis has shown that the eye movements *do* add to EMDR's beneficial effects (Lee & Cuijpers, 2013). This finding suggests that EMDR is not just a variant of imaginal exposure. Second, great efforts have been made to test *how* the eye movements exert their effect. Interestingly, the findings of these experiments also point towards an additive role of the eye movements. Moreover, they convergently support one theory about its underlying mechanism that is anchored in cognitive psychology: Working Memory Theory (e.g., Andrade, Kavanagh, & Baddeley, 1997; Gunter & Bodner, 2008; Van den Hout et al., 2001; for review, see Van den Hout & Engelhard, 2012).

Working Memory Theory

Andrade and colleagues (1997) explained that both eye movement and visual imagery make use of the visuospatial sketchpad (VSSP), a limited-capacity working memory system that processes visual information. The authors hypothesized that when both tasks are performed simultaneously they compete for the same resources. As a result of this competition, visual imagery becoming less vivid. They further put forward that less vivid images should evoke less extreme emotional responses. Using an unselected sample and strictly controlled laboratory model, these hypotheses were tested and confirmed. Recall plus eye movements, relative to mere imaginal exposure, causes a decrease in the self-reported memory vividness and emotionality of negative idiosyncratic recollections.

These findings were promising and provided a novel explanation of the eye movement benefits. Yet, they could not account for an important clinical observation. Typically, in EMDR, patients report less distress associated with recalling the trauma *after* the dual task, i.e. in absence of the eye movements (e.g., Van den Hout et al., 2001). To investigate this issue, subsequent experiments included a pre-test and a post-test (i.e., before and after the intervention) in which memory vividness and emotionality were assessed. Nicely corresponding to the clinical observation, these experiments consistently demonstrated that the eye movement benefits extend beyond the intervention (e.g., Engelhard, van den Hout, Janssen, & van der Beek, 2010; Gunter & Bodner, 2008; Van den Hout et al., 2011; Van den Hout et al.,

2001; Kemps & Tiggemann, 2007). Apparently, the immediate changes during the dual task are maintained afterwards, an effect that several authors accounted for by memory reconsolidation (e.g., Maxfield, Melnyk, & Hayman, 2008; Van den Hout et al., 2010).

In the past few years, the working memory hypothesis has been extensively put to test. A prime example is the elegant series of experiments that was conducted by Gunter & Bodner (2008). Their studies compared the working memory account with two competing accounts. First, the *investigatory-reflex* account (e.g., Kuiken, Bears, Miall, & Smith, 2002; MacCulloch & Feldman, 1996) posits that the eye movements induce a strong sense of relaxation (up to 10 minutes after the eye movements) that becomes associated with the trauma memory. Accordingly, memory recall shortly after the eye movements should also lead to reductions in memory vividness and emotionality. In contrast, the working memory account holds that eye movement benefits only occur when they are performed *during* memory recall. A first experiment demonstrated that eye movement during memory recall, but not just before memory recall, produced the pre- to post-test reductions in memory vividness and emotionality. This experiment thus provided evidence in support of the working memory account. Second, the *increased hemispheric communication* account (e.g., Christman, Garvey, Propper, & Phaneuf, 2003) purports that horizontal eye movement increases the communication between the left and right brain hemispheres, which enhances memory retrieval and thereby facilitates desensitization. Crucially, vertical eye movement should not exert this effect. Indeed, by assuming that horizontal and vertical eye movements tax VSSP resources to a similar degree, the working memory account predicts that the two dual tasks lead to comparable decreases in memory vividness and emotionality. A second experiment showed exactly this: vertical as well as horizontal eye movements produce the rating benefits, again providing support for the working memory account. Gunter and Bodner (2008) did, however, propose a slight adjustment of Working Memory Theory. They hypothesized that "the eye movement benefits occur at the level of the central executive" (p. 923). That is, the eye movements may not only tax the VSSP, but might also draw upon the central executive, a memory system that is capable of directing attention to relevant information. An overload of the central executive may thus lead to attention being diverted from the task at hand. Crucially, the *central executive account* predicts that there is nothing special about the eye movements and that any distractor task, primarily visual or not, 'should do the trick'. In line with this hypothesis, a third experiment demonstrated that not only making eye movements, but also listening to a speech recording causes decreases in memory vividness and emotionality. This finding was corroborated by other experiments, for example demonstrating the efficacy of mental arithmetic (Van den Hout et al., 2010).

Several other predictions made by the working memory account have been tested and convergently support the theory. For example, Engelhard, van den Hout, and Smeets (2011) showed that there is an inverted-U relation between the level of taxation and the size of the effects on memory emotionality. That is, relatively easy tasks (causing minimal distraction) and relatively hard tasks (causing too much distraction to be able to recall the memory at all) show less beneficial effects compared to tasks that are in between. Furthermore, individual working memory capacity should be related to dual task performance and therefore should also correlate with the size of the dual task's effects. This has indeed been found (Gunter & Bodner, 2008, Exp. 3; Van den Hout et al., 2010).

In conclusion, EMDR is an effective therapy for PTSD and its key component, the execution of horizontal eye movement during recall of the trauma memory, adds to its effectiveness and its efficacy can be explained in cognitive psychological terms.

Combining two fields of research

Now that we have a better understanding of how the eye movements in EMDR work, there may also be new areas of application. Before shifting the focus to EMDR, this introduction outlined the major challenge in the treatment of anxiety-related disorders: reducing the return of fear. It was explained that fear reduction via UCS deflation might be associated with less return of fear. However, as elucidated later on, the UCS deflation techniques that have been documented in the literature seem to have limited clinical application. In this context, the current thesis will examine whether the execution of eye movements during aversive ideation, a technique that is used in EMDR therapy and thus has clinical applicability, can be used as a UCS deflation process. Theoretically, this can be expected, because the dual task typically results in a less negative reassessment of aversive memory. Specifically, it will be tested whether the dual task is a valuable technique in the attenuation of fear renewal.

Thesis outline

The current thesis comprises five research chapters (chapters 2-6) that each aim to answer a specific question. The goal of **chapter 2** was to get an (even) better understanding of how the eye movements in EMDR work. Specifically, the chapter examines whether the immediate reductions in memory vividness and emotionality are maintained at a 24 h follow-up test. The results of this study shed will light on what mechanism may underlie the (relatively) long-term benefits that are typically observed following EMDR treatment.

Chapter 3 describes a study that investigated whether the dual task that is used in EMDR can be conceptualized as a UCS deflation process. Specifically, we tested whether eye movements during recall of a UCS, relative to a control condition, reduces memory emotionality and consequently diminishes conditional fear. **Chapter 4** examines whether the dual task causes attenuation of fear renewal after extinction. An *ABA-renewal* paradigm was used, in which fear conditioning takes place in context "A", extinction takes place in context "B", and testing takes places in context "A" again. This paradigm robustly elicits context-driven return of fear. We tested whether eye movements during recall of the UCS, just before returning to context "A", reduces the intensity of renewed fearful responding.

Chapter 5 describes a study that investigated the relation between UCS deflation and (renewed) fearful responding. Specifically, we explain that the intensity of conditional responding depends on (1) the subjective probability of UCS occurrence, and (2) the subjective cost of the UCS, and that especially the latter should be affected by UCS deflation. Therefore, we expected that the anticipated reduction in fear renewal caused by UCS deflation is primarily expressed in UCS cost estimates. The aim of this study was thus to provide a better understanding of *how* UCS deflation techniques, such as eye movements during UCS recall, may counter fear renewal.

Finally, in **chapter 6,** a potential risk of the eye movement intervention is addressed. We explain that the blurring of potentially useful information, i.e. perceptual features of stimuli that signal danger, may actually become a risky endeavor. That is, it can be expected that a decrease in the vividness and/or the accessibility of a CS representation increases the threshold for perceptually similar stimuli to be categorized as 'safe'. Accordingly, it was hypothesized that eye movements during CS recall broadens the range of stimuli capable of evoking a fear response. Clearly, such an outcome is undesirable and would imply that therapists should be alert in determining what memory features to include in the dual task.

The thesis will conclude with a general discussion (**chapter 7**).

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Chapter 2

How eye movements in EMDR work: Changes in memory vividness and emotionality

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Abstract

Eye movements (EM) during recall of an aversive memory is a treatment element unique to Eye Movement Desensitization and Reprocessing (EMDR). Experimental studies have shown that EM reduce memory vividness and/or emotionality shortly after the intervention. However, it is unclear whether the immediate effects of the intervention reflect actual changes in memory. The aim of this study was to test whether immediate reductions in memory vividness and emotionality persist at a 24 h follow up and whether the magnitude of these effects is related to the duration of the intervention. Seventy-three undergraduates recalled two negative autobiographical memories, one with EM ("recall with EM") and one without (“recall only"). Half of participants recalled each memory for four periods of 24 s, the other half for eight periods of 24 s. Memory vividness/emotionality were self-rated at a pre-test, an immediate post-test, and a 24 h follow-up test. In both duration groups, recall with EM, but not recall only, caused an immediate decrease in memory vividness. There were no immediate reductions in memory emotionality. Furthermore, only the ‘eight periods’ group showed that recall with EM, but not recall only, caused a decrease in both memory emotionality and memory vividness from the pre-test to the follow-up. The findings suggest that recall with EM causes 24-h changes in memory vividness/emotionality, which may explain part of the EMDR treatment effect, and these effects are related to intervention duration. A limitation is that only self-report measures were used.

Introduction

Eye movement desensitization and reprocessing (EMDR) is a treatment for posttraumatic stress disorder (PTSD; APA, 2004; NICE, 2005), and an element unique to EMDR is that patients are instructed to make eye movements (EM) while recalling traumatic memories. Meta-analyses have shown that EMDR for PTSD is effective (e.g., Bisson et al, 2007; Seidler & Wagner, 2006), and that the EM component adds to its effects (Lee & Cuijpers, 2013). Until recently, however, there was little consensus on *how* EM might contribute to the effectiveness of EMDR (for an overview and critical testing of the competing theories, see Gunter & Bodner, 2008). This is relevant because theory on how EM work may inform how to best apply the EM in EMDR therapy.

Recent research has focused on the mechanism that underlies the EM effect and there is now much experimental data that support a working memory (WM) account (e.g., Van den Hout & Engelhard, 2012; Gunter & Bodner, 2008; Maxfield, Melnyk, & Hayman, 2008). WM theory emphasizes the limited capacity of WM and the finding that when two demanding tasks are performed simultaneously performance degrades (e.g., Baddeley & Andrade, 2000). Accordingly, when both EM and mental imagery are performed simultaneously, short-term storage capacity and rehearsal processes are divided and the memory comes to mind in a degraded form. Early evidence for WM theory came from experimental studies that showed that EM during recall of negative autobiographical memories renders imagery less vivid and emotional *while* EM are made compared to recall without EM (Andrade, Kavanagh, & Baddeley, 1997, exp. 4; Kavanagh, Freese, Andrade, & May, 2001).

These results, however, cannot account for the clinical observation that EMDR affects the vividness/emotionality of memory recall *after* a treatment session. Therefore, other studies have included a test in which memory vividness/emotionality were assessed before and shortly after the intervention phase. These studies consistently demonstrated that the effects of EM extended beyond the intervention (e.g., Engelhard, Van den Hout, Janssen, & Van der Beek, 2010; Gunter & Bodner, 2008; Kemps & Tiggemann, 2007; Van den Hout et al., 2001; Van den Hout et al., 2011a). Thus, the experimental data corroborate clinical observations, but it is unclear how the effects shortly after the intervention might be explained. Van den Hout and colleagues (2001) offered two competing accounts. First, because the post-test takes place *immediately* after the intervention, the memory vividness/emotionality ratings at the post-test may become confused with what was experienced during the intervention. Accordingly, reductions in memory vividness/emotionality from the pre-test to the post-test may *not* reflect

changes in the phenomenological quality of memory. If this is true, one would not expect any reductions caused by EM from pre-test to a substantially delayed post-test. Second, the observed reductions from the pre-test to the post-test may reflect actual *changes* in memory. Several authors have suggested that the EM procedure changes the experience of the memory and that this changed memory is (re)consolidated in long-term memory (e.g., Maxfield et al., 2008; Van den Hout et al., 2010; Van den Hout & Engelhard, 2012). If this explanation is correct, then one would expect reductions in memory vividness/emotionality from the pre-test to a delayed post-test. Thus, to get a better understanding of how EM work, it is relevant to investigate (relatively) long-term effects.

To the best of our knowledge, five studies have been conducted so far that (1) compared a "recall with EM" to a "recall only" condition and (2) employed a pre-test and a delayed post-test (i.e., a 1-week follow-up) (Gunter & Bodner, 2008, exp. 2; Kavanagh et al., 2001; Lee & Drummond, 2008; Lilley, Andrade, Turpin, Sarbin-Farrell & Holmes, 2009; Schubert, Lee & Drummond, 2011), and one of them used a clinical sample (Lilley et al., 2009). To determine whether these studies provide evidence that EM change memory, we examined the differences between ratings at pre-test and ratings at follow-up (cf. Gunter & Bodner, 2008, exp. 2). The alternative method, to compare ratings at immediate post-test to ratings at follow-up, is subordinate, because, as noted before, it is unclear what the ratings at immediate post-test reflect (Van den Hout et al., 2001). All five studies assessed memory vividness. One did not find long-term decreases, neither in recall with EM nor in recall only (Lilley et al., 2009), and one only reported that changes over time did not differ between the two interventions (but not whether there was a main effect of time; Lee & Drummond, 2008). Two studies showed that vividness ratings were lower at follow-up than at pre-test in both recall with EM and recall only (Kavanagh et al., 2001; Schubert et al., 2011). Only Gunter and Bodner (2008) demonstrated that reductions were larger in recall with EM than in recall only, which means that the decreases in memory vividness could be attributed to the EM procedure rather than imaginal exposure. Thus, only one out of five studies showed that EM caused long-term decreases in memory vividness. Three of the five studies also assessed memory emotionality (Gunter & Bodner, 2008; Kavanagh, et al., 2001; Lilley et al., 2009). One reported that memory emotionality ratings were lower at follow-up than at pre-test in both recall with EM and recall only (Kavanagh et al., 2001; there was only a marginal effect in Lilley et al., 2009). Again, only in Gunter and Bodner (2008) reductions were larger in recall with EM than in recall only. Thus, only one out of three studies showed that EM caused long-term decreases in memory emotionally.

Overall, it may be concluded that the findings are mixed and there is yet little evidence that EM change memory vividness/emotionality. The inconclusiveness of the data may not be surprising considering the diversity in experimental designs. Kavanagh et al. (2001) speculated that the absence of long-term effects in their study may have been affected by their short recall duration (64 s), which may also account for the null results reported in Lilley et al. (2009) who used an identical recall period (also note that Lilley et al. tested a clinical sample that may not benefit from such a brief intervention). For EM effects to be detected after one week, perhaps a longer intervention is needed. In line with this suggestion, the only study that demonstrated long-term changes employed a recall period of 96 s (Gunter & Bodner, 2008). In addition, both Lee and Drummond (2008) and Schubert et al. (2011) used recall periods up to 45 min but did not assess memory emotionality. It is thus unclear whether changes in memory emotionality persist or disappear when the intervention duration exceeds 96 s.

The goal of the current experiment was (1) to replicate Gunter and Bodner's (2008) reductions in memory vividness and emotionality at a delayed post-test and (2) to test whether intervention duration is involved in the magnitude and/or detection such effects. Whereas the studies discussed earlier scheduled their follow-up test seven days after the intervention, we tested 24 h later. Note that the previous studies aimed to test 'long-term' effects of the EM procedure. In contrast, the objective of the current study was to critically test an explanation of the well-documented 'immediate' effects of the EM procedure. As extending the period in between intervention and follow-up likely increases drop-out rates, and thereby reduces power, the current design seems to provide a better test of our hypothesis. All participants recalled one aversive autobiographical memory with EM and one without. To investigate the effect of intervention duration half of participants recalled each memory for four periods of 24 s (cf. Gunter & Bodner, 2008), the other half for eight periods of 24 s. Memory vividness and emotionality were self-rated at a pre-test, a post-test, and a 24 h follow-up. We predicted that (1) reductions from the pre-test to the follow-up would be larger for recall with EM than for recall only and (2) that intervention duration would be positively related to the magnitude of these effects.

Methods

Participants

Seventy-three undergraduates with a mean age of 21.52 years (range = 18-28; *SD* = 2.30; 44 women) participated in exchange for a financial reward or course credits. They were

randomly assigned to one of two groups: 'four periods', *n* = 36; 'eight periods', *n* = 37. Exclusion criteria were knowledge about EMDR, prior participation in an experiment that required participants to recall memories, or recent intake of tranquilizers. Sixty-nine participants (response rate: 95%; four periods, *n* = 34; eight periods, *n* = 35) returned for the follow-up assessment.

Materials and procedure

Participants were seated in a dim room about 45 cm in front of a computer screen. After receiving oral and written instructions, they provided written informed consent. By order of appearance they were assigned to the four periods or eight periods group. On day 1, all participants recalled one memory with eye movements ("EM") and another memory without (recall only: "RO") following the procedure used by Van den Hout et al. (2001). They provided ratings for memory vividness and emotionality before and shortly after the intervention, and 24 h later.

On day 1, the experiment started with a memory selection phase. Participants were instructed: 'Try to recall two autobiographical events that make you fearful or sad and still have emotional impact on you, for example ‘going unprepared into an examination’ or ‘witnessing an accident’. Form an image of each memory and write down some keywords on a label that easily remind you of the memory'. They were left alone for a few minutes and then reported their memories to the experimenter, rated unpleasantness of each memory (0=*not unpleasant at all*, 10=*very unpleasant*), and recalled another event when their rating was lower than 6. Based on these ratings, memories were ranked and balanced over the EM and RO conditions.

Next, there was a pre-test, a recall phase, and a post-test for each condition. These phases directly followed each other up and order of conditions was balanced. First, participants were provided one of the two memory labels and received instructions: 'Form an image of this memory and keep your eyes open. Remember where it happened, who was present, and anything else you can think of. Bring it to mind as if it were happening right now and please indicate when the image is vivid'. After 10 s of recall, participants rated their memory on 100 mm computerized visual analogue scales (VASs) for vividness, emotionality, and difficult recalling (0 = *not vivid/unpleasant/difficult at all,* 100 = *very vivid/unpleasant/difficult*). Then, participants recalled their memory four or eight times for 24 s and simultaneously followed a grey dot (Ø 1 cm) that horizontally moved 21.5 cm across a black computer screen at 1 cycle per second (EM), or looked at a black computer screen (RO). Recall periods were separated by 10 s breaks during which participants were instructed to stop recalling the memory and to focus on something else. At the post-test, they recalled their memory again for 10 s and rated it for

vividness and emotionality. When both conditions were finished, participants rated for each condition to what extent they actually recalled their memory during the 24 s recall periods (0=*not at all*; 100=*all the time*). Finally, they were asked to return to the lab 24 h later.

After a 24 h break (*M* = 24 h 22 min, *SD* = 37 min, range = 23 h 55 min - 27 h 10 min) participants recalled each memory again for 10 s, in balanced order, and rated its vividness and emotionality. They were then debriefed and given compensation.

Data analyses

There were 3 outliers in the four periods group and 1 in the eight periods group that were replaced[1] by *M* ± 2.5 *SD*. Immediate changes in memory ratings were analyzed by repeated measures ANOVAs with Time (pre-test vs. post-test) and Condition (EM vs. RO) as within-subjects factors and Duration (four periods vs. eight periods) as between-subjects factor. Twenty-four-hour changes were investigated with similar analyses that compared the ratings at pre-test and follow-up. To control for Type I error rates for multiple comparisons, the Bonferroni correction was applied to post-hoc comparisons, which resulted in testing at a .005 (.05/10) alpha level. Tests that were crucial to the hypothesis were one-tailed.

Results

Randomization check and a priori differences

No differences were found between the two duration groups in age, $t(71) = 1.45$, $p = .15$, or gender ratio, $\chi^2(1, N = 73) = 1.67$, $p = .20$. Furthermore, Duration x Condition ANOVAs on pre-test ratings showed no main effects or interactions for memory vividness, largest $F(1, 71) = 1.02$, $p = .32$, memory emotionality, Fs < 1, or difficulty recalling the memories, Fs < 1, meaning that randomization of memories to conditions and duration groups was successful. As expected, during EM (*M* = 38.59, *SD* = 25.14) participants were less able to recall their memory than during RO (*M* = 75.37, *SD* = 18.34), $F(1, 71) = 141.02$, $p < .001$, $\eta\rho^2 = .67$, which did not differ between duration groups, $F < 1$.

Immediate effects

First, we tested whether the widely reported immediate effects of EM were replicated.

Vividness

The ANOVA showed no main effects for Time, $F(1, 71) = 2.73$, $p = .10$, Condition, $F(1, 71) = 3.36$, $p = .07$, or Duration, $F(1, 71) = 1.58$, $p = .21$. There were interaction effects for Time x Condition, $F(1, 71) = 15.95$, $p < .001$, $\eta\rho^2 = .18$, and Time x Duration, $F(1, 71) = 4.39$,

[1] The reduction of outliers did not influence the absence or presence of effects.

$p < .05$, $\eta\rho^2 = .06$, but not for Condition x Duration, $F < 1$. The three-way interaction was not significant, $F < 1$, meaning that changes over time differed between EM and RO, irrespective of intervention duration. *T*-tests revealed that vividness ratings decreased in EM, $t(72) = 3.30$, $p = .002$, $d = .40$, but did not change in RO, $t(72) = 1.39$, $p = .17$. Thus, irrespective of the intervention duration, EM, but not RO, caused an immediate decrease in memory vividness (see Figure 1).

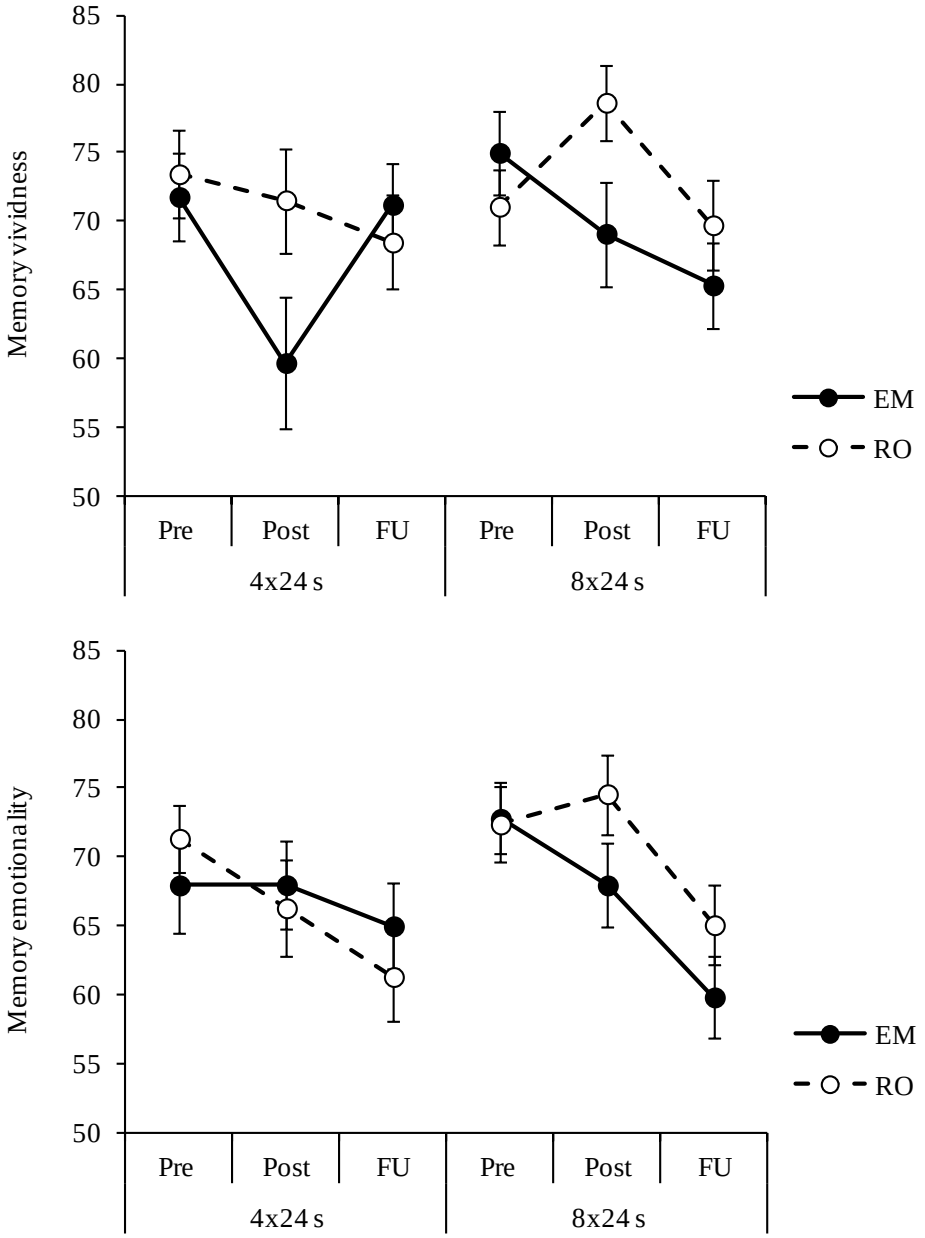

Fig. 1 Mean memory vividness (upper panel) and memory emotionality ratings (lower panel) before (Pre) and immediately after the intervention (Post), and at 24 h follow-up (FU). Error bars indicate SEM.

Emotionality

The analysis on emotionality ratings did not yield main effects or two-way interactions, largest $F(1, 71) = 1.77$, $p = .19$. There was, however, a three-way interaction, $F(1, 71) = 6.65$, $p < .05$, $\eta\rho^2 = .09$. Follow up 2x2 ANOVAs for each duration group showed no effects after four periods, largest $F(1, 35) = 1.73$, $p = .20$, and only a Time x Condition interaction effect after eight periods, $F(1, 36) = 6.70$, $p < .05$, $\eta\rho^2 = .16$. *T*-tests revealed that emotionality ratings did not decrease in either EM, $t(36) = 2.03$, $p = .05$, or RO, $t(36) = 1.04$, $p = .31$. Thus, no immediate reductions in memory emotionality were found.

Twenty-four-hour effects

Vividness

A comparison between pre-test and follow-up ratings showed a main effect for Time, $F(1, 67) = 5.82$, $p < .05$, $\eta\rho^2 = .08$, but not for Condition or Duration, Fs < 1. There were no two-way interactions, largest $F(1, 67) = 1.09$, $p = .30$, but the three-way interaction was significant, $F(1, 67) = 4.36$, $p < .05$, $\eta\rho^2 = .06$. To understand this interaction, Time x Condition ANOVAs were performed for each duration group. In the four periods group there were no main effects, Fs < 1, or interaction effect, $F(1, 33) = 1.05$, $p = .31$. In the eight periods group there was a main effect for Time, $F(1, 34) = 5.85$, $p < .05$, but not for Condition, $F < 1$. Crucial to our hypothesis, the Time x Condition interaction was significant, $F(1, 34) = 3.73$, $p = .031$ (one-tailed), $\eta\rho^2 = .10$. *T*-tests revealed that in EM vividness ratings were lower at follow up than at pre-test, $t(34) = 3.07$, $p = .004$, $d = .59$, but not in RO, $t < 1$.

Emotionality

A similar analysis on emotionality ratings showed only a main effect of Time, $F(1, 67) = 28.30$, $p < .001$, $\eta\rho^2 = .30$, and a three-way interaction, $F(1, 67) = 7.30$, $p < .01$, $\eta\rho^2 = .10$, all other Fs < 1. To break down the interaction effect, Time x Condition ANOVAs were performed for each duration group. In the four periods group there was no main effect for Condition, $F < 1$, but there was a main effect for Time, $F(1, 33) = 8.30$, $p < .01$, $\eta\rho^2 = .20$, and a significant interaction effect, $F(1, 33) = 4.42$, $p < .05$, $\eta\rho^2 = .12$. *T*-tests showed that, in contrast to expectations, emotionality ratings decreased in RO, $t(33) = 3.99$, $p < .001$, $d = .67$, but not in EM, $t(33) = 1.05$, $p = .30$. In the eight periods group there was no main effect for Condition, $F < 1$, but there was a main effect for Time, $F(1, 34) = 23.68$, $p < .001$, $\eta\rho^2 = .41$, which was, as hypothesized, qualified by a Condition x Time interaction, $F(1, 34) = 2.98$, $p = .047$ (one-tailed), $\eta\rho^2 = .08$. *T*-tests showed that emotionality ratings dropped in EM, $t(34) = 4.77$, $p < .001$, $d = .79$, but not in RO, $t(34) = 2.53$, $p = .02$ (note that $\alpha = .005$).

In sum, four periods of recall only caused a decrease in memory emotionality at the 24-h follow-up and eight periods of recall with EM caused decreases in both memory vividness and memory emotionality at the 24-h follow-up.

Discussion

The aim of this investigation was to replicate Gunter and Bodner's (2008) finding of EM effects at a delayed post-test and to test whether the magnitude of these effects is related to intervention duration. On the one hand, we did not replicate Gunter and Bodner (2008): our four periods group (cf. Gunter & Bodner, 2008) did not show changes in memory vividness/emotionality from the pre-test to the follow-up. This may relate to the immediate reductions that were smaller in our study. By comparing the two duration groups in the present study (Figure 1), it may be hypothesized that direct drops in scores are predictive of reductions 24 hrs later for memory emotionality, but not for memory vividness. Indeed, correlation analyses showed that immediate reductions caused by EM were related to reductions 24 h later for memory emotionality, $r = .55$, $p < .001$, but not for memory vividness, $r = .22$, $p = .064$. On the other hand, our eight periods group did provide the first replication of Gunter and Bodner (2008) that recall with EM produces changes in memory vividness/emotionality 24 h later compared to recall only. Hence, the hypothesis that intervention duration is positively related to the magnitude of these effects was confirmed.

In our four periods group we did not find effects of EM at the follow-up test, which agrees with previous investigations that employed a relatively short intervention duration (64 s; Kavanagh et al., 2001; Lilley et al., 2009). The authors of these studies speculated that EM do not directly affect desensitization (i.e., reduce emotional reactivity to the memory) but may rather serve an aid that facilitates exposure to highly emotive memory. This explanation, however, contradicts our findings in the eight periods group as well as Gunter and Bodner's (2008) follow-up effects. Gunter and Bodner (2008) found that immediate reductions in memory emotionality persisted at the follow-up and that immediate reductions in memory vividness were significantly smaller at the follow-up. They speculated that EM may not permanently change memory vividness, but may rather facilitate imaginal exposure (Kavanagh et al., 2001) and thereby foster desensitization. Alternatively, they also speculated that their intervention duration (96 s) may have been too short to produce persisting effects in memory vividness. The present findings support the latter hypothesis. The eight periods group employed a recall period that was twice as long as Gunter and Bodner (2008), and produced reductions in

memory vividness shortly after the intervention that persisted one day later. In addition, compared to the immediate post-test memory emotionality ratings were even lower at the follow-up. Together, these findings suggest that EM during recall may have long-term effects on emotional memories. Moreover, and in line with our hypothesis, these effects were related to intervention duration.

The findings have several implications. First, they provide corroborating evidence that EM change memory vividness and emotionality, which may explain part of the EMDR treatment effect. They are consistent with prior research suggesting that EM reduce memory accessibility (Van den Hout, Bartelski, & Engelhard, 2013) and with other work demonstrating that EM attenuate fear potentiated startle responses during recall of the memory (Engelhard, Van Uijen, & Van den Hout, 2010). The 24-h changes further correspond with recent suggestions that the reductions in vividness/emotionality during the intervention retrievals are reconsolidated in long-term memory and therefore recall of the 'updated' memory elicits less distress (e.g., Maxfield et al., 2008; Van den Hout et al., 2010; Van den Hout et al., 2011a). Note, however, that the mere observation of lasting effects does not proof the reconsolidation account of EM. Reconsolidation, by definition, implies that the original memory trace is changed (Nader & Hardt, 2009). There are at least two alternative explanations. First, it may be the case that EM cause the formation of a *new* memory trace, like exposure to a feared stimulus leads to the formation of a new memory (i.e., that the feared stimulus is not predictive of a catastrophic outcome) rather than erasing or updating the original 'fear memory' (Vervliet, Craske, & Hermans, 2013). Fear conditioning experiments have shown that retrieval/ expression of the new extinction memory is context-specific, i.e. extinction memory is typically activated in the context where it was created (e.g., Bouton 2002; Hermans, Craske, Mineka, & Lovibond, 2006; Vervliet et al., 2013). Notably, this (at least partially) explains the return of fear after extinction. Future research may test the hypothesis that EM affect updating of the original memory and that EM effects generalize across contexts. Second, the observed changes in ratings may reflect shifts in how participants *relate* to their memory rather than phenomenological changes. As Gunter and Bodner (2008) put it: "By experiencing the memory in a weakened form (…) people may change some of their beliefs about how dangerous their memories are, as well as their beliefs about their ability to cope with remembering them". Changes in memory emotionality may thus reflect changes in memory *appraisal*. In contrast, the (often) observed changes in memory vividness point towards changes in the phenomenological quality of the memory. One way to gain more insight into effects of EM on memory is to use a more comprehensive assessment of memory features (e.g., including

multiple perceptual and contextual details), such as the Memory Characteristics Questionnaire (Johnson, Foley, Suengas, & Raye, 1988).

The findings further encourage future studies that investigate (relatively) long-term effects of EM to use an intervention duration of eight periods of 24 s or longer. Note, however, that two studies using healthy student samples and sessions of up to 45 min of imaginal exposure with or without EM did not find direct or long-term differential reductions in memory vividness (Lee & Drummond, 2008; Schubert et al., 2011). These studies suggest that, in nonclinical samples, prolonged imaginal exposure with EM may be equally effective as prolonged imaginal exposure only. As the current hypothesis was tested using a student sample and required the presence of immediate effects, we employed an intervention that lasted only a few minutes. Future studies using clinical samples or investigating different hypotheses (e.g., about the optimal intervention duration) may be advised to employ a greater number of trials.

Several other issues deserve further attention. First, the results regarding immediate reductions corroborate earlier findings that EM during recall of aversive memory, relative to recall only, cause a *direct* decrease in memory vividness (e.g., Engelhard, Van den Hout et al., 2010; Gunter & Bodner, 2008; Kemps & Tiggemann, 2007; Kristjánsdóttir & Lee, 2011; Smeets, Dijs, Pervan, Engelhard, & Van den Hout, 2011; Van den Hout et al., 2001; Van den Hout et al., 2011a). Yet, in contrast to most earlier investigations (but see Maxfield et al., 2008, exp. 1; Van den Hout et al., 2011b), EM did not cause direct reductions in memory emotionality. Note, however, for the eight periods group, that both Figure 1 and the medium effect size of drops in scores (r = .32) suggest that the lack of statistical evidence for an effect resulted from the rather conservative testing procedure. Next, we found that four periods of mere recall caused a drop in memory emotionality at the delayed post-test. As this finding is inconsistent with both our expectations and previous findings (Gunter & Bodner, 2008; Kavanagh, et al., 2001; Lilley et al., 2009), we believe that at present any conclusions regarding this effect are premature. Finally, previous research suggests there is an optimal degree of taxing WM, i.e. that the dose-response relationship with beneficial effects is inverted U-shaped rather than linear (Engelhard, Van den Hout, & Smeets, 2011). Future studies may investigate whether there is an optimal intervention duration.

There were several limitations of this study. First, only self-report measures were used and demand effects cannot be ruled out. However, such effects seem unlikely. It has been shown that EM benefits occur when EM are made *during* recall but not when made *before* recall, while participants' expectancies about their effectiveness should not differ (Gunter & Bodner, 2008, exp. 1). Furthermore, EM effects have been replicated with measures that are less prone to

demand effects, like the fear potentiated startle response (Engelhard et al., 2010) and reaction times (Van den Hout et al., 2013). Second, a nonclinical sample was tested. Kavanagh et al. (2001) pointed out that student and clinical samples may differ in the degree of intrusiveness and emotionality of the memories involved. These groups may also differ in working memory capacity, which correlates with the effectiveness of EM (Gunter & Bodner, 2008; Van den Hout & Engelhard, 2012). It is therefore unclear whether the present findings may be generalized to clinical populations.

In sum, this study provides corroborating evidence that EM during recall causes reductions in memory vividness and emotionality at a delayed post-test and that the magnitude of these effects is related to intervention duration. Future investigations may focus on the optimal intervention duration.

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Chapter 3

Eye movements during recall of aversive memory decreases conditioned fear

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Abstract

Cognitive-behavioral therapy for anxiety disorders typically involves exposure to the conditioned stimulus (CS). Despite its status as an effective and primary treatment, many patients do not show clinical improvement or relapse. Contemporary learning theory suggests that treatment may be optimized by adding techniques that aim at revaluating the aversive consequence (US) of the feared stimulus. This study tested whether US devaluation via a dual task – imagining the US while making eye movements – decreases conditioned fear. Following fear acquisition one group recalled the US while making eye movements (EM) and one group merely recalled the US (RO). Next, during a test phase, all participants were re-presented the CSs. Dual tasking, relative to the control condition, decreased memory vividness and emotionality. Moreover, only in the dual task condition reductions were observed in self-reported fear, US expectancy, and CS unpleasantness, but not in skin conductance responses. Findings provide the first evidence that the dual task decreases conditioned fear and suggest it may be a valuable addition to exposure therapy.

Introduction

Although cognitive-behavioral therapy (CBT) is the primary and most effective treatment for anxiety disorders (Deacon & Abramowitz, 2004; Hofmann & Smits, 2008, NICE, 2011), there is room for improvement. About 20% to 50% of patients do not show clinically significant change after treatment (Barlow, Allen, & Choate, 2004). Moreover, relapse rates are considerable. For example, relapse rates are 18.5% to 23% among panic disorder patients (2-14 years post-treatment; Fava, Zielezny, Savron, & Grandi, 1995; Fava et al., 2001), and 13% among social phobia patients (2-12 years post-treatment; Fava, Grandi et al. 2001). Fear may return when CSs are encountered outside the extinction (i.e., therapeutic) context (renewal of fear), by the mere passage of time (spontaneous recovery), or because of confrontations with the US after fear extinction (reinstatement) (for reviews, see Bouton, 2002; Hermans et al., 2006). The explanation for these phenomena is that the fear memory ("CS predicts US") is not erased or destroyed as a result of exposure-based extinction learning. Rather, a new extinction memory ("CS does not predict US") is formed (e.g., Craske, Liao, Brown, & Vervliet, 2012). Since extinction learning hardly generalizes across contexts, confrontations with CSs outside the therapeutic context more readily activate the acquisition memory than the extinction memory, which causes a return of fear (e.g., Bouton, 2002). Therefore, several CSs and contexts may need to be targeted during exposure-based therapy in order to successfully reduce fear.

A theoretical suggestion on how to further optimize treatment comes from Davey's refined model of fear conditioning (1997). According to this model, the conditioned response (CR) is not only a consequence of the strength of the CS-US association, but also of the cognitive representation of the US. Accordingly, there are two ways to influence the strength of the CR (i.e., the fear response). The first pathway focuses on the memory-encoded association between the CS and the US. Strengthening the CS-US association (e.g., increasing the number of CS-US pairings) intensifies the CR, whereas weakening the CS-US association (e.g., CS presentations in absence of the US, as in exposure therapy) results in a decreased CR. The second pathway is nonassociative and capitalizes on revaluation processes that may affect the UCS representation. For example, post-conditioning information that suggests that the US is more threatening than previously conceived, may cause US *inflation*, which should lead to an increase in conditioned fear. This has indeed been found. After establishment of a CS-US association, experience with a similar US of greater intensity causes later CS presentations to elicit a stronger CR (White & McKenna, 1989). More important for the current issue, the US may be reassessed more favorably when information is acquired that suggests that the US is

less aversive than during conditioning, called US *devaluation*, which should decrease conditioned fear. Laboratory studies have shown that exposure to the US, in absence of a CS, that produces diminished responding to the US also results in a weaker CR upon next CS presentations (Davey & McKenna, 1983). Similarly, experience with the US at lower intensity compared to US intensity during acquisition evokes a weaker CR upon next CS presentations (Hosoba, Iwanaga, & Seiwa, 2001).

In view of their potential practical application it may be more fruitful to study memory processes that result in US devaluation, but do not require the physical presence of the US. For example, rehearsal (i.e., repeated imagination) of the US after fear acquisition maintains (Arntz, Spit, & Merckelbach, 1997; Jones & Davey, 1990; Joos, Vansteenwegen, & Hermans, 2012a) or increases (Davey & Matchett, 1994) conditioned responding. In line with Davey's theory, increases in self-reported aversiveness of the US are accompanied by increases in CR-strength (Matchett & Davey, 1995). Similar results were found for instructed worrying about the aversive consequences of a US (Gazendam & Kindt, 2012). Indeed, US devaluation by mental imagery may result in *reduction* of conditioned fear. Tentative support for this hypothesis was provided by Dibbets, Poort, and Arntz (2012) who tested the usefulness of imagery rescripting (IR; an effective treatment for various anxiety disorders; Holmes & Mathews, 2010) in reducing the return of fear after extinction. Participants first learned the association between a CS (picture of a car) and a US (picture of a mutilated child). Then the intervention group verbally rescripted the mental image of the US during extinction trials, while the control group was only exposed to extinction trials, which attenuated conditioned fear to the CS in a subsequent test phase. It should be noted, however, that both during the extinction phase and at offset, CR was stronger in the intervention condition than in the control condition, presumably because participants rehearsed the CS-US association as a part of IR *during* the extinction trials. Though the authors did correct for the offset difference, the question remains whether differences in the extinction process may have affected renewal.

The current study aimed to investigate another method to devalue a US representation that has been extensively studied in recent years. It involves a dual-task in which participants are typically instructed to visualize an aversive memory ('recall') and simultaneously make eye movements (EM) (e.g., Gunter & Bodner, 2008; Maxfield, Melnyk, & Hayman, 2008; Van den Hout, Muris, Salemink, & Kindt, 2001). Experiments have repeatedly shown that the dual-task, relative to recall alone, results in a decrease in self-reported memory vividness and emotionality. These findings are substantiated by non-self-report data like fear-potentiated startle (Engelhard, van Uijen, & van den Hout, 2010) and motor behavior i.e. reaction times

(Van den Hout, Bartelski, & Engelhard, 2012) data. Working memory theory offers an explanation: during the dual-task both tasks compete for limited working memory capacity (Andrade, Kavanagh, & Baddeley, 1997; Gunter & Bodner, 2008). As a result, the aversive memory will come to mind in a degraded form (i.e., less vivid and emotional) and will be reconsolidated as such (see Van den Hout & Engelhard, 2012).

As the dual-task resembles a memory devaluation technique, we expected that making EM during recall of the US devaluates the US representation and alleviates the CR to CSs. A differential conditioning paradigm was used, in which a CS+ was paired with aversive film fragments (US), and a CS- was not paired. Subsequently, participants recalled the US memory while making EM ("EM") or without EM (recall only: "RO"). The latter condition controlled for an imaginal exposure effect (cf. Gunter & Bodner, 2008; Engelhard & van Uijen, et al., 2010). Then, in a test phase, participants were exposed to CSs to test whether EM resulted in diminished CR. Conditioned fear was operationalized as ratings of self-reported fear and US expectancy, and skin conductance responses (as an objective measure of anxious arousal). In addition, we assessed evaluative CR, which involved ratings of CS pleasantness, because such responses are resistant to extinction (e.g., Engelhard, Leer, Lange, & Olatunji, 2014) and US revaluation has been shown to reduce the negative evaluation of the CS (Baeyens, Eelen, Van den Bergh, & Crombez, 1992; Walther, Gawronski, Blank, & Langer, 2009). Our hypotheses were that EM causes (1) a reduction in the vividness and emotionality of the US memory, (2) a reduction in conditioned fear, and (3) a reduction in the conditioned negative evaluation of the CS.

Method

Participants

Participants were 63 female students (mostly undergraduates), recruited at Utrecht University with a mean age of 22.83 years (range: 18-39, *SD* = 3.35) who participated for course credit or a financial reward. Exclusion criteria were prior knowledge about EMDR and prior participation in an experiment in which the dual-task paradigm was used. By order of appearance, participants were assigned to either the EM or the RO condition.

Stimuli

A 600 Hz low tone and a 1200 Hz high tone served as CSs. A disgusting film clip[1] from *YouTube* (Cjdragano, 2010) with images and sounds of a male vomiting in a toilet served as US. The film clip was split into 3 fragments of 4 s each that covered 25% of a black screen. The task was programmed using E-Prime 2.0 (Psychology Software Tools).

Questionnaires

State and trait anxiety were assessed with the 40-item State Trait Anxiety Inventory (STAI-DY; Spielberger, Gorsuch, & Lushene, 1970). Each construct contains 20 items that are scored on a 1-4-point scale (1=*not at all*, 4=*severely*). This questionnaire was included to allow for comparing the conditions on levels of anxiety, which been identified to affect fear learning (Grillon et al., 2006; Lissek et al., 2005).

Skin conductance responses

Two 9-mm Sensor Medics Ag/AgCl electrodes were attached to the medial phalanges of the middle and index fingers of the non-dominant hand (cf. Fowles et al., 1981). A constant current of 0.5 V was transmitted via the electrodes and registered with a Coulbourn Modular Instruments System (Allentown, PA, USA) via a Coulbourn Isolated Skin Conductance coupler (S71-23). SCRs were calculated by subtracting the mean SC level for the 2 s preceding CS onset from the largest value recorded during CS presentation (Pineles, Orr, & Orr, 2009). Amplitude changes smaller than 0.02 µS were scored as 0 and left in the analyses (cf. Soeter & Kindt, 2010). All values were then range corrected (SCR/SCR_{MAX}; Lykken & Venables, 1971) and transformed by square root to normalize the data (Dawson, Schell, & Filion, 2000). Finally, mean SCRs were calculated for each phase and outliers (> 2.5 *SD*) were replaced by $M \pm 2.5$ *SD*.

[1]A pilot study was conducted to select a film that participants could vividly recall and found distressing. Twelve students watched 4 clips (i.e., the current clip, a fragment from the aversive film used by Hagenaars, Van Minnen, Holmes, Brewin, & Hoogduin, 2008 and two clips showing a male vomiting used by Viar-Paxton & Olatunji, 2012) in counterbalanced order and rated each clip on unpleasantness (0=*not unpleasant at all*, 10=*very unpleasant*). They also rated their memory of each clip on vividness and emotionality (0=*not vivid/unpleasant at all*, 10=*very vivid/unpleasant*), and indicated how much they would dislike watching the clip again (0=*not at all*, 10=*very much*). The current film clip had highest ratings on unpleasantness ($M = 7.67$, $SD = 1.83$), memory vividness ($M = 8.63$, $SD = 1.03$), memory emotionality ($M = 7.00$, $SD = 2.05$), and aversion toward re-watching the clip ($M = 7.33$, $SD = 2.39$), Fs > 7.57, ps < .01. *T*-tests revealed that the current clip was rated higher than each of the other three clips on all of these measures, smallest $t = 2.92$, $p < .05$.

Procedure

Participants were seated in a dim, soundproof room, about 45 cm in front of a computer screen. They were informed about the experimental procedure, that disgusting film fragments would be shown, and they could decline further participation at any time. After providing written informed consent, they were asked to fill out the STAI. Next, they were given headphones and electrodes were attached. The experiment consisted of four phases of approximately 5 minutes each that directly followed each other: a habituation, acquisition, intervention and test phase.

Habituation phase. Each CS was presented twice in counterbalanced order. Participants were instructed to listen carefully to a high and a low tone and try to remember what was heard. CSs lasted for 6 s and were followed by a 14-s inter-stimulus-interval (ITI). Afterwards, participants recalled the last time they heard each tone (in counterbalanced order) and filled out two 100 mm Visual Analogue Scales (VASs) that were presented in a paper booklet: fear for the CS ('To what extent did you feel fearful when you heard this tone?' with 0=*no fear at all*; 100=*very fearful*) and CS pleasantness ('How pleasant did you find this tone?', with 0=*not pleasant at all*, 100=*very pleasant*).

Acquisition phase. Participants were told that both tones would be presented again, three times each, and one of them would be followed by a disgusting film fragment. They were encouraged to figure out after which tone the fragment was shown, and to pay close attention to each fragment, because some questions would be asked about them later. The CS+, but not CS-, was immediately followed by the 4-s US. Order of CSs was pseudo-random with a maximum of two similar consecutive CSs.

Afterwards, participants were asked which tone was consistently followed by the film fragment and to indicate on a VAS how confident they were about their answer (0=*not sure at all*; 100=*very sure*). Then, they rated fear for the CS and CS pleasantness again, and US expectancy on a VAS ('To what extent did you expect that this tone would be followed by a film fragment?', with 0=*not at all*, 100=*very much*). Finally, they completed two VASs that assessed the unpleasantness of the three combined film fragments ('How unpleasant did you find the film clip?', with 0=*not unpleasant at all*, 100=*very unpleasant*; 'How much would you dislike watching the film again?', with 0=*not at all*, 100=*very much*).

Intervention phase. Participants recalled the images and sounds of the three film fragments as vividly and detailed as possible, as if it was one film clip. After a 10-s recall period, they completed three VASs that assessed memory vividness and emotionality ('How vivid/unpleasant did you find the memory of the film that you just recalled?', with 0=*not*

vivid/unpleasant at all; 100=*very vivid/unpleasant*), and difficulty recalling the film clip (0=*not difficult at all*, 100=*very difficult*). Then, they were instructed to recall the film clip as vividly and detailed as possible, 4 times for 24 s each, with 10-s breaks in between (cf. Engelhard, Van den Hout, Janssen, & Van der Beek, 2010; Gunter & Bodner, 2008). During these recall periods, the EM group was instructed to visually track a grey dot (Ø 1 cm) that moved horizontally across the black screen (1 cycle per s). The RO group was instructed to keep their eyes open and watch the center of a black screen. After the intervention, participants recalled the film clip again for 10 s, as vividly and detailed as possible, and rated memory vividness and emotionality again.

Test phase. Instructions read that each tone would be presented once more and one of them might be followed by a previously presented film fragment. CSs were presented in counterbalanced order. The US was not presented during this phase, so that the US memory would not be updated and CS ratings would be unequivocal. Afterwards, participants rated fear for the CS, CS pleasantness and US expectancy, and to what extent they actually recalled their memory of the film clip during the intervention (0=*not at all*; 100=*all the time*) and during how many of the 4 recall periods (1, 2, 3, or 4). Finally, electrodes were removed, and participants were debriefed and given a financial reward or course credit.

Data analysis

Pearson correlation analyses showed that the memory vividness and emotionality ratings were moderately correlated (at pre-test: $r = .32$, $p = .01$; at post-test: $r = .24$, $p = .05$), suggesting appropriateness of MANOVA (Tabacknick & Fidell, 2007). Therefore, US devaluation was examined by a 2 (Condition: EM vs. RO) x 2 (Time: pre-test vs. post-test) split-plot MANOVA, followed by univariate analyses. The first hypothesis was tested using planned comparisons ($\alpha = .05$) that examined whether, in the EM condition, memory vividness and emotionality rating decreased from the pre-test to the post-test.

At the end of the acquisition phase, fear ratings and US expectancy ratings were moderately correlated (CS-: $r = .32$, $p = .02$; CS+: $r = .22$, $p = .08$), but these outcome variables did not correlated with SCR (largest $r = .08$, $p = .56$). Therefore, acquisition of conditioned fear was examined by a 2 (Condition) x 2 (CS Type: CS+ vs. CS-) split-plot MANOVA on fear ratings and US expectancy ratings, follow by univariate analyses, and a separate 2 x 2 ANOVA on SCRs (average acquisition trial 2 and 3). Acquisition of evaluative responding was investigated by a 2 x 2 ANOVA on CS pleasantness ratings.

At the end of the test phase, fear ratings and US expectancy ratings[2] showed moderate to high correlations (CS-: $r = .53$, $p < .001$; CS+: $r = .51$, $p < .01$). Therefore, the intervention effect on conditioned fear was investigated by a 2 (CS Type) x 2 (Time: acquisition phase vs. test phase) x 2 (Condition) split-plot MANOVA, followed by univariate analyses. The second hypothesis was tested using planned comparisons ($\alpha = .05$) that examined whether, in the EM condition, fear for the CS+ and US expectancy to the CS+ were lower at the end of the test phase compared to at the end of the acquisition phase. The intervention effect on evaluative responding was examined in a separate 2 x 2 x 2 split-plot ANOVA. The third hypothesis was tested using a planned comparison ($\alpha = .05$) that examined whether, in the EM condition, negative evaluation of the CS+ decreased from the end of the acquisition phase the end of the test phase.

To control for Type I error rates for multiple comparisons, the Bonferroni correction was applied to post-hoc comparisons and follow-up ANOVAs that did not lead to planned comparisons (i.e., ANOVAs for acquisition effects), which resulted in testing at a .0028 (.05/18) significance level.

Results

Randomization check

The conditions did not differ in age or questionnaire scores, *t*s < 1 (see Table 1). Furthermore, unpleasantness ratings of the US (EM: $M = 80.09$, $SD = 13.44$; RO: $M = 78.61$, $SD = 19.04$) and aversion towards re-watching the clip (EM: $M = 66.50$, $SD = 20.10$; RO: $M = 62.68$, $SD = 25.79$) were similar across conditions *t*s < 1.

Table 1. *Means (SD) for age and questionnaire data*

Condition	Age	Trait anxiety	State anxiety
RO	23.23 (3.45)	37.23 (7.75)	34.37 (6.50)
EM	22.48 (3.36)	38.97 (9.33)	35.35 (9.65)

[2] Because there was no evidence for the acquisition of SCR, SCR was not included as a dependent variable in the MANOVA on conditioned fear.

US devaluation

Figure 1 shows the mean memory vividness and emotionality ratings. Before the intervention, groups did not differ in memory vividness, $t(61) = 1.23$, $p = .22$, memory emotionality, $t < 1$, or difficulty recalling the memory (EM: $M = 14.38$, $SD = 10.17$; RO: $M = 16.00$, $SD = 15.41$), $t < 1$. Furthermore, there were no differences in the extent to which the US was recalled during the interventions (EM: $M = 74.81$, $SD = 19.66$; RO: $M = 82.71$, $SD = 17.43$), $t(61) = 1.69$, $p = .10$, and the reported number of recall periods in which participants actually recalled the US (EM: $M = 3.97$, $SD = .18$; RO: $M = 3.90$, $SD = .30$), $\chi^2(1, N = 63) = 1.14$, $p = .29$.

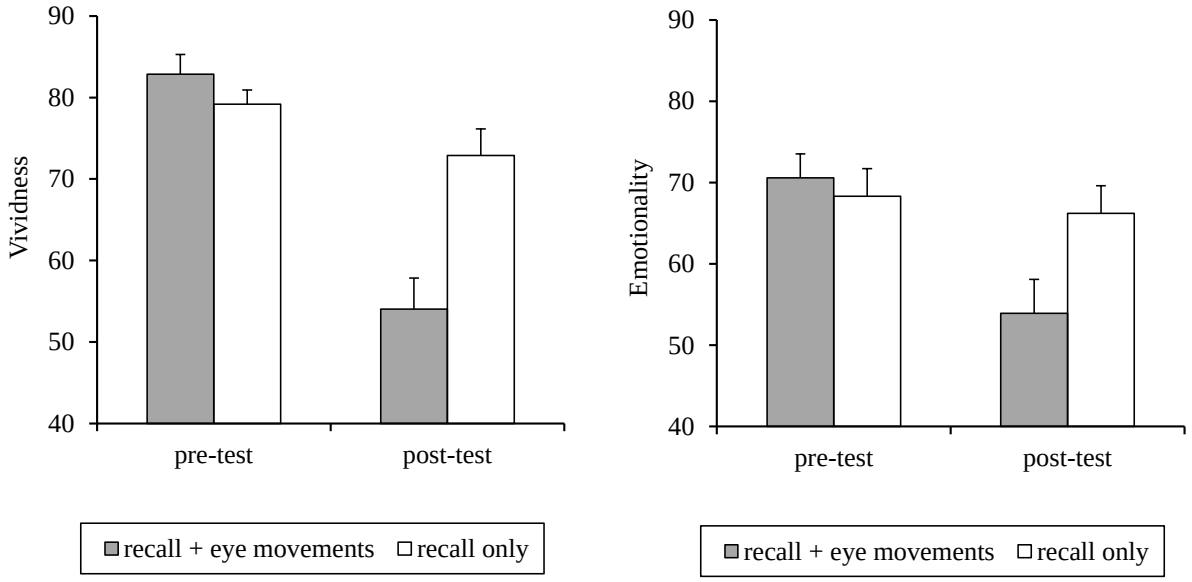

Fig. 1 Mean memory vividness and memory emotionality ratings before (pre-test) and after (post-test) the intervention. Error bars indicate SEM.

MANOVA showed a main effect of Time, $F(2, 60) = 33.78$, $p < .001$, $\eta\rho^2 = .53$, but not of Condition, $F(2, 60) = 2.64$, $p = .08$, and an interaction effect, $F(2, 60) = 15.30$, $p < .001$, $\eta\rho^2 = .34$. Univariate analyses on vividness ratings showed main effects of Time, $F(1, 61) = 56.84$, $p < .001$, $\eta\rho^2 = .48$, and Condition, $F(1, 61) = 4.96$, $p = .03$, $\eta\rho^2 = .08$, and an interaction effect, $F(1, 61) = 23.42$, $p < .001$, $\eta\rho^2 = .28$. The planned comparison revealed that from the pre-test to the post-test ratings decreased in EM, $t(31) = 7.56$, $p < .001$, $d = 1.60$. Post hoc comparisons indicated that ratings did not changes in RO, $t(30) = 2.39$, $p = .02$, and that ratings were lower in EM than in RO at post-test, $t(61) = 3.76$, $p < .001$, $d = .95$. ANOVA on emotionality ratings

showed a main effect of Time, $F(1, 61) = 17.49$, $p < .001$, $\eta\rho^2 = .22$, but not of Condition, $F(1, 61) = 1.28$, $p = .26$, and an interaction effect, $F(1, 61) = 10.54$, $p = .002$, $\eta\rho^2 = .15$. The planned comparison revealed that ratings decreased over time in EM, $t(31) = 4.79$, $p < .001$, $d = .82$. Post hoc comparisons indicated that ratings did not change over time in RO, $t < 1$, and that ratings did not differ between the conditions at post-test, $t(61) = 2.28$, $p = .03$.

In sum, EM, but not RO, resulted in US devaluation as evidenced by reductions in both memory vividness and emotionality, which confirmed the first hypothesis.

Acquisition of conditioned responding

Univariate analyses on fear ratings, pleasantness ratings, and SCRs, comparing CSs and conditions during the habituation phase, showed that there were no a-priori differences, Fs < 1. Therefore, any differences at the end of the acquisition phase were attributed to the conditioning procedure. MANOVA showed that, at the end of the acquisition phase, the combined DVs (i.e., fear ratings and US expectancy ratings) were significantly affected by CS Type, $F(2, 60) = 210.25$, $p < .001$, $\eta\rho^2 = .88$, but not by Condition or their interaction, Fs < 1. Univariate analyses on fear ratings revealed a main effect of CS Type, $F(1, 61) = 116.09$, $p < .001$, $\eta\rho^2 = .66$, with higher fear for the CS+ than for the CS-, other Fs < 1. Post hoc comparisons indicated that from habituation to acquisition fear for the CS+ increased, $t(62) = 9.18$, $p < .001$, $d = 2.33$, but fear for the CS- did not change, $t(62) = 1.50$, $p = .14$. Similarly, US expectancies to CS+ were higher than to CS-, $F(1, 61) = 408.29$, $p < .001$, $\eta\rho^2 = .87$, other Fs < 1. ANOVA on SCRs, however, did not yield a main effect of CS Type, $F(1, 61) = 2.75$, $p = .10$, or any other effects, $F < 1$. ANOVA on CS pleasantness ratings indicated that, as a result of the conditioning procedure, participants rated CS+ as less pleasant than CS-, $F(1, 61) = 61.25$, $p < .001$, $\eta\rho^2 = .50$, other Fs < 1. Post hoc comparisons indicated that in the course of acquisition the CS+ became less pleasant, $t(62) = 7.82$, $p < .001$, $d = 1.99$, and the CS- became more pleasant, $t(62) = 4.36$, $p < .001$, $d = 1.11$.

In sum, acquisition of conditioned fear was successful as evidenced by self-reported fear ratings and US expectancy ratings, but not SCR. Furthermore, acquisition of evaluative responding was successful.

Intervention effects on conditioned responding

Conditioned fear. MANOVA showed effects of CS Type, $F(2, 60) = 144.30$, $p < .001$, $\eta\rho^2 = .83$, Time, $F(2, 60) = 5.12$, $p = .01$, $\eta\rho^2 = .15$, Condition, $F(2, 60) = 5.08$, $p = .01$, $\eta\rho^2 = .15$, CS Type x Time, $F(2, 60) = 28.54$, $p < .001$, $\eta\rho^2 = .49$, and Time x Condition, $F(2, 60) = 5.15$, $p = .01$, $\eta\rho^2 = .15$, but not of CS Type x Condition, $F < 1$. The three-way interaction was marginally significant, $F(2, 60) = 3.02$, $p = .056$, $\eta\rho^2 = .09$. Univariate analyses on fear ratings

showed effects of CS Type, $F(1, 61) = 132.33$, $p < .001$, $\eta\rho^2 = .68$, Time x Condition, $F(1, 61) = 9.81$, $p = .003$, $\eta\rho^2 = .14$, and CS Type x Time, $F(1, 61) = 6.12$, $p = .02$, $\eta\rho^2 = .09$, but not of Time, $F(1, 61) = 3.35$, $p = .07$, and CS Type x Condition, $F < 1$. The three-way interaction was significant, $F(1, 61) = 4.23$, $p = .04$, $\eta\rho^2 = .07$. To understand this interaction, CS Type x Time ANOVAs were performed for each condition. In RO, there were main effects of CS Type, $F(1, 30) = 72.22$, $p < .001$, $\eta\rho^2 = .71$, and Time, $F(1, 30) = 11.09$, $p = .002$, $\eta\rho^2 = .27$, but no interaction effect, $F < 1$, meaning that this condition did not affect conditioned fear. In EM, there was a main effect of CS Type, $F(1, 31) = 59.86$, $p < .001$, $\eta\rho^2 = .66$, but not for Time, $F < 1$. The interaction was also significant, $F(1, 31) = 9.59$, $p = .004$, $\eta\rho^2 = .24$. The planned comparison showed that, in line with the hypothesis, fear for the CS+ decreased as a result of the intervention, $t(31) = 2.33$, $p = .03$, $d = .84$. Post hoc comparisons further indicated that fear for the CS- did not change over time, $t(31) = 1.80$, $p = .08$, and that at test, fear for the CS+ was still higher than fear for the CS-, $t(31) = 5.66$, $p < .001$, $d = 2.03$ (see Figure 2).

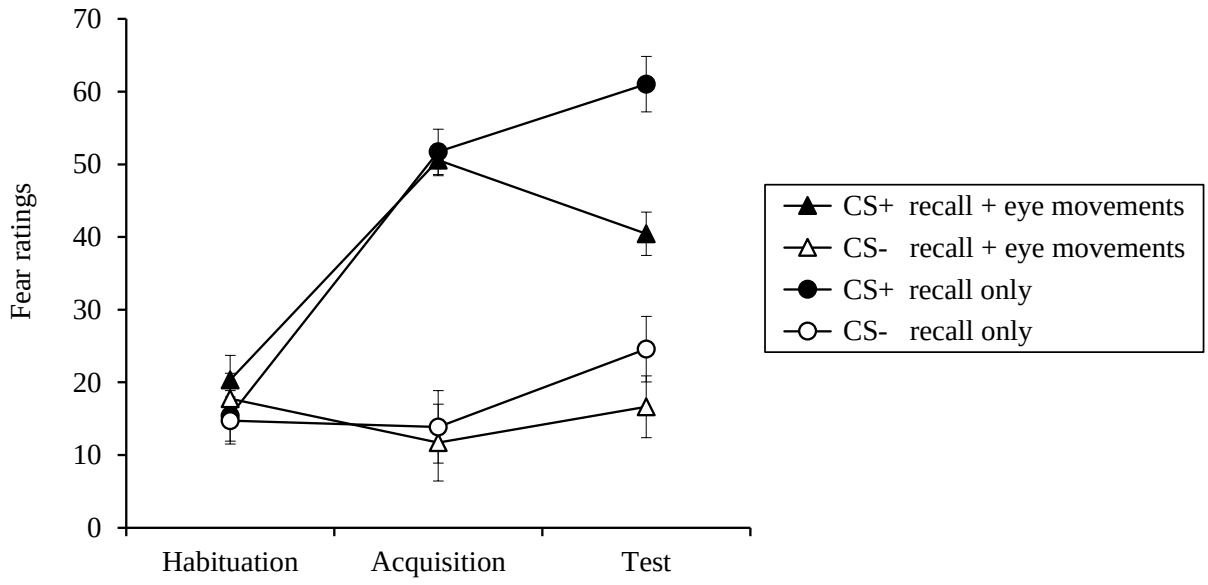

Fig. 2 Mean fear ratings for the habituation, acquisition and test phase. Error bars indicate SEM.

ANOVA on US expectancy ratings also showed effects of CS Type, $F(1, 61) = 282.99$, $p < .001$, $\eta\rho^2 = .82$, Time x Condition, $F(1, 61) = 5.42$, $p = .02$, $\eta\rho^2 = .08$, and CS Type x Time, $F(1, 61) = 57.77$, $p < .001$, $\eta\rho^2 = .49$, but not of Time, $F(1, 61) = 1.74$, $p = .19$, and CS Type x Condition, $F < 1$. The three-way interaction was significant, $F(1, 61) = 4.29$, $p = .04$, $\eta\rho^2 = .07$. To break down this interaction, CS Type x Time ANOVAs for each condition were conducted, that revealed a significant interaction in RO, $F(1, 30) = 13.25$, $p = .001$, $\eta\rho^2 = .31$, and EM, $F(1,$

31) = 54.72, $p < .001$, $\eta\rho^2 = .64$. We therefore tested whether, as suggested by Figure 3, the larger interaction effect in EM was primarily caused by differences in the CS+ rating. Separate Time x Condition ANOVAs for CS+ and CS- confirmed that change over time in CS- ratings did not differ between conditions, $F < 1$, and that change over time in CS+ ratings was greater in EM than in RO, $F(1, 62) = 16.40$, $p < .001$, $\eta\rho^2 = .21$. The planned comparison showed that CS+ ratings dropped in EM, $t(31) = 7.38$, $p < .001$, $d = 2.65$, which confirmed the hypothesis. Post hoc comparison indicated that US expectancy to the CS+ also dropped in RO, $t(30) = 4.64$, $p < .001$, $d = 1.70$, and that at test CS+ ratings were lower in EM than in RO, $t(61) = 3.59$, $p < .001$, $d = .92$ (see Figure 3).

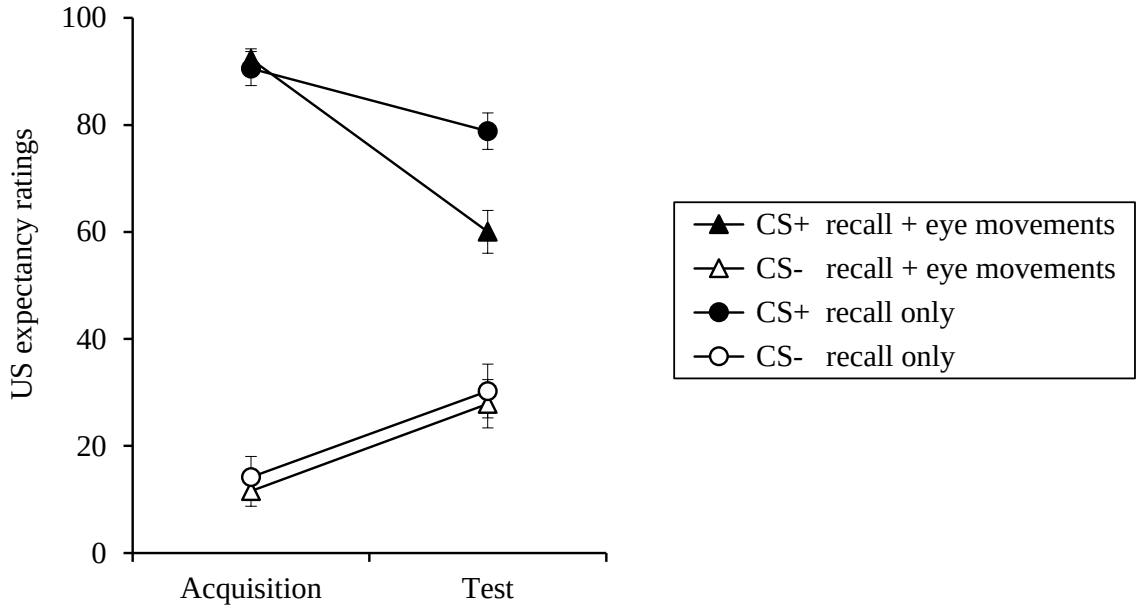

Fig. 3 Mean US expectancy ratings for the acquisition and test phase. Error bars indicate SEM.

Conditioned CS evaluation. ANOVA showed a main effect of CS Type, $F(1, 61) = 72.89$, $p < .001$, $\eta\rho^2 = .54$, but no other main effects, Fs < 1. There was no CS Type x Condition interaction, $F < 1$, or Time x Condition interaction, $F(1, 61) = 1.79$, $p = .19$, but there was a significant CS Type x Time interaction, $F(1, 61) = 5.55$, $p = .02$, $\eta\rho^2 = .08$. The three-way interaction was marginally significant, $F(1, 61) = 2.98$, $p = .09$. The planned comparison revealed that, in EM, CS+ became less unpleasant as a result of the intervention, $t(31) = 2.42$, $p = .02$, $d = .87$. Post hoc comparisons indicated that EM did not affect evaluation of the CS-, $t(31) = 1.95$, $p = .12$, and that in RO, CS evaluations did not change over time, ts < 1 (see Figure 4).

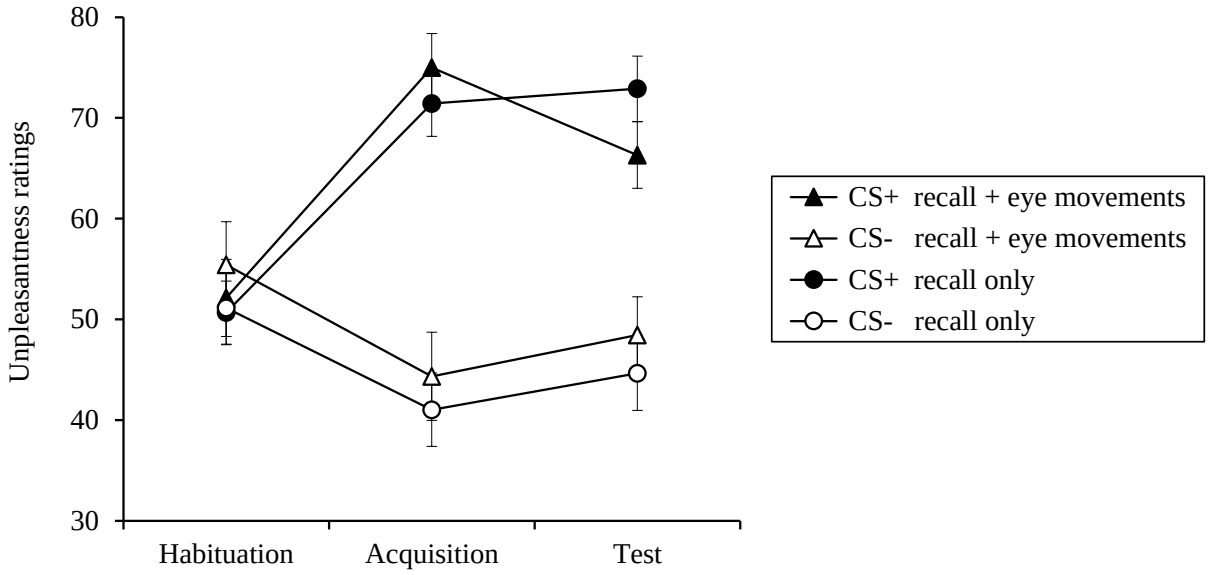

Fig. 4 Mean unpleasantness ratings for the habituation, acquisition and test phase. Error bars indicate SEM.

In sum, EM, but not RO, caused a decrease in conditioned fear as evidenced by self-reported fear ratings and US expectancy ratings, confirming the second hypothesis. Furthermore, and confirming the third hypothesis, EM, but not RO, caused a decrease in the negative evaluation of the CS+.

Discussion

The main findings of this investigation were that EM during recall of aversive memory, relative to recall only, reduced memory vividness and emotionality and caused a decrease in conditioned fear. In addition, the dual task attenuated the negative evaluation of the CS. Although anticipated, the results were not substantiated by skin conductance data.

The first hypothesis that making EM during recall of aversive memory reduces its vividness and emotionality was confirmed. This finding cannot be attributed to imaginal exposure during the intervention, as mere recall of the US did not affect memory emotionality ratings and only slightly reduced memory vividness ratings. A better explanation is provided by working memory theory: taxing working memory during recall disrupts mental imagery and thereby reduces its vividness and emotionality (Gunter & Bodner, 2008).

The dual task was already proven effective in devaluating autobiographical memories (e.g., van den Hout et al., 2001), flash-forwards (e.g., Engelhard & van den Hout, et al., 2010)

and mental images of aversive pictures (Andrade et al., 1997). This research extends earlier findings by showing similar results for mental images of aversive film fragments.

The second and main hypothesis that the dual task causes a decrease in conditioned fear was confirmed as well. Only the dual task condition showed a medium to large decrease in self-reported fear ratings. The mechanism driving this effect does not seem to be inhibition learning or habituation to the US memory, which are the proposed mechanisms of exposure therapy (Craske et al, 2012), since recall only even caused an increase in conditioned responding. Rather, change in the US memory representation seems to have played a causal role. This fits with Davey's (1997) refined model of fear conditioning that explains that after US devaluation the CS activates a less threatening US representation and therefore elicits a weaker CR. Moreover, our findings demonstrate that US devaluation does not require the physical presence of the US, but can be successfully accomplished through mental imagery (cf. Dibbets et al., 2012).

These results suggest that the dual task approach may be a valuable addition to exposure therapy. First, a decrease in the emotionality of aversive memory is likely to reduce resistance and fear associated with (imaginal) exposure. It may therefore be expected that the addition of US devaluation techniques shortens treatment duration and increases treatment adherence. Second, extinction learning is context specific, which explains CR recovery upon CS presentations outside the therapeutic context (e.g., Bouton, 2002). US revaluation, however, has been shown to generalize across contexts (e.g., Hall & Channell, 1985; Hall & Honey, 1989). It therefore seems important that future investigations test the hypothesis that US devaluation decreases the renewal of fear (Dibbets et al., 2012).

Two other remarkable findings need to be discussed. First, the dual task also reduced US expectancy to the CS. How might this be explained? Two recent experiments demonstrated that rehearsal of a CS-US contingency can affect conditioned responding (Joos et al., 2012a; Joos, Vansteenwegen, & Hermans, 2012b). It is possible that during the intervention phase in the current study participants recalled the CS-US contingency, and that blurring of the association caused a decrease in US expectancy. However, participants were explicitly instructed to recall the US, and it seems improbable that recalling the US automatically activated the CS-US association, as the CS and US in the present study shared little features (i.e., were low in belongingness, e.g., Hamm et al., 1989). Alternatively, the dual task may have rendered the US representation less accessible, which in turn reduced US expectancy (cf. the availability heuristic; Kahneman, Slovic, & Tversky, 1982). In support of this explanation, a recent reaction

time experiment showed that EM during memory recall, relative to recall only, decreases memory accessibility (van den Hout, Bartelski et al., 2012).

The reduction of threat expectancy is relevant as exposure therapy outcome is expected to be mediated by inhibitory learning (i.e., learning that a CS means "safety" rather than "danger", Craske et al., 2012). It should be noted that, although the dual task reduced US expectancies, there was still room for improvement. This is relevant as remaining threat expectations may incite avoidance behavior that could maintain such beliefs (Lovibond et al., 2008) and cause a return of fear. This stresses the importance of traditional exposure-based therapy, which primarily focuses on the predictive significance of CSs. Possibly, extinction of threat expectations via extinction learning may be accelerated when preceded by US devaluation. This may be elucidated by future research.

A second noteworthy finding relates to the CS valence. The aversive conditioning procedure resulted in a CS valence shift from neutral to unpleasant, a process referred to as evaluative conditioning (e.g., De Houwer, Thomas, & Baeyens, 2001; Hermans, Vansteenwegen, Crombez, Baeyens, & Eelen, 2002). In line with the third hypothesis, the dual task condition, but not the control condition, produced a medium to large reduction in CS unpleasantness ratings, supporting the notion that US revaluation affects evaluative responding (Baeyens et al., 1992; Walther et al., 2009; but see Baeyens, Vanhouche, Crombez, & Eelen, 1998). This is relevant to clinical practice as negative valence (e.g., dislike or disgust) is related to avoidance behavior (e.g., Krieglmeyer, Deutsch, De Houwer, & De Raedt, 2010) and resistant to extinction (e.g., Engelhard et al., 2012). Note, however, that the present study did not directly compare the dual task condition with an extinction learning condition.

There were several limitations of the present study. First, the results were not substantiated by our SCR data. This is important because psychophysiological measures are considered to be less prone to demand induced effects (Grillon & Baas, 2003). There was no physiological evidence for fear acquisition, which likely resulted from noise in the data. Approximately a third of participants did not show elevated SCR at all. Their data, however, were included in the analyses as instructed by protocol (Pineles et al., 2009). Future investigations may employ more acquisition trials or include the fear potentiated startle response as outcome measure. Demand effects seem, however, unlikely because participants did not have knowledge about EMDR. Moreover, although participants were likely familiar with the beneficial effects of imaginal exposure, there were no reductions in conditioned fear in the control condition. A second limitation was that state mood or emotion was only assessed prior to the experiment (i.e., state anxiety). Therefore, it is unclear, for instance, whether general

arousal diverged over the course of the two conditions. A third limitation is that we tested mostly undergraduate females. It is unclear whether the findings may be generalized to other populations.

In conclusion, this study provides the first evidence that EM during recall of aversive memory reduces conditioned fear. Future directions include replication of the current findings, also with measures that are less prone to demand effects, and testing the effect of the dual task on extinction learning and the return of fear after extinction.

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Chapter 4

Dual-tasking attenuates the return of fear after extinction

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Abstract

Return of fear following exposure treatment may be explained by ABA-renewal: fear acquired in context A, and extinguished in context B, may return in context A. Conditioning theory predicts that intensity of conditioned fear is mediated by the mental representation of the unconditioned stimulus (US) evoked by the conditioned stimulus (CS). This study tested whether US-devaluation via a dual-task – imagining the US while making eye movements – attenuates fear renewal. Participants acquired fear in context A, and underwent extinction in context A or B. Next, two groups did a filler task (AAA; ABA), one a dual-task of US imagination with eye movements (ABA-DT), and one merely imagined the US (ABA-RO). Finally, participants were re-presented the CSs in context A. ABA-renewal was found for US-expectancy. Dual-tasking, but not recall only, reduced fear renewal. No between-group differences were observed in reductions of vividness, emotionality, and startle responses to the US. Findings suggest that dual-tasking may attenuate fear renewal.

Introduction

Exposure-based treatments are effective for anxiety disorders (Bisson et al., 2007; Chambless & Ollendick, 2001; Deacon & Abramowitz, 2004; Hofmann & Smits, 2008), and involve exposure to the feared stimulus, which is the clinical analogue of a laboratory extinction procedure. This technique is based on the Pavlovian fear conditioning model, a widely accepted theory to explain the acquisition and extinction of fear (e.g., Deacon & Abramowitz, 2004; Eysenck & Martin, 1987; Mineka & Oehlberg, 2008). In a typical fear conditioning experiment, a person learns to expect that a conditioned stimulus (CS) is followed by an aversive unconditioned stimulus (US), which causes the CS to elicit a conditioned response (CR; e.g., fear). During an extinction procedure, the CS is repeatedly presented in the absence of the US and, as a result, the CR gradually diminishes. However, conditioned fear may (partly) return after it has weakened or extinguished (e.g., Hermans, Craske, Mineka, & Lovibond, 2006). Clinical data demonstrate considerable relapse rates after exposure treatment (e.g., social phobia: 13%; Fava, Grandi et al., 2001; panic disorder: 23%; Fava, Rafanelli et al., 2001; obsessive-compulsive disorder: 11%; Simpson et al., 2004). Therefore, strategies that challenge the return of fear are needed to boost treatment success.

Phenomena that give rise to the return of fear have been identified (for reviews, see Bouton, 2002, 2004; Hermans et al., 2006). Fear may return as time passes (spontaneous recovery) or in response to presentations of the US only (reinstatement). Additionally, a context switch after extinction can result in return of fear, which is known as *renewal of fear*. Typically, in these experiments, fear conditioning takes place in one context ("Context A"; e.g., lit room) and extinction learning takes place in another context ("Context B"; e.g., dimmed room). When CSs are presented again in context A, fear returns (ABA-renewal; Bouton & King, 1983). This phenomenon has proven to be robust in (sub)clinical samples (e.g., Mineka, Mystkowski, Hladek, & Rodriguez, 1999; Mystkowski, Craske, & Echiverri, 2002) and student samples (Effting & Kindt, 2007; Neumann & Kitlertsirivatana, 2010; Vansteenwegen et al., 2005). Strategies countering fear renewal include extinction in multiple contexts (e.g., Balooch & Neumann, 2011; Vansteenwegen et al., 2007; but see Bouton, García-Gutiérrez, Zilski, & Moody, 2006) and using retrieval cues for extinction (e.g., Dibbets & Maes, 2011; Vansteenwegen et al., 2006). These methods capitalize on changing the predictive quality of the CS (i.e., reducing US-expectancy), which may have a significant drawback: the original fear memory may be retrieved under particular circumstances.

Researchers have hypothesized that during extinction the original fear memory (CS-US) is not destroyed or unlearned (Bouton, 2002; Delamater, 2004; Hermans et al., 2006). Instead, as supported by animal research (Delamater, 1996; Rescorla, 1996), a new association (CS–no US) is formed that renders the old CS-US association less salient. Expression of this new association depends directly on the context in which the CS is encountered (as evidenced by the renewal phenomenon). Thus, after an extinction procedure, confrontation with the CS outside the extinction context seems to reactivate the original fear memory, which incites renewal of fear. Context-dependency of conditioned responding would not be a problem if pathological fear would involve only a single context. However, multiple contexts are likely to play a role in fear conditioning (e.g., Craske et al., 2008). Practically, this implies that all significant contexts and CSs should be considered in treatment to successfully prevent relapse, which may not be feasible.

An alternative method to reduce fear renewal may be to devaluate the cognitive representation of the US, such that it becomes less aversive. Davey's (1997) classical conditioning model stresses that CR strength is not just a function of the strength of the learned CS-US association, but also of the mental US representation. Thus, revaluation of the cognitive representation of the US may occur independently of any changes in the CS-US association, and such US revaluation will mediate change in CR strength. For example, mere exposure to the actual US may weaken the CR (i.e., habituation; Davey & McKenna, 1983), and (ruminative) cognitive rehearsal of the US may strengthen the CR (Jones & Davey, 1990). Since changes in the US representation are less likely to be context-dependent, it is both theoretically and clinically important to examine whether fear renewal can be reduced by US-devaluation.

Previous studies provide tentative support for this hypothesis. For example, in rats, habituation to the US reduces fear renewal (Rauhut, Thomas, & Ayres, 2001). In humans, US-devaluation via imagery rescripting (IR; an effective treatment for various anxiety disorders; Holmes & Mathews, 2010) has been used to attempt to reduce the renewal effect (Dibbets, Poort, & Arntz, 2012). During IR, a mental image of the aversive US is verbally rescripted into a more acceptable image. Dibbets and colleagues hypothesized that IR of the US reduces fear renewal. They administered a fear conditioning paradigm to undergraduates. Fear acquisition took place in context A, in which a CS+ (picture of a car), but not a CS- (picture of a motorcycle) was followed by a US (picture of a mutilated dead child). During extinction, only the CS+ and CS- were presented. Four extinction conditions were used: (1) for an AAA group, extinction took place in context A, (2) for an ABA group, extinction took place in context B, (3) for an ABA-IR group, extinction was in context B and was accompanied by IR, and (4) for an ABA-

IUn group, extinction was in context B and was accompanied by imagery unrelated to the US. Finally, a testing phase involved CS presentations in context A. The main findings were that the ABA-IR group showed both US-devaluation and less renewal of US-expectancy ratings, suggesting that the US representation had changed. Although these results were promising, there might have been a confounding factor. In the ABA-IR group, significantly less extinction took place than in the other groups, presumably because participants rehearsed the CS-US association as a part of IR *during* the extinction trials. Therefore, any conclusions on the renewal of fear may be premature.

The current study will focus on another potential US-devaluation technique, which involves a dual-task approach. In this type of task, a person keeps the US image in mind while, at the same time, attending to an external stimulus. In clinical and laboratory studies such a dual-task typically involves induction of horizontal eye movement (EM; e.g., by visually tracking a circle that moves from side to side across a computer screen). Analogue laboratory studies have shown that EM during recall of an emotional image render the image less vivid and/or emotional during future recollections, compared to recall alone (e.g., Engelhard, van den Hout, Janssen, & van der Beek, 2010; Engelhard, van den Hout, & Smeets, 2011; Gunter & Bodner, 2008; Maxfield, Melnyk, & Hayman, 2008; van den Hout et al., 2011). These findings agree with clinical data showing that Eye Movement Desensitization and Reprocessing (EMDR) therapy is effective for treating posttraumatic stress disorder (PTSD, e.g., Bisson et al., 2007), and that the EM component adds to its beneficial effects (Lee & Cuijpers, 2013). The effectiveness of EM can be explained by a working memory (WM) theory: as WM is limited, any taxing task during mental imagery competes for storage capacity or rehearsal processes and, therefore, disrupts mental imagery. As a result the vividness and emotionality of the mental representation decrease (e.g., Gunter & Bodner, 2008).

There is experimental support for the effectiveness of other competing tasks (e.g., drawing a complex figure [Gunter & Bodner, 2008], or counting backwards [Engelhard et al., 2011]), but most studies have used EM and repeatedly provided evidence for its effectiveness. Therefore, based on its reliability, we used EM as the competing task in the current study. Importantly, the beneficial effects of EM have, by and large, been demonstrated for *visual* memories (i.e., autobiographic material, e.g., Gunter & Bodner, 2008; or images provided by the investigator, e.g. Andrade, Kavanagh, & Baddeley, 1997). Hence, we decided to investigate fear conditioning with an aversive picture as US (cf. Dibbets et al., 2012) instead of a more traditional electrocutaneous or loud auditory stimulus (e.g. Lipp, 2006). Usually, picture-picture conditioning paradigms are used to investigate evaluative conditioning – the acquisition of likes

and dislikes (De Houwer, Thomas, & Baeyens, 2001). However, several studies have demonstrated differential skin conductance responses (SCRs) – a physiological signature of fear – in picture-picture paradigms with aversive pictures as US (e.g., Dawson, Rissling, Schell & Wilcox, 2007; Klucken et al. 2009).

The main objective of the present study was to examine whether recall of a US while making EM leads to less ABA-renewal. To test the hypothesis, four conditions were used. Two conditions were included to demonstrate fear renewal (AAA vs. ABA). In the third condition the dual-task was used between extinction and a test phase (ABA-DT). The fourth condition was added to explore the effect of imaginary exposure, in which the dual-task was replaced by mere imagery (recall only: ABA-RO). US devaluation was assessed by memory vividness and emotionality ratings. In addition, we measured potentiated startle during imagery as startle augmentation has been observed during imagery of unpleasant scripts (e.g., Miller, Patrick, & Levenston, 2002) and startle diminution has been observed after dual-tasking (Engelhard, van Uijen, & van den Hout, 2010).

Method

Participants

A total of 109 Utrecht University students enrolled in this study. A priori, we decided to include 20 in each condition, with the requisite that each participant acquired the CS-US contingency and showed extinction (see *Data reduction*). Further exclusion criteria were: knowledge of EMDR and prior participation in research on EMDR. The final sample involved 80 participants (61 women; *M* age = 21.97 years, *SD* = 2.93).

Stimuli

CSs were pictures of a black triangle and a black square with a width and height of approximately 7.5 cm. CSs were presented against a brightly colored background (yellow or cyan) that served as context and was manipulated as a function of experimental phase and condition. The US was a picture of a mutilated dead child (IAPS picture 3051; cf. Dibbets et al., 2012) that covered 75% of the screen, which allowed participants to perceive the contextual background color. Dibbets and colleagues (2012) showed that this picture serves a useful US: Self-Assessment-Manikin ratings indicated that participants evaluated this picture as highly unpleasant. Moreover, the US-picture received significantly larger SCR than did both CS-pictures at first presentation. The experimental paradigm was presented with E-Prime 2.0 (Psychology Software Tools).

Outcome measures

US-expectancy. US-expectancy was assessed with a question ('to what degree do you expect the aversive picture of the child now') rated on a Visual Analogue Scale (VAS) on the screen that ranged from 0 (=*not at all*) to 100 (=*definitely*). Anticipation of the US was also measured with SCR that was registered with a Coulbourn Modular Instruments System (Allentown, PA, USA) via a Coulbourn Isolated Skin Conductance coupler (S71-23). A constant current of .5 V was transmitted via 9-mm Sensor Medics Ag/AgCl electrodes that were attached to the medial phalanges of the middle and index finger (cf. Fowles et al., 1981).

Cognitive US representation. Quality of the cognitive US representation was examined with three VASs for emotional intensity (0=not at all unpleasant, 100=extremely unpleasant), vividness (0=*not at all vivid*, 100=*extremely vivid*), and difficulty recalling the image (0=*not at all difficult*, 100=*extremely difficult*), and with startle responses. Loud tones (50 ms, 95 dB) with instant rise and fall time served as startle probes and were presented through headphones. Electromyographic (EMG) activity was recorded with two 4-mm Sensor Medics Ag/AgCl electrodes placed over the orbicularis oculi region of the right eye. A 9-mm ground electrode was placed in the middle of the forehead. The raw EMG signal, sampled at 1,000 Hz, was amplified (10 K) and filtered (13 Hz high-pass; 150 Hz low-pass) by a Coulbourn V75-04 Isolated Bioamplifier with Bandpass Filter (Blumenthal et al., 2005; cf. Engelhard, van Uijen et al., 2010).

Questionnaires. Neuroticism was assessed with the 22 neuroticism items of the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975; Sanderman, Arrindell, Ranchor, Eysenck, & Eysenck, 1995) using the binary response format (0=*no*, 1=*yes*). Items from the extraversion scale were administered as well, and were mixed with the neuroticism items, to minimize order-related artifacts (e.g., adopting a negative response tendency).

State and trait anxiety were assessed with the State Trait Anxiety Inventory (STAI-DY; Spielberger, Gorsuch, & Lushene, 1970; Van der Ploeg, Defares, & Spielberger, 1980) that consists of 20 items measuring state anxiety (α = .86 in current study) and 20 measuring trait anxiety (α = .92 in current study) scored on a 1-4-point scale (1=*not at all*, 4=*severely*).

These questionnaires were included to control for differences across the conditions in levels of neuroticism and anxiety, as both constructs have been identified to affect fear conditioning (e.g., Grillon et al., 2006; Lissek et al., 2005).

Procedure

Participants were seated in a dim, soundproof room, approximately 60 cm in front of a computer screen. First, they received oral and written information. After signing informed consent, they filled out the questionnaires. Next, the skin was cleaned, electrodes were placed, and headphones were given. Each participant was randomly allocated to one of the four conditions: AAA, ABA, ABA-DT (dual-task), or ABA-RO (recall only).

Acquisition phase. Before acquisition, participants practiced rating the VAS, and were told to indicate their expectancy of the US by a mouse click on the scale, each time they saw a CS picture. All conditions underwent similar acquisition procedures in context A (yellow background), using 4 CS+ and 4 CS- trials. Each CS lasted for 6 s, and only the CS+ was directly followed by the 2-s US. For the CS-, the intertrial interval (ITI) was extended by 2 s. Order of CSs was pseudo randomized with a maximum of 2 similar CSs in succession. The 14-s CS-CS interval comprised a blank yellow screen. SCR was continuously recorded. The US-expectancy s presented on the bottom of the screen during each CS. The instructions were to indicate during each CS-presentation, by means of a mouse-click on the VAS, to what degree the aversive picture was expected to follow the CS.

After the acquisition phase, participants were told: "*Form an image of the mutilated child that you just saw a few times. Keep your eyes open and bring the image to mind as vividly as possible. Press the space bar when the image is vivid.*" Then, 11 s after the space bar was pressed, participants were asked to rate vividness, emotionality, and difficulty recalling the image, followed by the first startle measurement. Participants received 9 startle probes (mean interstimulus interval = 3 s; range 2–4 s) to facilitate habituation. Next, instructions were to recall the image again and to ignore the probes as much as possible. After 500-2500 ms, 3 startle probes (mean ITI 3.5 s; range 2.5–4.5 s) were presented to elicit startle reflexes. This recall period (11 s) was repeated once, resulting in a total of 6 startle measurements.

Extinction phase. The extinction phase involved context A (AAA: yellow background) or B (other groups: cyan background). Initially, all groups underwent a similar extinction phase, in which each CS was presented 6 times, without the US. Again, both US-expectancy and SCR were assessed. The instructions were to indicate to what degree the aversive picture was expected, and that *if* changes had occurred in the CS-US relationship, participants should translate this in their US-expectancy ratings. Subsequent to the extinction phase, all groups were instructed to recall the image of the US for 11 s, and to rate image vividness and emotionality for the second time.

In the ABA-DT and ABA-RO groups, the distance between the participant and computer screen was reduced to 30 cm. In ABA-DT, a black circle moved from left to right and back across the screen (at 1 cycle per s) in context B (cyan background). In ABA-RO, a stationary black circle was shown in the middle of the screen. Participants in these conditions were told to think of the image and watch the circle without moving their head at the same time. Both conditions comprised 6 sets of 24 s, with 10 s breaks in between. The duration of these sets and breaks were based on the protocol used by others (e.g., Engelhard, van den Hout, Dek et al., 2011; Gunter & Bodner, 2008; van den Hout, Muris, Salemink, & Kindt, 2001). During this time, the other groups (AAA and ABA) did a filler task, in which 6 guitar pictures were shown twice. Participants were asked to indicate whether they saw each picture for the first or second time. To keep the duration of exposure to the contextual background identical over all groups, the filler task was displayed against the extinction background as well. Finally, all groups were instructed to recall the US image again for 11 s, and to rate image vividness and emotionality for the third time.

Test phase. All groups received a similar test phase in context A (yellow background). Both CSs were presented twice, with the restriction that the CS+ and CS- occurred in the first two trials, and the order was counterbalanced across conditions (cf. Dibbets et al., 2012). The procedure was comparable to the extinction phase. The instructions read: Again, please indicate to what degree you expect the aversive picture. Directly after the test phase, startle responses were measured again during US-imagination, which was identical to the first startle measurement after the acquisition phase.

Data preparation

Data reduction. Since this study used only two CSs and a 100% contingency between the CS+ and US in the acquisition phase, participants unaware of the CS-US contingency were considered to be inattentive or having insufficient understanding of the experimental procedure (cf. Dibbets et al., 2012). Moreover, drawing conclusions on the renewal of fear is only appropriate when both fear acquisition and fear extinction are verified. Therefore, participants were only included in data analyses when their US-expectancy score at the final acquisition trial was at least 70 for the CS+ and less than 30 for the CS-. At the final extinction trial, US-expectancy scores for both CSs needed to be less than 30. Based on these criteria, 29 participants were excluded from analyses, resulting in a final sample of 80 participants. Due to 1 missing value in the ABA group, renewal of US-expectancy was analyzed using 79 participants.

Skin conductance response. Responses were calculated by subtracting the mean SC level for the 2 s preceding CS onset from the largest value recorded during the full 6-s CS interval (cf. Pineles, Orr, & Orr, 2009). The minimum response amplitude was set at .05 µS (Dawson, Schell, & Filion, 2000). All other responses were scored as 0 and left in the analyses. After applying a square root transformation to all SCR data, no outliers were found. Finally, an individual range correction (SCR/SCR_{max}) was used to minimize interindividual variance (Lykken & Venables, 1971).

Startle responses. Response amplitudes were computed as the difference between the maximum EMG value within 20 to 150 ms after stimulus onset and the average EMG value during baseline (-40 to +10 ms around stimulus onset). Response onset latency was set at 21–80 ms (Blumenthal et al., 2005). All obtained amplitudes were transformed by square root. Next, an individual range correction was applied by standardizing each blink amplitude using all scores for a given subjects as the reference distribution (Blumenthal et al., 2005; cf. Grillon & Baas, 2003). To this end, *T*-scores were calculated (50 + 10*[(raw score – *M*) / *SD*]). For each group separately, outliers were replaced by $M \pm 2.5\ SD$. Finally, following the Blumenthal et al. (2005) recommendation, mean response *magnitudes* for the 6 trials per block were calculated by the product of the mean amplitude of all detected responses and response probability (i.e., ratio detected responses / elicited responses).

Results

In the following analyses, when the sphericity assumption was violated, Greenhouse-Geisser or Huynh-Feldt corrections were applied. In post-hoc *t*-tests Bonferroni corrections were applied.

Table 1. *Mean (SD) and ratio for demographic information and questionnaire data*

Group	Age	F/M	Neuroticism	Trait anxiety	State anxiety
AAA	22.45 (3.50)	15/5	8.37 (5.96)	36.90 (11.15)	32.95 (7.06)
ABA	21.75 (2.71)	15/5	6.35 (4.07)	34.05 (7.18)	32.30 (6.01)
ABA-DT	21.45 (2.72)	17/3	6.60 (5.02)	37.65 (10.21)	35.05 (6.11)
ABA-RO	22.25 (2.85)	14/6	10.00 (4.57)	39.60 (6.75)	37.05 (8.45)

Table 1 shows the data for background variables. No differences were found between the four experimental conditions in age ($F < 1$), neuroticism scores, $F(3, 75) = 2.38$, $p = .08$, state anxiety, $F(3, 76) = 1.91$, $p = .14$, or trait anxiety, $F(3, 75) = 1.26$, $p = .30$. Furthermore, gender ratio was comparable over conditions, $\chi^2(3) = 1.31$, $p = .73$.

US-expectancy

Self-report. First, US expectancies during CS+ and CS- were examined separately for the acquisition and extinction phases. Then, renewal of US-expectancy was studied in the test phase. Figure 1 illustrates differential responding over time, for each condition.

For the acquisition phase, a 2 (CS type: CS+ vs. CS-) x 4 (Trial) x 4 (Condition: AAA vs. ABA vs. ABA-DT vs. ABA-RO) ANOVA showed main effects for CS type, $F(1, 76) = 744.02$, $p < .001$, and Trial, $F(2.39, 181.88) = 15.39$, $p < .001$, with US expectancies for the CS+ being overall higher than for the CS-, and increasing over time. There was no main effect for condition, $F < 1$. There was a significant CS type x Trial interaction, $F(2.25, 170.80) = 245.38$, $p < .001$, but the CS type x Trial x Condition interaction was not significant, $F < 1$. Post hoc paired *t*-tests comparing US expectancies at the first and last acquisitions trials, showed that over time US-expectancy increased for CS+, $t(79) = 12.48$, $p < .001$, and decreased for CS-, $t(79) = 20.02$, $p < .001$, indicating differential learning over time. Crucially, US expectancies were higher during the last CS+ trial compared to during the last CS- trial, $F(1,76) = 4379.39$, $p < .001$, which did not differ across conditions, $F < 1$.

The extinction phase demonstrated main effects for CS type, $F(1, 76) = 5.96$, $p < .05$, and Trial, $F(2.62, 199.26) = 212.51$, $p < .001$, with US expectancies being overall higher for CS+ than CS-, and decreasing over time. There was no main effect for condition, $F(3, 76) = 1.84$, $p = .15$. The CS type x Trial interaction was significant, $F(2.35, 178.25) = 5.65$, $p < .01$, which can be explained by higher US expectancies during CS+ than CS- at the first extinction trial, $t(79) = 3.01$, $p < .01$. Furthermore, the three-way interaction was not significant, $F(7.04, 178.25) = 1.35$, $p = .23$. Crucially, US expectancies were similar during the last CS+ trial compared to the last CS- trial, $F < 1$, which did not differ across the conditions, $F(3, 76) = 1.67$, $p = .18$. Therefore, differences between the conditions in the renewal of US-expectancy can be attributed to the intervention that followed the extinction phase.

Renewal of US-expectancy was examined by comparing ratings at the last extinction trial to the first test trial in a CS type x Trial x Condition ANOVA. All main effects and two-way interaction effects were significant at $p < .05$, except for the main effect of Condition, $F(3, 75) = 1.80$, $p = .15$. Importantly, the three-way interaction was significant, $F(3, 75) = 5.94$, $p < .01$, indicating that differential responding over time differed between conditions.

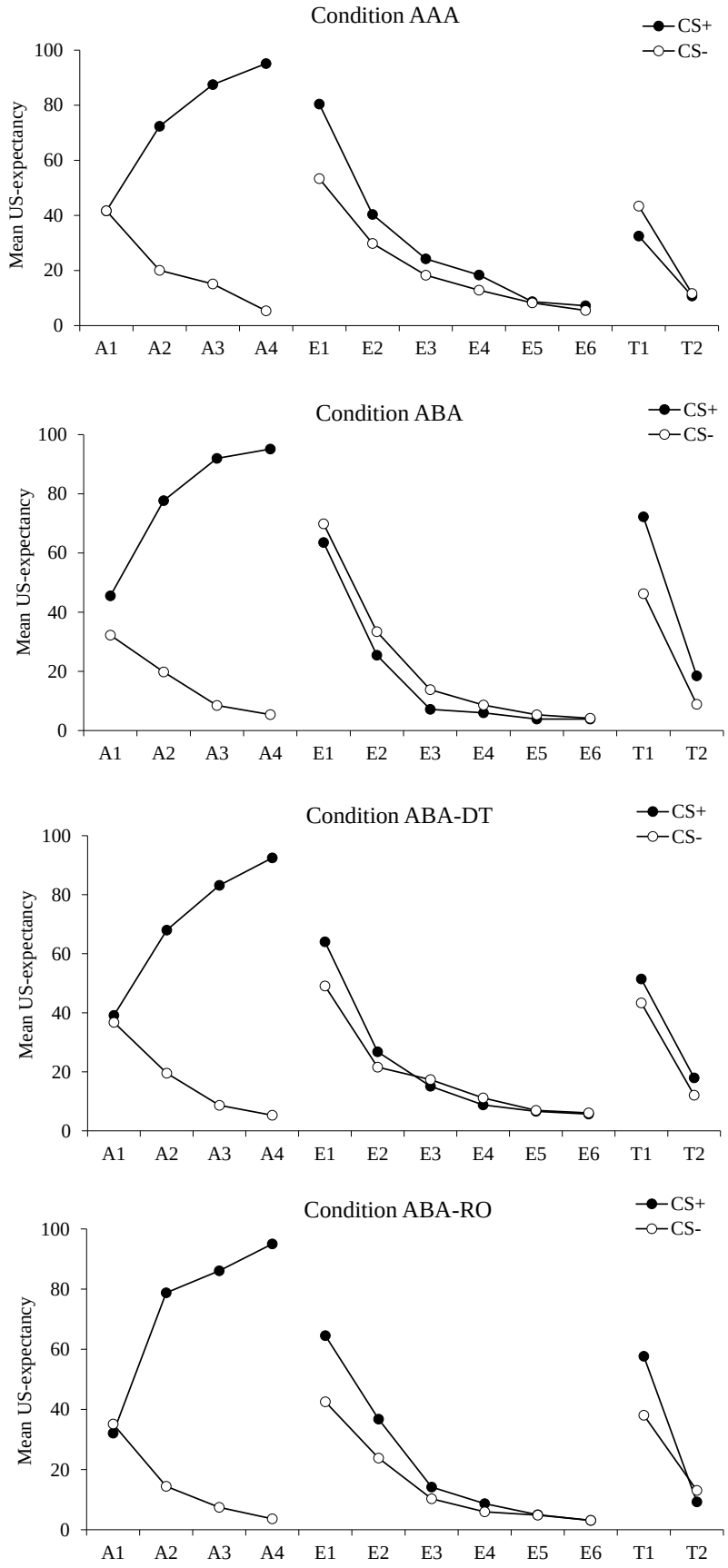

Fig. 1 Mean US-expectancy for CS+ and CS- during acquisition (trials A1-A4), extinction (trials E1-E6), and test (trial T1 and T2).

To test the hypotheses, separate CS type x Trial x Condition ANOVAs were performed that directly compared the conditions of interest. First, an ANOVA that compared conditions AAA and ABA revealed a significant three-way interaction, $F(1, 37) = 14.70$, $p < .001$. To break down this three-way interaction, separate CS type x Trial ANOVAs were performed. For AAA, the interaction effect was not significant, $F(1, 19) = 3.42$, $p = .08$, meaning that change in US-expectancy over time was similar for CS- and CS+. For ABA, there was a significant interaction effect, $F(1, 18) = 12.15$, $p < .001$. Post-hoc *t*-tests showed that US-expectancy increased over time for both CS-, $t(19) = 7.78$, $p < .001$, and CS+, $t(18) = 13.77$, $p < .001$. Crucially, this increase was larger for CS+ than for CS-, $t(19) = 3.49$, $p < .01$, serving a proof of principle for ABA renewal.

Next, to test the main hypothesis, conditions ABA and ABA-DT were compared. The ANOVA revealed a significant three-way interaction, $F(1, 37) = 3.33$, $p < .05$, meaning that differential responding over time differed between the conditions. To further understand ABA-DT, a separate CS type x Trial ANOVA was conducted for this condition. This analysis revealed that the interaction effect was not significant, $F(1, 19) = 1.81$, $p = .19$, indicating that, in line with the hypothesis, there was no renewal of US-expectancy in ABA-DT.

Finally, conditions ABA and ABA-RO were compared. The three-way interaction was not significant, $F < 1$, showing that differential responding over time did not differ between these conditions. A separate CS type x Trial ANOVA for ABA-RO yielded a significant interaction effect, $F(1, 19) = 7.28$, $p < .05$. Post-hoc *t*-test showed that US-expectancy increased over time for CS-, $t(19) = 5.74$, $p < .001$, and CS+, $t(19) = 8.14$, $p < .001$, and that this increase was larger for CS+ than for CS-, $t(19) = 2.70$, $p < .05$. These analyses suggest that there was renewal of US-expectancy in both ABA and ABA-RO. However, a direct comparison between conditions ABA-DT and ABA-RO did not yield a three-way interaction, $F(1, 38) = 1.34$, $p = .26$.

In sum, ABA-renewal was found. Extinguished fear returned in condition ABA, but not in condition AAA. In line with the hypothesis, fear renewal was absent in condition ABA-DT. Lastly, there was differential responding over time in ABA-RO, but this condition did not differ significantly from either ABA-DT or ABA.

Skin conductance responses

US aversiveness. To assess whether the employed US elicited a physiological response, indicative of fear, a 3 (Stimulus: CS-, CS+, US) x 4 (Condition) ANOVA was conducted on SCR to the first presentation of each stimulus. This analysis only yielded a significant main effect of Stimulus, $F(1.78, 126.63) = 12.63$, $p < .001$. Post-hoc comparisons showed that SCR

was larger in response to the US (M = .027, SD = .042) than to CS- (M = .008, SD = .022), $t(74) = 4.01$, $p < .001$, and CS+ (M = .010, SD = .023), $t(78) = 3.80$, $p < .001$, but CS+ and CS- did not differ from each other, $t < 1$ (cf. Dibbets et al., 2012; note that the relatively low mean values are the result of transformations).

Differential conditioning. The percentage of participants with elevated SCR (larger than .05 µS) to the CS+ was 17.0% during acquisition, 6.7% during extinction, and 7.7% during the test phase. Due to the minority of participants providing useful data we will report but not interpret the results. A 2 (CS type) x 4 (Condition) ANOVA on average SCR was conducted, for each experimental phase. During acquisition, SCR was higher for CS+ than for CS-, $F(1, 76) = 4.71$, $p < .05$. This difference was absent during extinction, $F < 1$, and did not reach significance during test, $F(1, 76) = 2.10$, $p = .15$. No between-group differences were present in any phase.

Change in the cognitive US representation

No differences were observed between the conditions in difficulty recalling the US-image (M = 23.02, SD = 18.63), $F < 1$. Self-reported vividness and emotional intensity of the recalled US memory were analyzed for the three time points: (1) post-acquisition, (2) pre-intervention (i.e., after extinction, but before the dual-task, recall only, or filler task), and (3) post-intervention. The first measurement served as baseline. In line with expectations, no group differences were present at the baseline or pre-intervention for vividness, $F_{\text{baseline}}(3, 76) = 1.34$, $p = .27$, $F_{\text{pre-intervention}}(3, 76) = 1.27$, $p = .29$, or emotionality: Fs < 1. Notably, substantial reductions between the first two measurements (in the course of extinction) were observed for both vividness and emotionality (see Figure 2).

To examine the effects of each intervention, two separate 2 (Time: pre-intervention vs. post-intervention) x 4 (Condition: AAA vs. ABA vs. ABA-DT vs. ABA-RO) ANOVAs were conducted. For vividness ratings, there was a main effect of time, $F(1, 76) = 7.82$, $p < .01$, with ratings decreasing over time. There was no significant main effect of condition, $F(3, 76) = 1.89$, $p = .14$, and no interaction effect, $F < 1$, meaning that the reductions over time were similar across the four conditions. Similarly, for emotionality ratings, there was a main effect of time, $F(1, 76) = 21.90$, $p < .001$, with ratings decreasing over time, but no significant main effect of condition, $F(3, 76) = 1.52$, $p = .22$, and no interaction effect, $F < 1$. Note, however, that the reductions in the course of intervention are subordinate to the reductions in the course of extinction (Figure 2).

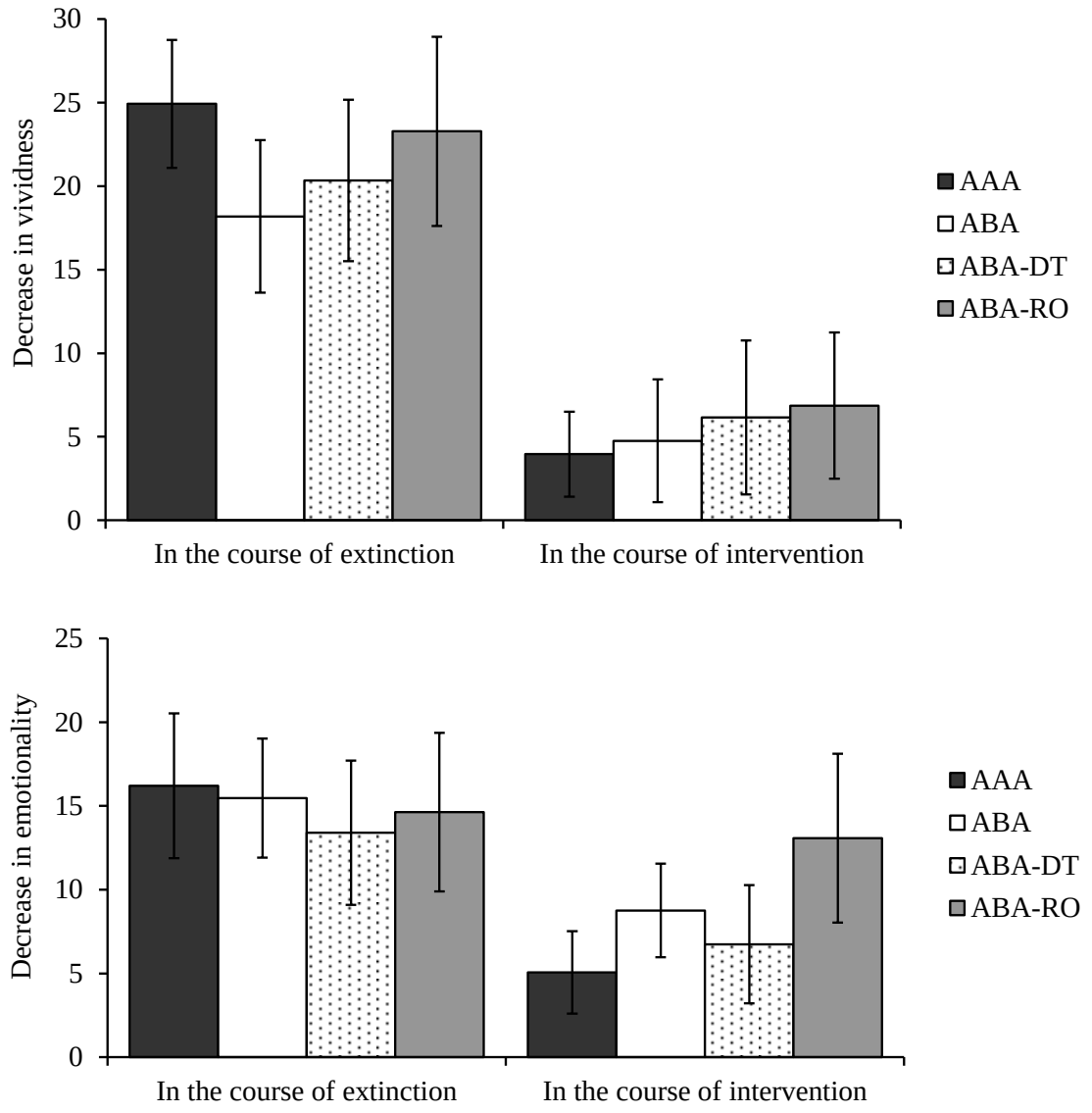

Fig. 2 Mean decrease in vividness and emotionality for each condition. Error bars reflect SEM.

Startle responses were analyzed for the two time points: (1) pre (i.e., after acquisition) and (2) post (i.e., after the test phase). Figure 3 illustrates the startle responses per condition. A 2 (Time: pre vs. post) x 4 (Condition: AAA vs. ABA vs. ABA-DT vs. ABA-RO) ANOVA on blink magnitudes revealed a main effect of time, $F(1, 76) = 13.98$, $p < .001$, with startle responses decreasing over time. There was no significant main effect for condition, $F(3, 76) = 1.49$, $p = .22$, and no interaction effect, $F < 1$. To test the hypothesis that the dual-task reduced startle responses to the recalled US memory, a planned pairwise comparison was carried out between reductions in ABA-DT ($M = 3.87$, $SD = 5.66$) and reductions in AAA and ABA together ($M = 1.86$, $SD = 6.26$). Contrary to the hypothesis, the analyses did not show greater reductions in arousal in ABA-DT compared to the other conditions, $t(58) = 1.20$, $p = .12$.

Thus, the second hypothesis was not confirmed: there was no evidence that the dual-task reduced vividness, emotionality, or physiological responses to the US representation compared to merely recalling the US or fulfilling a filler task.

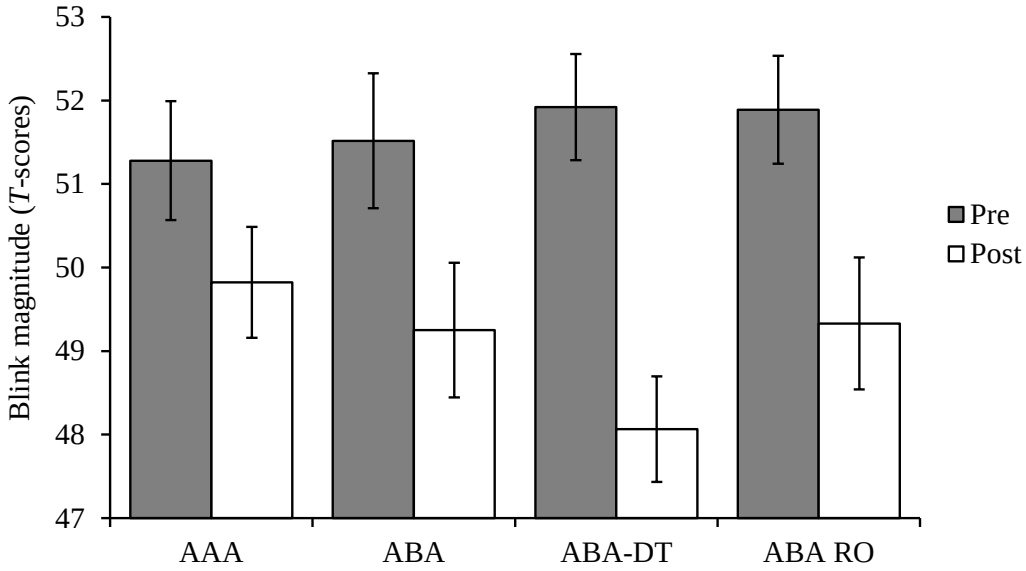

Fig. 3 Mean blink magnitudes at the pre-intervention and post-intervention for each condition. Error bars reflect SEM.

Discussion

This study examined whether EM during recall of memory for an aversive US can reduce fear renewal. As predicted, dual-tasking was shown to attenuate the renewal of US-expectancy, compared to doing a filler task. Mere recall of the US did not attenuate fear renewal, but the renewal ratings in this condition did not significantly differ from those in the dual-task condition. No direct evidence was found, however, that the fear renewal attenuation effect was mediated by US-devaluation: decreases in vividness, emotionality, and startle responses over time did not differ between the conditions.

The results corroborate the findings by Dibbets et al. (2012), who showed a decrease in renewal of US-expectancy following IR during extinction. In the current study, the intervention (i.e., dual-task) was scheduled after extinction, thus ruling out that differences in fear renewal could be explained by differences in extinction. Dual-tasking, like IR, was considered to be a technique that may devaluate the US representation. However, the current study did not provide evidence for US devaluation actually taking place. Decreases in memory vividness, emotionality and startle potentiation were observed, but did not differ between conditions. This is inconsistent with earlier studies that demonstrated that recall with EM relative to mere recall

leads to reductions in vividness and emotionality of images or autobiographical memories (e.g., Andrade et al., 1997; Gunter & Bodner, 2008; van den Hout et al., 2001; Maxfield et al., 2008; van den Hout et al., 2011), as well as reductions in potentiated startle to imagery of aversive memories (e.g., Engelhard, van Uijen et al., 2010). It seems likely that this disparity is due to methodological differences. In earlier studies that reported beneficial effects of the dual task, participants typically recalled distressing autobiographical memories (e.g., Engelhard et al., 2011; Gunter & Bodner, 2008; van den Hout et al., 2011). The present study is the first to our knowledge that integrates the dual task into a fear conditioning paradigm. As the conditioning procedure requires a-priori defined stimuli with fixed duration and exact presentation timing, we employed an aversive picture as the US. It may be assumed that personal memories selected for their unpleasantness are more aversive than the new, short-lived picture that was shown in the current study. Following the first US recall phase, 20 participants (25%) rated the unpleasantness of their memory for the US below 50 (0=*not at all unpleasant*, 100=*extremely unpleasant*), and 18 participants (22.5%) rated the vividness of their memory for the US below 50 (0=*not at all vivid*, 100=*extremely vivid*). Lower scores leave little room for decreases after the intervention. The present results therefore do not doubt the (clinical) efficacy of the dual task but rather the appropriateness of the current design to test its efficacy.

Furthermore, it cannot be ruled out that reductions in memory vividness, emotionality, and startle potentiation were observed in all conditions because all participants underwent periods of US recall (for the assessment of memory vividness and emotionality) and therefore were potentially subject to the effects of US devaluation. However, habituation after such a brief exposure (144 s in the current study) may not be expected (for overview of studies, see Marks, 1987; p. 267–273).

As devaluation of the US representation cannot be brought forward as the mediating mechanism for reduced fear renewal, what can be? A recent study showed that EM during memory retrieval renders the memory less *accessible* (van den Hout, Bartelski, & Engelhard, 2013). Participants that recalled an earlier presented image while making EM both reproduced and recognized less details of the image later on, compared to participants in a recall only condition (experiment 1). This finding was replicated with a reaction time task that assessed memory accessibility (experiment 2). Possibly, dual-tasking in the present study rendered the US representation less accessible. As a result, subsequent encounters with a CS+ may have evoked a much weaker US representation, thereby reducing US-expectancy (cf. the *availability heuristic*; Kahneman, Slovic, & Tversky, 1982).

In sum, the present findings partly confirmed the hypotheses and raise important questions. First, we did not demonstrate US devaluation. The hypothesis that US devaluation reduces fear renewal thus still awaits critical testing. Second, we did observe reduced fear renewal. To test the hypothesis that fear renewal can be affected by memory accessibility, future studies may employ techniques that directly target memory accessibility. Due to these empirical questions we believe that, at present, any clinical implications are premature. If, however, corroborating evidence suggests that changes in the US memory or its accessibility can reduce fear renewal, then it might be possible to optimize traditional exposure-based treatment by adding techniques that directly target the US memory.

Several issues deserve further attention. First, it is unclear how the findings regarding merely recalling the US (ABA-RO) should be interpreted. Renewal scores did not differ from ABA and ABA-RO, which suggests that recall only did not attenuate fear renewal. At the same time, ABA-RO and ABA-DT did not differ, suggesting that dual-tasking did not lead to significantly less renewal than recall only. Possibly, watching a fixed circle (ABA-RO) was not completely effortless, and slightly interfered with US recall. This may have rendered the US memory somewhat less accessible and may explain why renewal scores for ABA-RO were in between ABA and ABA-DT.

Next, on a physiological level, we did not observe fear acquisition, extinction or renewal, as only a minority of participants physiologically responded to the CSs. On the one hand, this lack of responding is not in line with studies that demonstrated differential SCR to aversive pictures (Dawson et al., 2007; Dibbets et al., 2012; Klucken et al., 2009), and may indicate that anticipation of the US was insufficiently arousing. On the other hand, the US actually resulted in a higher SCR than both CSs at the first presentation, suggesting that the US elicited a fear response. This agrees with findings by Dibbets et al. (2012), who used the same US, and also showed larger SCR to the US than to CS+ or CS- at first presentations. Furthermore, analyses on the available SCR data showed differential responding during the acquisition phase. Yet, considering the low response rate, it may be advised to employ a more aversive US in future studies, like a mild electric shock or loud tone (e.g., Ohman & Mineka, 2001; Vansteenwegen et al., 2005).

Finally, Figure 1 shows a substantial increase in expectancy ratings for CS- during the first extinction trial. This may be explained by the instructions right before the extinction phase: participants were told that possible changes may occur in the CS-US relationship, which may have increased US-expectancy (a “better safe-than-sorry strategy”). Nevertheless, at the first extinction trial, expectancy ratings were still higher for CS+ than for CS-, $F(1, 76) = 9.58$, $p <$

.01, which did not differ across conditions, $F(3, 76) = 2.48$, $p = .07$, suggesting that conditioned discrimination transferred well across phases and contexts.

A limitation of this study involves the low physiological responding during anticipation of the US. Future studies may employ a US that is more typical for fear conditioning (e.g., electric shock or loud tone) or employ a design that impedes quick habituation to the US. Furthermore, mostly undergraduate females participated in this study, and it is unclear whether the findings may be generalized to other populations. The current findings need replication, but may serve as a starting point for future studies exploring the role of dual-tasks in preventing fear renewal.

In conclusion, this study explored a new way to counter the return of fear after extinction. The findings suggest that imagery of a US representation while conducting a taxing dual-task may attenuate the renewal of fear.

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Chapter 5

Countering fear renewal: Changes in the UCS representation generalize across contexts

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Abstract

After treatment of anxiety disorders, fear often returns. Analogue studies show that outside the extinction context the conditional stimulus (CS) activates the acquisition memory (CS predicts unconditional stimulus; UCS), rather than the extinction memory (CS does not predict UCS). Conditioning theory postulates that fear also diminishes after a reduction in the subjective cost of the UCS, which can occur in absence of in any changes in the CS-UCS association. We hypothesized that fear reduction via 'UCS deflation' generalizes across context. Healthy students underwent acquisition in context A with neutral CSs and 100 dB white noise as UCS. One group received post-conditioning UCS exposure, in which UCS intensity decreased over time ('ABAdefl'). Another group received UCS presentations at equal intensity ('ABActrl'). Two groups did a filler task ('ABB'; 'ABA'). Then, all groups underwent extinction in context B and were retested in context A (ABA-groups) or B (ABB-group). During each CS participants rated UCS expectancy and UCS cost. Results showed the typical increase in UCS expectancy following the context switch from extinction to test phase. In contrast, UCS deflation caused a reduction in cost ratings that was maintained after the context change. Findings suggest that UCS deflation techniques may reduce fear renewal.

Introduction

Cognitive behavioral therapy (CBT) is the gold standard treatment for anxiety disorders (e.g., Hofmann & Smits, 2008; Olatunji, Cisler, & Deacon, 2010). One of its central features is exposure to the feared (conditional) stimulus (CS, e.g., Arch & Craske, 2009). When patients learn that the CS is not followed by the feared outcome (unconditional stimulus, UCS), learned fear (conditional response; CR) decreases. Although many patients show clinically significant improvement after CBT (50-80%; Barlow, Allen, & Choate, 2004), there is much room for improvement. For example, studies have found that relapse rates following exposure-based therapies are about 23% among panic disorder patients (2-14 years post-treatment; Fava, Rafanelli et al., 2001) and 13% among social phobia patients (2-12 years post-treatment; Fava, Grandi et al., 2001). Hence, there is a clear need for research about relapse prevention.

Experimental research has revealed several conditions that trigger the return of fear after extinction, including being exposed to CSs outside the extinction context (renewal), the passage of time (spontaneous recovery), and being exposed to an unsignaled UCS after extinction (reinstatement) (for reviews, see Bouton, 2002; Hermans, Craske, Mineka, & Lovibond, 2006; Craske, Liao, Brown, & Vervliet, 2012, Vervliet, Craske, & Hermans, 2013). These phenomena reflect context effects (Bouton, 2002). In ABA renewal, fear is acquired in context "A", extinguished in context "B", and typically returns when CSs are represented in context "A". In spontaneous recovery, “the passage of time may naturally provide a gradually changing context” (Bouton, 2002). Finally, reinstatement is only observed when the CS is tested in the same context in which UCS re-exposure took place, which indicates that the effect is due to context conditioning. Notably, these phenomena strongly suggest that extinction learning does not erase the learned association between the CS and UCS, but rather leads to the development of an *inhibitory* CS-UCS association (e.g., Bouton, 2002; Myers & Davis, 2002). Accordingly, return of CRs likely depends on the retrieval of the conditioning memory or the extinction memory.

Based on these insights, different methods have been examined that aim to strengthen the extinction memory and/or facilitate its retrieval. Examples are increasing the number of extinction trials, using cues during extinction that serve as later reminders of the procedure, conducting extinction in multiple contexts, and the administration of drugs (i.e., D-cycloserine; for an overview, see Vervliet et al., 2013). Theoretically, only inducing and strengthening the extinction memory means that the conditioning memory is left untouched (Vervliet et al., 2013). This may be problematic. For instance, extinction retrieval cues may not always be present or

CSs might be encountered in contexts that were not targeted during extinction, which may cause relapse. In sum, pre-clinical research should aim to overcome the context-driven return of fear and it seems fruitful to study methods that aim to change the conditioning memory.

Several studies have examined whether extinction learning immediately after fear learning disrupts the consolidation of the conditioning memory. These studies were inconclusive: immediate relative to delayed extinction reduced relapse (Norrholm et al., 2008), increased relapse (Huff, Hernandez, Blanding, & LaBar, 2009), or had no effect (Schiller et al., 2008). A promising and more feasible approach is to interfere with the reconsolidation of conditioning memory. This approach is based on the notion that when a consolidated memory is retrieved from long-term memory, it enters a labile state in which it can be 'updated', and whereupon it restabilizes (i.e., reconsolidates, Nader & Hardt, 2009). Kindt and colleagues have shown that administration of propranolol shortly before or after memory retrieval resulted in erasure of the CR (i.e., fear-potentiated startle response) that was maintained after a reinstatement procedure (e.g., Kindt, Soeter, & Vervliet, 2009; Sevenster, Beckers, & Kindt, 2012; Soeter & Kindt, 2010) and after a context switch (Soeter & Kindt, 2012).

An alternative method to change the conditioning memory, that does not involve a drug manipulation, is 'UCS revaluation'. Contemporary conditioning theory posits that CR intensity depends not only on the strength of the CS-UCS association, but also on the subjective cost of the UCS (Davey, 1997). As Nelson, Lickel, Sy, Dixon, and Deacon (2010) put it: "without the perception of negative consequences, an extremely likely event would not create fear" (p. 214). A revaluation of the UCS may thus change CR intensity. For instance, if post-conditioning information causes a more negative evaluation of the UCS, called UCS *inflation*, then CRs get stronger. Conversely, if a post-conditioning experience renders a less negative evaluation of the UCS (UCS *deflation*), then CRs get weaker. Experimental studies support this theory. CR strength enhances (White & Davey, 1989) or weakens (Hosoba, Iwanaga, & Seiwa, 2001) following stepwise increases or decreases, respectively, in the intensity of UCS only presentations after conditioning, compared to a control condition that receives UCS only presentations of equal intensity.

These experiments show that UCS revaluation and subsequent changes in the CR can occur independently of any changes in the CS-UCS association. If context-driven return of fear results from a failure to activate the extinction memory, and if fear reduction following UCS deflation does not depend on the formation and activation of extinction memory, then fear reduction acquired via UCS deflation should *generalize across contexts*. Using the ABA renewal paradigm, two humans studies that tested this hypothesis have been reported so far.

Rather than achieving UCS deflation by repeated UCS exposure, these studies used imagery to devalue the UCS memory representation. UCS expectancy ratings were used as a self-report measure of fear. In the first study, participants learned the association between a CS picture (car) and a UCS picture (mutilated child; Dibbets, Poort, & Arntz, 2012). During subsequent extinction trials they imagined and rescripted their mental image of the CS, context, and UCS into a more acceptable image. The intervention reduced the negative evaluation of the UCS as well as renewal of UCS expectancy. However, the intervention group showed higher UCS expectancy compared to the control group both during the extinction phase and at the last extinction trial. Because the authors only corrected their analyses for the latter difference, it is unclear whether differences during extinction may have affected renewal. In the second study, a dual task approach was examined. Experimental studies showed that making eye movements during recall of aversive memory renders the memory less vivid and emotional (for review, see Van den Hout & Engelhard, 2012; see also Engelhard et al., 2011). According to working memory theory, eye movements use working memory resources that can therefore not be used for recall. As a result, the memory comes to mind less vividly and emotionally and is reconsolidated as such. In a study that built on the work of Dibbets et al. (2012), participants learned and unlearned the association between a CS (geometrical shape) and a UCS (same aversive picture) and then recalled the UCS image while making eye movements or keeping the eyes stationary (Leer, Engelhard, Dibbets, & Van den Hout, 2013). In contrast to what was expected, the groups did not differ in reductions in memory vividness/emotionality, thus no evidence was provided for UCS deflation. However, the dual task did reduce renewal of UCS expectancy. The authors speculated that maybe the dual task rendered the UCS representation less accessible. This was indeed found in a recent reaction time experiment (Van den Hout, Bartelski, & Engelhard, 2013).

In sum, two studies have provided tentative support for the hypothesis that UCS deflation reduces fear renewal. The current study aimed to replicate and extend these findings. Previous investigations into the effect of UCS deflation on CR have only measured self-reported UCS expectancy. Notably, however, UCS deflation primarily reflects a reduction in the (anticipated) subjective cost of the UCS and can occur in absence of any changes in the CS-UCS association (Davey, 1997; Hosoba et al., 2001). Therefore, and in line with other research in the field of

anxiety (e.g., Butler & Mathews, 1983; Carr, 1974; Nelson et al., 2010; Smith & Bryant, 2000), the present study assessed both probability and *cost estimates*[1].

An ABA renewal paradigm was used to test the hypotheses. This paradigm is preferable to a reinstatement procedure, because reinstatement undoes the effect of UCS deflation (i.e., reestablishes the UCS representation), and to a spontaneous recovery procedure, which is less time-efficient. Context-driven recovery of UCS expectancy was examined by comparing an 'ABA' group to an 'ABB' group. A third group received post-conditioning UCS exposure, in which UCS intensity decreased over time ('ABAdefl'). We tested whether this latter intervention reduces cost estimates during subsequent CS presentation and whether this reduction persists after a context switch. Previous research suggests that exposure to the UCS, just like exposure to the CS, inhibits the CS-UCS association (Dickinson & Burke, 1996; Storsve, McNally, & Richardson, 2012). To control for the effects of UCS exposure, a fourth group received the same number of UCS presentations at *equal* intensity ('ABActrl', cf. Hosoba et al., 2001). We hypothesized that the UCS deflation procedure (1) immediately reduces cost and probability estimates, and (2) that only the reduction in cost estimates is maintained after a context switch.

Method[2]

Participants

Participants were 121 females (mostly undergraduates). A priori exclusion criteria were being diagnosed with an anxiety disorder, a heart problem, or epilepsy, and recent intake of tranquilizers. By order of appearance, participants were randomly assigned to one of four groups: ABB, ABA, ABAdefl, ABActrl. Five participants were excluded because of an error in the conditioning task. Eleven participants were excluded because their UCS expectancy score at the final acquisition trial was below 70 for the CS+ (i.e., CS followed by UCS) or above 30 for the CS- (i.e., CS not followed by UCS) (cf. Leer et al., 2013), because this likely indicates that they did not understand the experimental procedure or did not pay sufficient attention to provide reliable data (Dibbets et al., 2012). The final sample consisted of 105 females (age: M = 21.27 years, SD = 2.49, range = 18-40): ABB (n = 24), ABA (n = 27), ABAdefl (n = 25), and ABActrl (n = 29).

[1] The term 'cost estimate' is distinguished from the term 'unpleasantness rating', which in the present study reflects the unpleasantness of the UCS that participants had received.
[2] The study was approved by the local ethical committee.

Apparatus and stimulus material

The conditioning task was programmed in E-Prime 2.0 (Psychology Software Tools) and presented via a 17-inch monitor. CSs were black triangles with legs of 150 mm and a hypotenuse of 215 mm. One triangle had its right angle in the lower left corner, the other in its upper right corner. The UCS was white noise that was presented at 100 dB(A) for 1 s through stereo headphones (cf. Hosoba et al., 2001).

Questionnaire

The State Trait Anxiety Inventory (STAI-DY; Spielberger, Gorsuch, & Lushene, 1970) was used to assess state and trait anxiety, because they may affect fear learning (Grillon et al., 2006; Lissek et al., 2005). Each construct was measured by 20 items that were scored on a 1-4-point scale (1=*not at all,* 4=*severely*).

Procedure

Testing took place in a dimmed room. Participants were seated in front of a computer and received written and oral information about the experimental procedure. They were informed that loud noises would be presented that would not be painful or harmful, and they could decline participation at any time. After providing written informed consent and filling out the STAI, participants put on headphones. They were told that figures would be presented on the monitor and that one figure might be followed by a short, loud white noise tone. They were instructed to figure out after which figure the tone was presented and to answer two questions during the presentation of each figure by clicking on visual analogue scales (VASs) at the bottom of the screen. The first VAS assessed UCS expectancy: 'To what extent do you expect this figure to be followed by a white noise tone?' (0=*certainly no white noise tone*; 100=*certainly a white noise tone*). The second VAS assessed UCS cost: 'How annoying would you find it *if* this figure would be followed by a white noise tone?' (0=*not at all annoying*; 100=*very annoying*). CSs were randomly presented with a maximum of two similar CSs in succession. CS duration was 12 s. Each VAS had a fixed duration of 6 s. Inter-stimulus-intervals (ISIs) randomly varied between 5, 8 and 11 s.

Practice phase. First, participants practiced completing the two VASs. In between two practice trials, they were told that one may expect a white noise tone *without* finding it annoying if it would follow, and vice versa. They were told: “if you expect the white noise (high expectancy), but don’t find it annoying (low cost), then please click on the right side of the first VAS and the left side of the second VAS” and “if you don’t expect the white noise (low expectancy), but find the noise annoying if it does follow (high cost), then please click on the left side of the first VAS and the right side of the second VAS”.

Acquisition phase. During acquisition, participants were exposed to six CS+ presentations that were each immediately followed by the UCS, and six CS- only presentations. This phase took place in context "A". Context was defined by the background color of the monitor (cyan or yellow) that was arranged via a counterbalancing schema.

Intervention phase. The intervention phase took place in context "B". Participants in the ABAdefl group underwent a UCS deflation procedure. They were given twenty 1-s presentations of white noise with 5-s ISIs. Intensity decreased from 100 to 70 dB in the following steps: 1x100 dB, 1x90 dB, 1x80 dB, 17x70 dB. The ABActrl group received twenty 1-s presentations of white noise, all at 100 dB, with 5-s ISIs. Groups ABB and ABA did a filler task that took place in context “B” and was matched on time to the interventions. In this task, participants were presented six different guitar pictures, each twice, in a random order, and indicated whether they saw the picture for the first or the second time (cf. Leer et al. 2013; Engelhard, Leer, Lange, & Olatunji, 2014).

Extinction phase. Participants were prompted via the monitor to place their hand on the mouse. Then the extinction phase started. Participants were exposed to eight presentations of each CS type in context "B". The UCS was never presented.

Test phase. The test phase immediately followed the extinction phase, and took place in context "A" (ABA groups) or "B" (ABB group). Each CS type was presented twice. The UCS was never presented.

Post-experimental questionnaire. After the experiment, participants filled out a questionnaire that served a manipulation check. The first question, 'Did you have the impression that the intensity of the white noise tone changed during the experiment?', was answered by forced choice: 'No, the white noise tone did not change in intensity', 'Yes, the white noise tone became stronger', or 'Yes, the white noise tone became weaker'. If the answer to the first question was 'Yes', participants indicated whether they had the impression that 'At some point, the original white noise tone was presented weaker/stronger' or 'At some point, another, new tone was presented'. Next, participants were asked to recall and rate the first and last white noise tone they heard during the experiment on VASs that ranged from 0=*not at all unpleasant* to 100=*very unpleasant*. Finally, they were debriefed and remunerated.

Data analysis

Preliminary analyses. First, as a randomization check, one-way ANOVAs were used to examine between-groups differences in age, state anxiety, and trait anxiety. Second, as a manipulation check, a 2 (Time: pre-intervention vs. post-intervention) x 3 (Group: ABB/ABA vs. ABAdefl vs. ABActrl) ANOVA was conducted on UCS unpleasantness ratings. Data of

groups ABB and ABA were collapsed in this analysis, because they did the same filler task. Third, to provide a proof of principle for ABA-renewal, we examined whether UCS expectancy increased during the acquisition phase, extinguished during the extinction phase, and returned after the context-switch. Acquisition and extinction were tested by comparing the first and last trial of each phase[3] (cf. Sevenster, Beckers, & Kindt, 2012; Soeter & Kindt, 2012). The context effect was tested with a 2 (Trial: E8 vs. T1) x 2 (CS) x 4 (Group) ANOVA. Follow-up ANOVAs were used to test (1) whether the ABA groups showed stronger renewal of UCS expectancy compared to ABB, (2) whether renewal was comparable across the three ABA groups, and (3) whether differential responding increased over time in the ABA groups and was unaffected in the ABB group.

Confirmatory analyses. The first hypothesis that UCS deflation reduces cost and probability estimates was investigated by 2 (Trial: A6 vs. E1) x 2 (CS) x 3 (Group) ANOVAs. Ratings of the two filler task groups (ABB; ABA) were collapsed in these analyses. Using follow-up ANOVAs we compared ABAdefl with ABA/ABB and ABAdefl with ABActrl. The critical test of the hypothesis was a planned comparison that examined whether in ABAdefl ratings decreased from trial A6 to trial E1. Using post-hoc comparisons we explored whether ratings changed from trial A6 to trial E1 in ABB/ABA and ABActrl.

The second hypothesis that reductions in cost estimates maintain after a context switch was investigated using a 2 (Trial: A6 vs. T1) x 2 (CS) x 4 (Group) ANOVA[4]. In a follow-up ANOVA we compared ABAdefl with ABA. The critical test of the hypothesis was a planned comparison that examined whether in ABAdefl ratings decreased from trial A6 to trial T1. Post-hoc, we explored rating changes from trial A6 to trial T1 in ABA.

Exploratory analyses. To explore whether cost estimates changed during the acquisition phase and whether the estimates were comparable for the two CS types, a 2 (Trial: A1 vs. A6) x 2 (CS) x 4 (Group) ANOVA was conducted. Next, to test whether the expected intervention effect from trial A6 to trial T1 continued or ceased at trial T2, a 2 (Trial: A6 vs. T2) x 2 (CS) x 4 (Group) ANOVA was performed. Furthermore, two unexpected patterns were explored. First,

[3] This strategy is preferable to including comparison of all trials, because we were interested in the result of the procedures, i.e. whether participants successfully (un)learned the CS-UCS contingency, rather than the trial-by-trial performance during each phase.

[4] Typically, renewal of fear (e.g., UCS expectancy ratings or fear-potentiated startle responses) is tested comparing the last extinction trial with the first test trial. However, as we anticipated that cost estimates are insensitive to contextual information, which should be the case in each group, we did not expect any changes over time or between groups from trial E8 to trial T1. Therefore, the 'E8 vs. T1' contrast was not used as a test of the second hypothesis.

change in cost ratings during the extinction phase was examined using a 8 (Trial: E1-E8) x 2 (CS) x 4 (Group) ANOVA. Second, change in cost ratings from trial E8 to trial T1 was examined using a 2 (Trial: E8 vs. T1) x 2 (CS) x 4 (Group) ANOVA.

Alpha adjustment. Expected effects were tested at $\alpha = .05$. All post-hoc and exploratory analyses were controlled for Type I error rates for multiple comparisons. Applying the Bonferroni correction resulted in testing at a .002 (.05/25) alpha level.

Results

Randomization and manipulation check

Groups did not differ in age, $F < 1$, state anxiety, $F < 1$, or trait anxiety, $F(3, 104) = 1.02$, $p = .389$, indicating successful randomization. Most participants that underwent the UCS deflation procedure ($n = 18/25$) reported that the tone became weaker, and all of them (18/18) reported that it was the original tone getting weaker. Six participants did not report a change in volume, for 1 participant these data were missing. In the control group, 24 participants reported that the tone remained of equal intensity, 4 reported that it became louder, and 1 reported that it became weaker. In the filler task groups, 46 participants reported that the intensity of the tone had not changed during the experiment, 4 reported that it became louder, 1 reported that it became weaker.

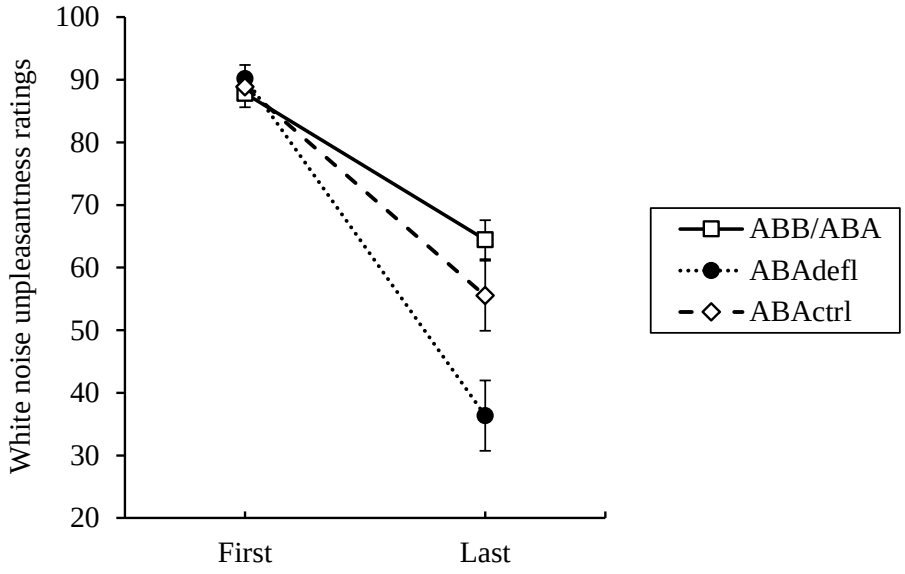

Fig. 1 Mean (± SEM) unpleasantness ratings of the first and last white noise presentations. ABB/ABA represents the ABA and ABB groups combined.

Figure 1 shows unpleasantness ratings of the first and last white noise presentations. Ratings of groups ABB and ABA were collapsed as they underwent the exact same procedure up to here. ANOVA showed effects of Time, $F(1, 102) = 165.30$, $p < .001$, $\eta\rho^2 = .618$, Group, $F(2, 102) = 6.32$, $p = .003$, $\eta\rho^2 = .110$, and of Time x Group, $F(3, 102) = 9.83$, $p < .001$, $\eta\rho^2 = .162$. Follow-up ANOVAs showed that reductions were larger in ABAdefl compared to ABB/ABA, $F(1, 74) = 19.26$, $p < .001$, $\eta\rho^2 = .207$, and compared to ABActrl, $F(1, 52) = 6.71$, $p = .012$, $\eta\rho^2 = .114$. ABActrl, however, did not differ from ABB/ABA, $F(1, 78) = 2.45$, $p = .122$. In sum, the UCS deflation procedure successfully reduced UCS unpleasantness ratings.

Probability estimates

Acquisition. Figure 2 depicts the mean UCS expectancy ratings. ANOVA showed main effects for Trial, $F(1, 101) = 18.51$, $p < .001$, $\eta\rho^2 = .155$, and CS, $F(1, 101) = 1131.94$, $p < .001$, $\eta\rho^2 = .918$, which were qualified by a Trial x CS interaction, $F(1, 101) = 1069.02$, $p < .001$, $\eta\rho^2 = .914$; UCS expectancy at trial A6 was higher to CS+ than to CS-, $t(104) = 83.81$, $p < .001$, $d = 14.97$, indicating that acquisition was successful. There were no main or interaction effects involving Group, largest $F(3, 101) = 2.03$, $p = .115$, indicating that the differential change over time was comparable across the groups.

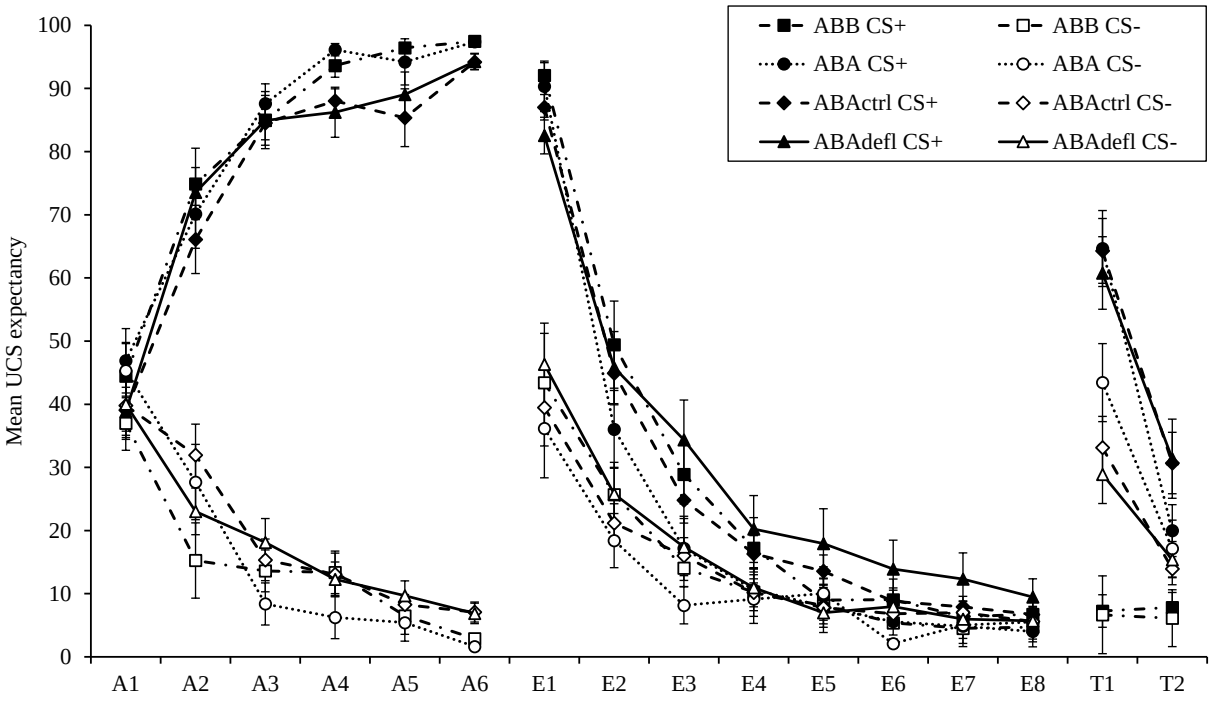

Fig. 2 Mean (± SEM) UCS expectancy ratings during the acquisition phase (A1-A6), extinction phase (E1-E8), and test phase (T1-T2).

Immediate intervention effect. ANOVA did not yield a Trial x CS x Group interaction, $F < 1$. This result was opposite to our expectation that exposure to the UCS reduces UCS expectancy, and means that any changes in cost estimates produced by the UCS deflation procedure cannot be explained by changes in the CS-UCS association.

Extinction. ANOVA showed main effects for Trial, $F(1, 97) = 600.46$, $p < .001$, $\eta\rho^2 = .861$, and CS, $F(1, 97) = 125.14$, $p < .001$, $\eta\rho^2 = .563$, with a decrease in ratings over time and overall higher ratings for CS+ (see Figure 2), which was qualified by a Trial x CS interaction, reflecting that the decrease in UCS expectancy was larger for CS+ than for CS-. At trial E8, CS+ and CS- ratings did not differ, $t(104) = 1.34$, $p = .183$, indicating that UCS expectancy had fully extinguished. There were no significant main or interaction effects involving Group, largest $F(3, 97) = 1.73$, $p = .166$, indicating that differential change over time did not differ between groups.

Renewal. Using an ANOVA we investigated whether the context manipulation produced renewal of UCS expectancy. There were main effects of Trial, $F(1, 101) = 437.42$, $p < .001$, $\eta\rho^2 = .812$, CS, $F(1, 101) = 32.88$, $p < .001$, $\eta\rho^2 = .246$, and Group, $F(3, 101) = 23.50$, $p < .001$, $\eta\rho^2 = .411$, that were qualified by two-way interactions of Trial x CS, $F(1, 101) = 26.85$, $p < .001$, $\eta\rho^2 = .210$, Trial x Group, $F(3, 101) = 45.32$, $p < .001$, $\eta\rho^2 = .574$, and CS x Group, $F(3, 101) = 3.45$ $p = .020$, $\eta\rho^2 = .093$. As expected, the three-way interaction was significant, $F(3, 101) = 3.43$, $p = .020$, $\eta\rho^2 = .093$. A follow-up ANOVA comparing ABB with the three ABA groups combined revealed a significant three-way interaction, $F(1, 103) = 9.92$, $p = .002$, $\eta\rho^2 = .088$, demonstrating that the return of UCS expectancy was larger in the ABA groups than in ABB. Separate ANOVAs showed that in the ABA groups there was a Trial x CS interaction, $F(1,80) = 30.81$, $p < .001$, $\eta\rho^2 = .278$, reflecting differential change for the two CSs over time, but not in ABB, $F < 1$. In the ABA groups, ratings were higher to CS+ than to CS- at trial T1, $t(104) = 5.60$, $p < .001$, $d = .69$, indicating that the context switch after extinction caused a return of UCS expectancy. Finally, a 2(Trial: E8 vs. T1) x 2(CS) x 3(ABA groups) ANOVA did not yield a three-way interaction, $F < 1$, meaning that the UCS deflation procedure did not affect renewal of UCS expectancy.

Cost estimates

Acquisition. Figure 3 depicts the cost estimates collapsed across the two CSs, because the intervention effects were comparable across CS types, and as a percentage of trial A6. As such, the expected differential change from trial A6 to trial E1 and from trial A6 to trial T1 can directly be seen. Note that the analyses were conducted on the original data (see Table 1), and not on these standardized cost estimates. ANOVA showed only a main effect of Trial, $F(1, 100)$

= 62.27, $p < .001$, $\eta\rho^2 = .384$, and a Trial x CS interaction, $F(3, 100) = 13.13$, $p < .001$, $\eta\rho^2 = .116$, with higher ratings to CS- ($M = 53.75$, $SD = 31.38$) than to CS+ ($M = 44.45$, $SD = 28.51$) at trial A1, $t(104) = 3.75$, $p < .001$, $d = .31$, and comparable ratings at trial A6 (CS-: $M = 65.30$, $SD = 35.01$; CS+: $M = 74.42$, $SD = 26.11$), $t(103) = 2.17$, $p = .033$.

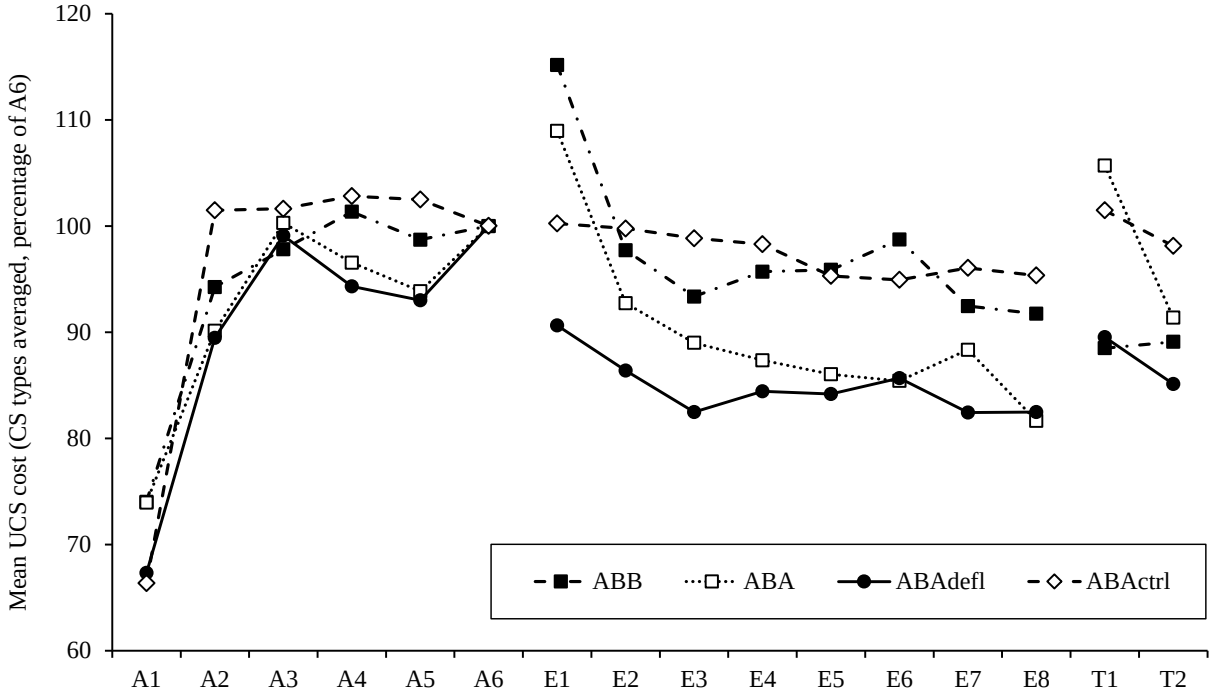

Fig. 3 Mean UCS cost ratings during the acquisition phase (A1-A6), extinction phase (E1-E8), and test phase (T1-T2), averaged across CS types, and presented as a percentage of the last acquisition trial.

Immediate intervention effect. ANOVA showed a Trial x CS effect, $F(1, 100) = 4.00$, $p = .048$, $\eta\rho^2 = .038$, reflecting that the difference in ratings between the two CS types was smaller at E1 than at A6, and the crucial Trial x Group effect was significant, $F(2, 100) = 6.23$, $p = .003$, $\eta\rho^2 = .111$. No other significant main or interaction effects were found. This means that change in UCS cost ratings over time differed between the interventions, irrespective of whether ratings were provided during CS+ or CS-. To follow-up, a similar ANOVA that compared ABB/ABA to ABAdefl showed a Trial x Group effect, $F(1, 73) = 13.68$, $p < .001$, $\eta\rho^2 = .158$; ratings decreased from A6 to E1 in ABAdefl, $t(24) = 2.45$, $p = .022$, $d = .35$ (planned comparison), and increased in ABB/ABA, $t(50) = 3.43$, $p = .001$, $d = .38$. Ratings did not change in ABActrl, $t < 1$ (see Figure 3). Further analyses showed no significant differences in cost estimates between ABActrl and ABB/ABA, $F(1,77) = 3.55$, $p = .063$, and between ABActrl

and ABAdefl, $F(1,50) = 1.79$, $p = .188$. In sum, the results support the first hypothesis: the UCS deflation procedure causes an immediate reduction in cost estimates. The effect of the control procedure, however, was indistinct.

Extinction. Figure 3 suggests overall decreases in cost ratings and no differences between conditions. Indeed, ANOVA showed a main effect of Time, $F(7, 693) = 6.40$, corrected $p = .001$, ηρ2 = .061, ε = .337, but not of CS, $F(1, 99) = 3.62$, $p = .060$, or Condition, $F < 1$. There were no interaction effects, Fs < 1, indicating that in all groups, cost estimates decreased in the course of the extinction phase. Exploratory analyses further showed a decrease in ratings from trial E1 to E2, $F(1, 100) = 8.28$, $p = .005$, $\eta\rho^2 = .077$, that was irrespective of Group, $F(3, 100) = 1.40$, $p = .247$. There was no evidence for an additional decrease in ratings from trial E2 to trial E8, $F(2.23, 222.97) = 1.47$, $p = .230$.

Renewal. Our second hypothesis was that the immediate reduction in cost estimates caused by the UCS deflation procedure would persist after a context switch. Figure 3 indeed suggests that the initial decrease from trial A6 to trial E1 in ABAdefl, relative to ABA, was maintained after the context change just before trial T1. ANOVA showed a main effect of CS, $F(1, 100) = 5.01$, $p = .027$, $\eta\rho^2 = .048$, but not of Trial, $F(1, 100) = 2.07$, $p = .153$, or Group, $F(3, 100) = 1.91$, $p = .133$. The crucial Trial x Group interaction was significant, $F(3, 100) = 3.05$, $p = .032$, $\eta\rho^2 = .084$. None of the other interactions were significant, indicating that change in UCS cost ratings over time differed between the groups, irrespective of whether ratings were provided during CS+ or CS-. A follow-up ANOVA that compared group ABAdefl with group ABA showed a significant Trial x Group interaction, $F(1, 50) = 12.90$, $p < .001$, $\eta\rho^2 = .205$; ratings at T1 were lower than at A6 in ABAdefl, $t(24) = 2.90$, $p = .008$, $d = .38$ (planned comparison), and there was a trend towards higher ratings at T1 compared to at A6 in ABA, $t(26) = 2.07$, $p = .048$ (note the Bonferroni correction; see Figure 3). The results therefore confirmed the second hypothesis. Notably, the beneficial effect of the intervention ceased at trial T2: ANOVA comparing trial A6 to trial T2 did not show Trial x Group or Trial x CS x Group effects, Fs < 1.

Finally, Figure 3 shows an unexpected pattern: ratings seemed to increase from trial E8 to trial T1 in the ABA groups, but not in the ABB group, which may imply that cost estimates are partly context-dependent. ANOVA showed only a significant main effect of Trial, $F(1, 101) = 8.12$, $p = .005$, $\eta\rho^2 = .074$, which was qualified by a significant Trial x Group interaction, $F(3, 101) = 3.60$, $p = .016$, $\eta\rho^2 = .097$; ratings increased from trial E8 to trial T1 in ABA, $t(26) = 3.33$, $p = .003$, $d = .57$, but not in ABB, $t < 1$, ABActrl, $t < 1$, or ABAdefl, $t(24) = 1.17$, $p = .253$.

Table 1. *Mean (SEM) cost estimates, averaged across CS types, during the acquisition phase (A1-A6), extinction phase (E1-E8), and test phase (T1-T2).*

Trial	A1	A2	A3	A4	A5	A6	E1	E2	E3	E4	E5	E6	E7	E8	T1	T2
Group																
ABB	48.35	61.56	63.88	66.20	64.48	65.32	75.22	63.83	60.98	62.51	62.62	64.49	60.39	59.92	57.80	58.19
	(5.19)	(5.34)	(5.31)	(4.95)	(4.00)	(3.86)	(3.86)	(5.50)	(5.90)	(6.57)	(7.14)	(7.33)	(7.12)	(6.88)	(7.21)	(7.14)
ABA	54.43	66.34	73.82	71.07	69.09	73.61	80.20	68.26	65.51	64.30	63.33	62.87	65.03	60.10	77.80	67.26
	(5.07)	(5.53)	(4.99)	(4.69)	(4.66)	(4.60)	(4.24)	(6.71)	(6.85)	(6.88)	(6.96)	(6.93)	(7.02)	(7.41)	(4.12)	(6.21)
ABAdefl	49.64	65.98	73.09	69.56	68.60	73.75	66.84	63.70	60.82	62.26	62.07	63.18	60.79	60.82	66.03	62.77
	(5.54)	(3.85)	(3.90)	(4.80)	(4.94)	(3.79)	(5.04)	(5.77)	(6.23)	(5.78)	(6.26)	(6.28)	(6.67)	(6.65)	(4.31)	(5.33)
ABActrl	44.28	67.73	67.82	68.62	68.40	66.74	66.90	66.58	65.97	65.59	63.60	63.36	64.10	63.63	67.73	65.49
	(5.43)	(4.39)	(4.75)	(4.12)	(4.29)	(4.30)	(4.41)	(4.84)	(5.16)	(5.38)	(5.66)	(5.69)	(5.86)	(5.89)	(3.97)	(5.00)

Discussion

The present study investigated the effect of UCS deflation on UCS probability and cost estimates. Findings showed ABA renewal of UCS expectancy, which corroborates earlier research (e.g., Effting & Kindt, 2007; Neumann, Lipp, & Cory, 2007) and supports the validity of our paradigm. The first hypothesis was partly confirmed: as expected, the UCS deflation procedure reduced cost estimates. Note, however, that this effect was not statistically different from the effect of the control procedure. Inconsistent with the hypothesis, probability estimates were not affected. In line with the second hypothesis, results indicated that the reduction in cost estimates persisted after a context switch. An unexpected finding was that cost estimates decreased as a result of CS only presentations and increased (but only in group ABA) following the context switch.

Several explanations may account for the finding that our UCS deflation intervention reduced cost estimates. First, the intervention may have *updated* the UCS representation, meaning that non-associative learning (i.e., UCS deflation) took place. In support, we found that in group ABAdefl the UCS expectancy ratings showed the usual renewal effect after the final ABA context switch, indicating that test context "A" retrieved the conditioning memory. The UCS cost ratings, however, were not affected by the context switch, suggesting that the original CS-UCS association was activated but that the subjective aversiveness of the UCS had changed. Alternatively, associative learning may account for our findings. For instance, our procedure may have caused participants to expect a different stimulus (i.e., the 70 dB tone) rather than the original UCS. This explanation, however, is not in line with Wagner (1981) who postulated that an excitatory association between two stimuli develops only when they are presented together (i.e. paired or successively). In the current study the CS and 70 dB tone were never paired. It might also be proposed that exposure to the UCS only weakened the CS-UCS

association (Dickinson & Burke, 1996), which suppressed activation of the UCS representation upon CS presentation and thereby affected cost estimates. Yet this explanation can also be ruled out because no reduction in UCS expectancy was observed after the intervention phase, and UCS presentations at equal intensity (ABActrl) did not affect cost estimates. In sum, it is likely that UCS deflation accounts for the reduction in cost estimates.

The main finding of this experiment was that the immediate reduction in cost estimates (in ABAdefl) was maintained after the context switch. Earlier research has shown that administration of propranolol shortly after reactivation of the conditioning memory, which supposedly disrupts its reconsolidation, diminishes renewal of fear (Soeter & Kindt, 2012). The current findings suggest that changing the conditioning memory without the use of pharmacological agents may attenuate renewal of cost estimates, which is an important determinant of conditional fear.

Before discussing possible clinical implications, several issues deserve further attention. First, Carr (1974) noted that the intensity of conditional fear is a "multiplicative function of the subjective cost of an event and its subjective probability" (p. 315). Although the present experiment is novel in demonstrating that the two estimates are differently informed by context, this study was limited because it only measured conditional fear indirectly. Any firm conclusions regarding fear renewal thus await future research that may include more direct measures of fear, e.g. self-reported fear or fear-potentiated startle responses. Second, Dibbets et al. (2012) showed that UCS deflation reduced UCS expectancy, but we did not. One explanation is that our UCS deflation procedure did not work. However, the observed reductions in UCS unpleasantness ratings and cost estimates disconfirm this hypothesis. Alternatively, differences in methodology may account for the discrepancy: Dibbets et al. (2012) used a rescripting manipulation, in which participants were asked to change the script of a car (CS) causing a deadly accident, as illustrated by the picture of a mutilated child (UCS), and imagine another script in which the child survived the accident and recovered. On the one hand, it seems logical for participants to therefore adjust their UCS expectancy ratings. On the other hand, it is unclear whether these effects would also have been found if cost estimates were assessed next to probability estimates. In contrast to our expectation, the present study did not find that exposure to the UCS only causes the formation of an inhibitory CS-UCS association (Dickinson & Burke, 1996), since no immediate reduction in UCS expectancy ratings was observed. Rather, this finding supports a model in which post-conditioning CS only exposure, but not UCS only exposure, leads to the development of an inhibitory CS-UCS association (Wagner, 1981). Third, Figure 2 suggests that the deflation procedure had an effect on renewal

of UCS expectancy ratings to the CS- compared with the ABA group. However, exploratory analyses showed that this effect was not significant. Therefore, it seems unlikely that it may be an alternative explanation of the observed renewal effects of cost estimates. In addition, given that the deflation procedure did not directly affect UCS expectancy ratings (i.e., on trial E1), any effects on renewal of UCS expectancy would be unlikely. Fourth, Figure 3 suggests that there may have been some UCS deflation in ABActrl; relative to groups ABB and ABA ratings decreased from trial A6 to trial E1. This effect, however, did not reach statistical significance. Moreover, there was no evidence for a decrease in UCS unpleasantness in ABActrl compared to the ABB and ABA groups and compared to ABAdefl. Hence, it is unclear how the effect of the control procedure should be interpreted. Fifth, there was a general decrease in cost estimates over the course of the extinction phase. Non-reinforced CS presentations might also modify the UCS representation. This explanation has been put forward to account for the typically observed reinstatement of fear following post-extinction UCS exposure, which is believed to reestablish the UCS representation (Rescorla & Heth, 1975). Alternatively, the mere *deactivation* of the UCS representation that results from extinction learning (e.g., Vervliet et al., 2013) may have caused participants to downshift their cost judgments. Only the latter account predicts that (re)activation of the UCS representation, for example by contextual input, causes an increase in cost estimates. Indeed, control group ABA showed an increase in cost estimates following the context change from extinction to test phase (see Figure 3). Seemingly, test context "A" reactivated the excitatory CS-UCS association that was temporarily inhibited, but was unchanged. Finally, the sample consisted of female college students, which may impede generalizability of the findings to others, such as male students or older people.

Provided that future studies replicate the current findings, with more direct measures of fear, there may be practical implications. Currently, CBT consists of techniques that aim to correct both elevated probability and cost estimates (i.e., by evaluating evidence for risk and challenging negative core beliefs; e.g., Craske & Pontillo, 2001). While we do not know any studies that tested the immediate benefits of interventions targeting the CS-UCS contingency (e.g. exposure approaches, e.g. framed as 'behavioral experiment') or UCS cost (e.g. cognitive interventions challenging the aversiveness of the feared event), the present data suggest that changes in contingency beliefs are more prone to relapse, compared to changes in cost estimates, and that therapeutically targeting the latter will reduce relapse rates. Next to challenging the client's beliefs about the actual cost of feared outcomes, imagery techniques may be useful. For example, experimental data showed that making eye movements during recall of emotional past and prospective memories reduces the vividness and emotionality of

these memories (see Van den Hout & Engelhard, 2012). This dual task serves as a UCS deflation technique, because it reduces the subjective aversiveness of negative events. A recent study further showed that the dual task reduces conditional fear (Leer, Engelhard, Altink, & Van den Hout, 2013).

To conclude, this study provided the first evidence that reductions in the subjective cost of the UCS, in contrast to reductions in the subjective probability of the UCS, are context-independent. Future research may elucidate whether targeting cost estimates reduces fear renewal.

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Chapter 6

Blurring the memory of a conditional stimulus intensifies fear generalization

Based on:

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Abstract

Eye movement desensitization and reprocessing (EMDR) is a treatment for posttraumatic stress disorder, and involves having the patient recall a traumatic image and make horizontal eye movements (EM) at the same time. Experimental research has revealed that this dual task procedure decreases vividness, accessibility, and emotionality of the aversive memory. Another key element of EMDR is that after the dual task the patient is asked what thought or image comes to mind, and told to focus on that while making EM again. This latter step has received little empirical attention, but may have adverse effects. Specifically, the blurring of danger cue representations may reduce discriminability between danger cues and perceptually similar stimuli, which may increase fearful responding to safe stimuli. This hypothesis was tested in two experiments with healthy individuals. Experiment I demonstrated that EM during stimulus recall reduces memory vividness and slows down the speed at which the stimulus is discriminated from other stimuli. Experiment II showed that EM during recall of a stimulus that signals danger intensifies fearful responding to a perceptually similar yet non-threat-related stimulus, as evidenced by increases in danger expectancies and skin conductance responses. The latter results were not paralleled by startle EMG data.

Introduction

Eye movement desensitization and reprocessing (EMDR) is among the most effective treatments for posttraumatic stress disorder (PTSD; Bisson et al., 2007; Bradley, Greene, Russ, Dutra, & Westen, 2005; Seidler & Wagner, 2006). Accordingly, it is recommended as a treatment-of-choice for PTSD (e.g., NICE, 2005; Trimbos, 2011). Until recently, however, little was known about its underlying mechanisms (e.g., Gunter & Bodner, 2008). Central to EMDR is that the client holds a distressing memory in mind and makes horizontal eye movements (EM) at the same time by following the therapist's fingers that move back and forth. Next, the therapist asks ”what comes up” and directs the client to focus on this new (feature of the) memory and engage in another set of EM (de Jongh & ten Broeke, 2009). In recent years, many experimental studies have been conducted that have (1) elucidated that the EM component adds to EMDRs effectiveness (Lee & Cuijpers, 2013) and (2) significantly advanced our understanding of *how* the EM work (for a review, see van den Hout & Engelhard, 2012). Specifically, it has been shown that EM work via taxation of working memory (e.g., Gunter & Bodner, 2008). Importantly, these novel insights may lead to improvement of the therapy, which is urgently needed given that about one-third of PTSD patients does not show clinical improvement (Bradley et al., 2005). The current research builds on these insights and investigates how EDMR may further be optimized.

The commonly used laboratory model to investigate the effects of making EM involves having non-clinical participants select a negative autobiographical memory and rate its vividness and emotionality (van den Hout, Muris, Salemink, & Kindt, 2001). They are then asked to visualize the memory for a fixed period of time, either with or without EM. Finally, they retrieve and rate the memory again. Findings are that the dual task, relative to recall only, reduces the memory’s vividness and emotionality (for a meta-analysis, see Lee & Cuijpers, 2013). Additionally, EM reduce self-reported memory completeness (Gunter & Bodner, 2008) and, as demonstrated by a reaction time task, decrease the objective accessibility of the memory (van den Hout, Bartelski, & Engelhard, 2013). An explanation of these findings is that EM use working memory recourses that can therefore not be used for memory recall. As a result, recall is hampered and the memory comes to mind in a ‘blurred’ form. Notably, the widely reported immediate effects are still observed 24 h (Leer, Engelhard, & van den Hout, 2014) and 1 week after the intervention (Gunter & Bodner, 2008), which suggests that the dual task may cause a *permanent* loss of memory detail.

Although these findings seem to advocate the use of dual tasks in the treatment of PTSD, caution is warranted. As mentioned above, the EMDR protocol holds that EM are not only made during recall of the 'hotspot' in traumatic memory (i.e., the worst moment that causes a peak level of emotional distress; e.g., Holmes, Grey, & Young, 2005), but also during thoughts or images that are associated with it ("What comes up?"; de Jongh & ten Broeke 2009). For example, in case of a violent attack it is likely that, next to the hotspot (e.g., the moment the knife was put to my neck), contextual stimuli are recalled as well (e.g., the physical environment or the appearance of the perpetrator). In the current paper we argue that, whereas the blurring of emotional hotspots may have beneficial effects on stress-related complaints, the loss of related (useful) information may actually become a risky endeavor. Specifically, we predict that the blurring of danger cue representations impair their discrimination from perceptually similar stimuli and thereby increases fearful responding to previously safe stimuli.

Contemporary learning theory explains that conditional fearful responding follows from the activation of the trauma memory (e.g., Davey, 1997). Through memory-encoded associations, formerly neutral cues and contexts (conditional stimuli; CSs) that were present at the time of the traumatic event (unconditional stimulus; UCS) are capable of activating the UCS memory and produce a conditional response (CR). For instance, following an assault, the victim may cross the same street (CS) or encounter a person that perceptually resembles the perpetrator (CS). Remembering the assault (UCS) will then automatically elicit a fear response (CR). It can be inferred that, to the degree that one is able to discriminate CSs from perceptually similar stimuli the CR is confined to a small range of stimuli. Evidently, continued fearful responding to original CSs serves an adaptive function: one has good reason to be on guard when coming across the same perpetrator. Generalization of fear and avoidance to objectively *safe* stimuli, however, is maladaptive, and may disrupt daily functioning. Accordingly, fear generalization is seen as a defining feature of clinical anxiety (e.g., Dymond, Dunsmoor, Vervliet, Roche, & Hermans, 2014). Indeed, experimental research has shown that patients suffering from an anxiety-related disorder, relative to healthy controls, fearfully respond to a broader range of perceptually similar stimuli (panic disorder: Lissek et al., 2010; PTSD: Lissek & Grillon, 2012; generalized anxiety disorder: Lissek et al., 2014). Also, using a prospective design, it has recently been demonstrated that stronger generalization is a vulnerability factor for the development of anxiety (Lenaert et al., 2014).

Importantly, these findings show that the degree of generalization not only depends on the actual amount of attributes shared between the novel stimulus and the original CS, but is also affected by individual differences and other factors (Lissek et al., 2008). Most relevant for

the current investigation is the observation that the forgetting of stimulus attributes is related to generalized responding (e.g., Anderson & Riccio, 2005; Riccio, Rabinowitz, & Axelrod, 1994). Both animal and human studies have demonstrated that, as the retention interval lengthens, discrimination between the training stimulus (CS) and novel stimuli deteriorates (Riccio, Richardson, & Ebner, 1984). In addition, studies examining aversively motivated learning have revealed that response strength to the CS is not affected, indicating that particular features of the CS, rather than the CS-UCS contingency, are forgotten over time (e.g., Thomas & Riccio, 1979; see Riccio et al, 1984). More recent research has further shown that individual differences in the specificity of memory retrieval are significantly associated with the extent of generalization (Lenaert et al., 2012). Together, these findings indicate that impaired recall of stimulus characteristics increases the threshold for perceptually similar stimuli to be categorized as 'different'. Accordingly, it can be expected that any intervention that impedes recall of danger cues may broaden the range of stimuli capable of evoking a fear response. Given that 'overgeneralization' is a core feature of anxiety disorders, it is clear that actions that facilitate it should be avoided.

In sum, research on fear generalization suggests that the blurring of memory features other than the trauma hotspot, which is standard EMDR practice, might actually worsen the psychopathology. Obviously, such an outcome is undesirable and would imply that therapists should be alert in determining what memory features to include in the dual task. Nevertheless, this has not been investigated yet. The aim of the current experiments was to test whether EM during CS recall decreases the vividness of the CS representation and thereby (1) hampers its discrimination from other stimuli, and (2) increases the generalization of fearful responding.

Experiment I

Experiment I tested the hypothesis that the dual task reduces vividness of memory of a neutral image and impedes stimulus discrimination. Participants first encoded a picture of a neutral male face. Then they recalled the image with EM (experimental condition) or without EM (control condition), and rated memory vividness. Finally, in a reaction time task, they were presented novel faces that perceptually resembled the original face, and indicated whether or not these images were identical to the original one. We predicted decreases in memory vividness, in discrimination accuracy, and/or in speed in the experimental condition.

Method

Participants

Twenty-seven (mostly) undergraduates participated in exchange for remuneration or course credits. A priori exclusion criteria were pregnancy, serious medical conditions, (past or present) psychiatric diagnoses, having an electronic implanted device (e.g., a pacemaker), and pain or problems related to hands or wrists. After the experiment, knowledge about EMDR was assessed: one participant had linked the experimental procedure to the EM employed in EMDR, and was excluded. The final sample comprised 26 participants (age: *M* = 19.96 years, *SD* = 2.96; 5 males). The experiment was approved by the local ethical committee.

Stimulus material

Two different image sets ('A' and 'B') were created by selecting images of neutral male faces (511x768 pixels) from the Radboud Faces Database (Langner et al., 2010). Each set comprised one 'target image' and three 'test images' (see Figure 1). Test images were morphs between the target image and other neutral male faces (created with Abrosoft Fantamorph software) and resembled the target image in decreasing steps of perceptual similarity.

Fig. 1 Stimulus sets used in Experiment I. The target image and test image 2 from stimulus set A were used as CS and GS, respectively, in Experiment II.

Procedure

Participants were seated in a dimly lighted room about 42 cm in front of a 17-inch monitor (1440x900 pixels). They received oral information about the study and provided written informed consent. The order of the experimental and control conditions was counterbalanced within subjects. When image set A was used in the experimental condition, then image set B was used in the control condition, and vice versa, which was balanced as well. The experiment consisted of 5 phases. During phase 1, participants were presented the target image for 30 s and instructed to encode as much detail as possible, because they would be asked some questions about the image later on. During phase 2, they brought the target image to mind as vividly as

possible and rated its mental representation by filling out a 100 mm visual analogue scale (VAS: 0 = *not vivid at all*; 100 = *very vivid*). During phase 3, they recalled the target image for 24 s while watching a black screen (control condition) or making EM by following a white dot (Ø = 1 cm) that moved horizontally across a black screen at 1 cycle per second (experimental condition). In the experimental condition, and only during this phase, the distance to the monitor was reduced to 30 cm. After the recall period participants were instructed to focus on something else for 10 s. During phase 4, they retrieved the target image and rated its vividness for a second time. Phase 5 involved a reaction time task. Participants were presented the three test images in random order. This procedure was repeated once, providing a total of six presentations. Image duration was 8 s and inter-trial-intervals ranged between 15-25 s (M = 20 s). Participants were told that they would see several images of faces and that it was their job to indicate as quickly and correctly as possibly whether or not each face was identical to the image they had seen and recalled in the previous phases. Response options were pressing a green key ('same') or a red key ('different') on a keyboard. Participants were told that they could press only once and that each image would remain on the monitor for exactly 8 s. At the end of the experiment, they were debriefed, asked about their knowledge on EMDR, and compensated for their time.

Results

Memory vividness

The left graph of Figure 2 shows the memory vividness ratings of Experiment I. The drop in scores over time was larger in the experimental condition (pre-test: M = 78.00, SD = 11.19; post-test: M = 63.38, SD = 14.24) than in the control condition (pre-test: M = 79.46, SD = 11.37; post-test: M = 71.69, SD = 13.27). Repeated measures ANOVA showed a significant Time x Condition interaction, $F(1, 24) = 7.71$, $p = .010$, $\eta p^2 = .243$, that was not qualified by a Time x Condition x Image set (A vs. B) interaction, $F<1$. Thus, irrespective of the type of target image (A or B) that was retrieved, recall plus EM, relative to recall only, caused a larger decrease in self-reported memory vividness.

Discrimination accuracy

Accuracy in discriminating test images from target images was defined as the ratio of 'different' responses to the total number of responses. First, we examined the data of the two sets of images together. Test image 1, which was perceptually closest to the target image, was classified as different in 41% of trials in the control condition and in 40% of trials in the experimental condition. A chi-square test, comparing the proportion of same/different

responses between the two conditions, showed that this difference was not significant, $\chi^2(1, N = 99) = .03$, $p = .872$. For test image 2, accuracy was higher and still comparable between the two conditions: 72% in the control condition and 71% in the experimental condition, $\chi^2(1, N = 99) = .004$, $p = .950$. Test image 3 was accurately discriminated in 96% (control condition) and 90% (experimental condition) of trials. Again, the difference between the conditions was not significant, $\chi^2(1, N = 101) = 1.58$, $p = .209$. Analyzing the data for image sets A and B separately yielded very similar results: both sets showed the expected generalization gradient, and there were no significant differences between conditions, largest $\chi^2(1, N = 49) = 1.68$, $p = .195$. In sum, accuracy in discriminating test images from the target image increased as a function of perceptual dissimilarity between the two image types, and was not affected by the EM procedure.

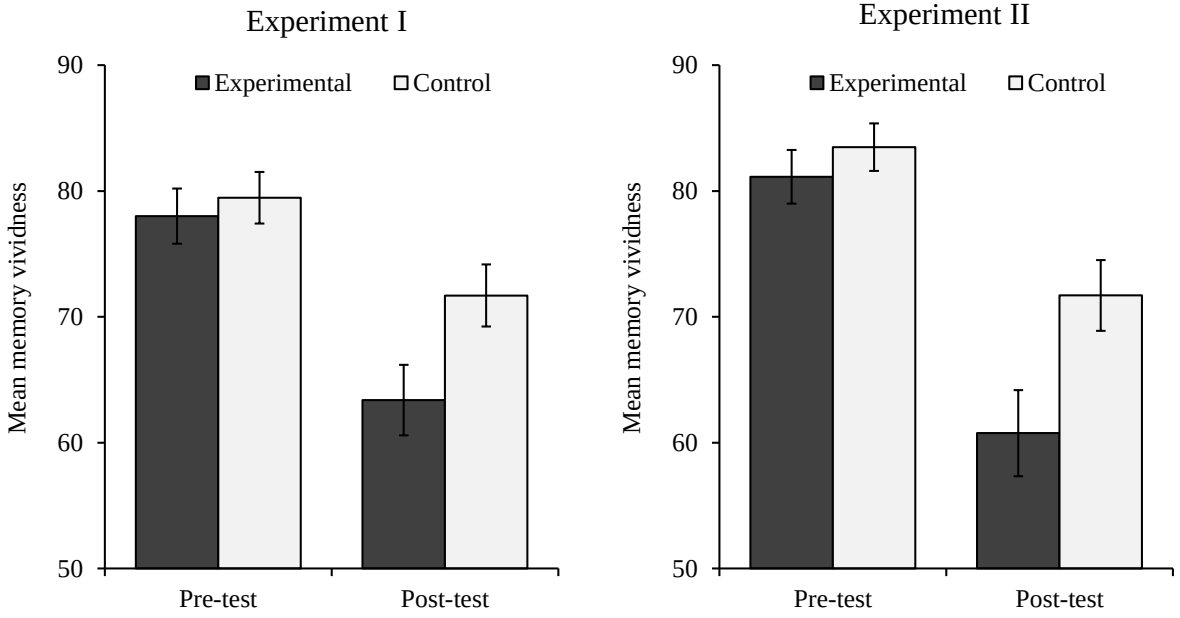

Fig. 2 Mean memory vividness before and after recall with eye movements (Experimental) or without (Control). The graph on the left shows the data of Experiment I. The graph on the right shows the data of Experiment II. Error bars reflect standard errors of the mean.

Discrimination speed

Table 1 shows the raw RT data. Standardized RTs (*z*-scores) were calculated to account for interindividual differences in mean values (see Figure 3). These standardized data were analyzed using a 2(Condition) x 2(Image set) x 3(Test image) repeated measures ANOVA. There was a main effect of Condition, $F(1, 24) = 7.44$, $p = .012$, $\eta p^2 = .237$, with overall faster responses in the control condition than in the experimental condition. This finding supports the hypothesis that a reduction in the vividness of a stimulus representation impedes the process of

discriminating this stimulus from other stimuli. There was also a main effect of Image set, $F(1, 24) = 41.54$, $p < .001$, $\eta p^2 = .634$, with overall faster responses to test images from set A than set B. Finally, there was a main effect of Test image, $F(2, 23) = 11.65$, $p < .001$, $\eta p^2 = 503$. Follow-up analyses revealed that participants classified test image 3 faster compared to test images 1 and 2. The latter result corresponds with the finding that nearly all participants correctly classified test image 3 as dissimilar from the target image. The ANOVA did not yield any two-way or three-way interactions.

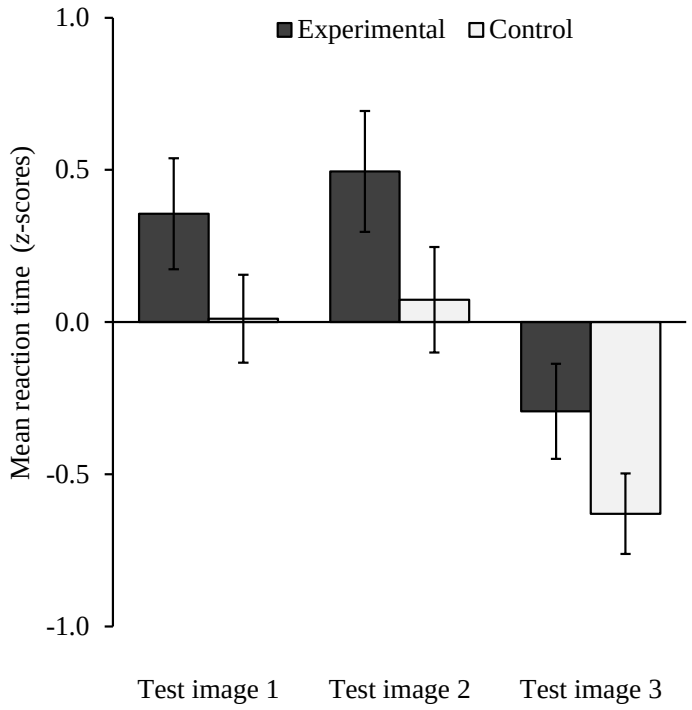

Fig. 3 Mean standardized reaction times. Error bars reflect standard errors of the mean.

Table 1. *Mean reaction times (SDs) in milliseconds for image sets A and B*

	Test image 1	**Test image 2**	**Test image 3**
Experimental (set A)	2418.69 (1471.15)	3103.19 (1781.63)	2183.15 (1550.09)
Control (set A)	2519.12 (1480.66)	2346.73 (1596.72)	1914.81 (1244.85)
Experimental (set B)	3372.50 (1814.11)	3301.31 (1929.41)	2790.77 (1694.03)
Control (set B)	2884.08 (1464.07)	3449.35 (1889.33)	2362.42 (1330.58)

Discussion Experiment I and introduction to Experiment II

Experiment I demonstrated that making EM during recall of an image (1) reduces memory vividness when the image is recalled again later, (2) slows down the process of comparing the stimulus with subsequently perceived stimuli, and (3) does not affect discrimination accuracy. This pattern of results matches the findings of van den Hout et al. (2013) who demonstrated that the blurring of an earlier encoded image increases reaction speed during a task that requests participants to indicate whether or not fragments are part of the original image. The authors suggested that the slowing down in decision-RT may have represented a shift in the speed-accuracy trade-off from speed to accuracy, but also did not find an effect on decision accuracy.

The aim of Experiment II was to test whether the dual task increases generalization of conditional fear. The rationale was as follows. Confrontation with a stimulus that reminds one of a danger cue, thus signaling potential threat, will motivate the individual to fully activate, examine, and compare the memory representation of the danger cue with the stimulus at hand. Based on Experiment I and the results by van den Hout et al. (2013), it can be expected that when the mental representation of the danger cue is blurred, this process will take more time. And, the longer it takes to classify an ambiguous stimulus as 'safe', the more likely it will be that people *react* as if it were 'dangerous' (i.e., adopt a better-safe-than-sorry strategy). In Experiment II we tested this hypothesis. First, participants underwent a fear conditioning procedure, in which a picture of a neutral male face (CS) was followed by an electrical shock (UCS), and participants rated shock expectancy. Next, they recalled the image with EM (experimental group) or without (control group), and rated memory vividness. Finally, during a test phase, they saw a novel face that perceptually resembled the original face (generalization stimulus; GS), and rated shock expectancy again. We predicted decreases in memory vividness and stronger generalization of fear from the CS to the GS in the experimental condition. Fear was operationalized as self-reported shock-expectancy, elevation of electrodermal responding, and potentiation of the startle eye blink reflex.

Method

Participants

Fifty-four (mostly) undergraduates participated in exchange for remuneration or course credits. The procedure was approved by the local ethical committee. Exclusion criteria were similar to Experiment I. The first participant was excluded because startle probes were not properly presented and only part of the data was saved. Another participant was excluded

because of linking the experimental procedure to the EM employed in EMDR. The final sample comprised 52 participants (age: *M* = 21.67 years, *SD* = 7.03; 13 males). By order of appearance, they were allocated to an experimental (*n* = 25) or control group (*n* = 27).

Stimulus material

The standardized RT data of Experiment I were explored to see which test image was associated with the largest impact of the EM procedure. Discrimination speed for test image 2 from set A showed the largest difference between conditions (experimental: *M* = .42, *SD* = .32; control: *M* = -.41, *SD* = .17; the raw data indicated the same, see Table 1). An independent samples *t*-test comparing the two *z*-scores showed that this difference was significant, *t*(24) = 2.32, *p* = .032, *d* = .909. Therefore, in Experiment II, the target image and test image 2 from set A were used as CS and GS, respectively. A 2-ms electrocutaneous stimulus was used as UCS. Shocks were generated via a constant current stimulator (Digitimer DS7A) and delivered through a pair of AgCl electrodes (Coulbourn, 8-mm V91-01) that were filled with K-Y Jelly (Johnson & Johnson) and attached to the wrist of the dominant hand. Shock intensity was individually set (range: 2-64 mA; *M* = 23.46 mA, *SD* = 15.11; see *Procedure*).

Questionnaire

To control for interindividual differences in baseline anxiety levels, which are known to affect fear learning (Grillon et al., 2006; Lissek et al., 2005), the State Trait Anxiety Inventory was administered (STAI-DY; Spielberger, Gorsuch, & Lushene, 1970). State anxiety and trait anxiety were both measured with 20 items that were scored on a 1-4-point scale (1 = *not at all*, 4 = *severely*).

Skin conductance responses

Skin conductance responses (SCRs) were recorded by attaching a pair of AgCl electrodes (Coulbourn, 8-mm V91-01) to the hypothenar palm of the non-preferred hand. The electrodes transmitted a constant current of .5 V that was registered by an isolated skin conductance coupler (Coulbourn, V71-23). Data were sampled at 10 Hz.

Startle EMG

Startle blink electromyographic (EMG) responses were elicited by binaural acoustic presentation of 50-ms white noise probes at 102 dB. EMG activity was recorded by placing two AgCl electrodes (Coulbourn, 4-mm V91-02) over the orbicularis oculi region of the right eye, and a third one (Coulbourn, 8-mm V91-01) approximately 3-4 cm superior to the upper borders of the inner brows (Fridlund and Cacioppo, 1986). These sites were first cleaned with exfoliating cream (Louis Widmer) and the electrodes were filled with gel (Coulbourn, X11-71 microlyte electrode gel). The raw EMG signal was amplified by an isolated bioamplifier

(Coulbourn, V75-04) with high pass (13 Hz) and low pass (1 KHz) filters. The signal was then rectified and smoothed by a multifunction integrator (Coulbourn, V76-24) with a time constant of 20 ms. Data were sampled at 1 KHz.

Procedure

Participants were seated in a dimly lighted room about 42 cm in front of a 17-inch monitor (1440x900 pixels). They were told that pictures of faces would appear on the monitor with breaks in between, that they would occasionally receive electrical shocks, and that it was their job to learn to predict shock occurrence. After providing written informed consent, they filled out the STAI. Next, electrodes were attached and shock intensity was individually set via a work up procedure (cf. Fonteyne, Vervliet, Hermans, Baeyens, & Vansteenwegen, 2009). The experimenter presented a series of shocks. The intensity started with 1 mA and was increased stepwise. Participants rated each shock on a scale ranging from 1 to 10 (0 = *I don't feel anything*; 1 = *I feel something, but this is not painful, it is just a sensation*; 2 = *it starts to feel painful, but it is still a very small pain*; 10 = *this is the maximum tolerable pain for me in this experiment*). Participants were urged to notify the experimenter when their maximum level was reached or when they wanted the intensity to be turned down. They were further asked whether they would be able to tolerate occasional shocks at their chosen intensity. Lastly, headphones were put on. The experiment was programmed using Affect 4.0 software (Spruyt, Clarysse, Vansteenwegen, Baeyens, & Hermans, 2010) and comprised five consecutive phases: a practice, acquisition, intervention, test, and extinction phase.

Practice phase. There were 4 baseline trials and 4 image trials, which alternated, starting with a baseline trial. During each trial, a shock expectancy VAS (0 = *certainly no shock*; 100 = *certainly a shock*) appeared on screen that disappeared after 6 s or 1 s after response registration. The CS was presented only during image trials for 6 s. Participants were told that this phase served to practice filling out the VAS and that no shocks would be delivered yet. They were encouraged to indicate shock expectancy as quickly as possible each time the VAS appeared on screen. Startle probes were presented 4 or 5 s after each VAS onset. This phase thus comprised a total of 8 startle probe presentations that served as habituation trials. Inter-probe-intervals ranged between 17-25 s (21 s on average).

Acquisition phase. There were 12 baseline trials and 12 image trials that were similar to the practice trials. During each trial the VAS appeared on screen and only during image trials the CS appeared on screen. During this phase, nine randomly chosen image trials were immediately followed by shock (75% reinforcement schedule). Baseline trials were included to allow for examination of differential learning. Participants were told that they would

occasionally receive electrical shocks during this phase and that it was their job to indicate shock expectancy each time the VAS appeared on screen.

Intervention phase. Shock electrodes were disconnected and participants were told that no shocks would be presented during this phase. Subsequently, they were exposed to the CS for 10 s and received instructions to encode as much detail as possible because they would be asked some questions about the image later on. Following the same procedure as in Experiment I, participants then rated the vividness of the CS memory (pre-test), recalled it for 24 s with EM (experimental group) or without EM (control group), and finally provided a second vividness rating (post-test).

Test phase. Shock electrodes were connected again, but no shocks were given during this phase. First, to habituate the startle response, 4 probes were presented with 17-25 s (M = 21 s) intervals in between while participants looked at a blank screen. Subsequently, participants were presented three baseline trials and three image trials, which alternated and started with a baseline trial. During the image trials, the GS appeared on the screen for 6 s. Participants were told that this phase would start with a few startle probe trials, and that then the same face as in the previous phases and other, somewhat different, faces would be shown. They were further told that the original face would sometimes be followed by shock, but that the other faces would never be followed by shock.

Extinction phase. Without further instructions, the extinction phase started. This phase was similar to the acquisition phase, except that no shocks were delivered, and it served to invalidate the CS-UCS contingency. After the experiment, participants were debriefed and compensated for their time.

Data preparation

PSPHA software was used to analyze the physiological data (De Clercq, Verschuere, De Vlieger, & Crombez, 2006). SCRs were calculated by subtracting the baseline value (average SC level for the 2 s preceding stimulus onset) from the peak value that was recorded in the period of 0-4 s following VAS onset (Pineles, Orr, & Orr, 2009; note that half of the startle probes occurred 4 s after VAS onset). Minimum response amplitude was set at .02 μS. All other responses were scored as 0 and left in the analyses (Pineless et al., 2009). The data were T-transformed to adjust for interindividual differences ($50 + 10*[(\text{raw score} - M) / SD]$; Dawson, Schell, & Filion, 2007; Haesen & Vervliet, 2014). Finally, block averages were calculated per three trials, resulting in four acquisition blocks and one test block.

Startle amplitudes were calculated by subtracting the baseline value (average EMG level 0-20 ms after probe onset) from the peak value that was identified in the period of 21-200 ms

after probe onset. Each response was visually inspected. Trials that showed substantial noise during the baseline period (11.1%) or did not show a response to the probe (5.8%) were excluded from statistical analyses. To account for interindividual differences, all data were *T*-transformed (Blumenthal et al., 2005). Finally, three-trial block averages were calculated.

Results

Groups did not differ in state anxiety (experimental: $M = 33.28$, $SD = 6.81$; control: $M = 31.67$, $SD = 9.75$), $t < 1$, trait anxiety (experimental: $M = 37.12$, $SD = 7.08$; control: $M = 34.30$, $SD = 7.72$), $t(50) = 1.37$, $p = .176$, age, $t(50) = 1.39$, $p = .172$, gender ratio, $\chi^2(1, N = 52) = .642$, $p = .423$, or shock intensity (experimental: $M = 22.92$, $SD = 12.76$; control: $M = 23.96$, $SD = 17.23$), $t < 1$, indicating successful randomization.

Memory vividness

Mean memory vividness ratings are shown in the right graph of Figure 2. Repeated measures ANOVA showed a significant Time x Group interaction, $F(1, 50) = 5.55$, $p = .022$, $\eta p^2 = .100$. As expected, reductions over time were larger in the experimental condition (pre-test: $M = 81.12$, $SD = 10.67$; post-test: $M = 60.76$, $SD = 17.14$) than in the control condition (pre-test: $M = 83.48$, $SD = 9.81$; post-test: $M = 71.70$, $SD = 14.61$).

Shock expectancy

Differential learning was investigated by a 2(Group) x 2(Stimulus: CS vs. baseline) x 12(Trial) repeated measures ANOVA. There were main effects of Stimulus, $F(1, 45) = 313.11$, $p < .001$, $\eta p^2 = .874$, and Trial, $F(11, 495) = 2.16$, $p = .040$, $\eta p^2 = .046$, that were qualified by a Stimulus x Trial interaction, $F(11, 495) = 14.10$, $p < .001$, $\eta p^2 = .239$, meaning that differential shock expectancy increased over time. No other effects were found, Fs < 1.

The strength of conditional and generalized shock expectancy was defined as expectancy during image presentation minus expectancy during baseline. The data in Figure 4 suggest that at the end of the acquisition phase, conditional shock expectancy was stronger in the control group compared to the experimental group, which was an unanticipated finding. This was confirmed by a *t*-test that compared the two groups on the average difference score of the last three acquisition trials (experimental group: $M = 59.70$, $SD = 33.49$; control group: $M = 76.71$, $SD = 17.50$), $t(50) = 2.32$, $p = .029$, $d = .637$. Because the intensity of generalized responding is directly related to the intensity of conditional responding, we corrected for this difference before analyzing the test phase data. For each participant, the difference scores of the last three acquisition trials and the three test trials were used to compute *z*-scores. These *z*-scores allow

us to interpret the strength of (generalized) responding during the test phase as *relative to* the strength of (conditional) responding at the end of the acquisition phase. A 2(Group) x 3(Trial: test trials 1-3) repeated measures ANOVA on these standardized data only showed a main effect of Group, $F(1, 47) = 4.70$, $p = .035$, $\eta p^2 = .091$, other Fs < 1. This finding indicates that generalization of shock expectancy was stronger in the experimental group compared to the control group, and supports the hypothesis.

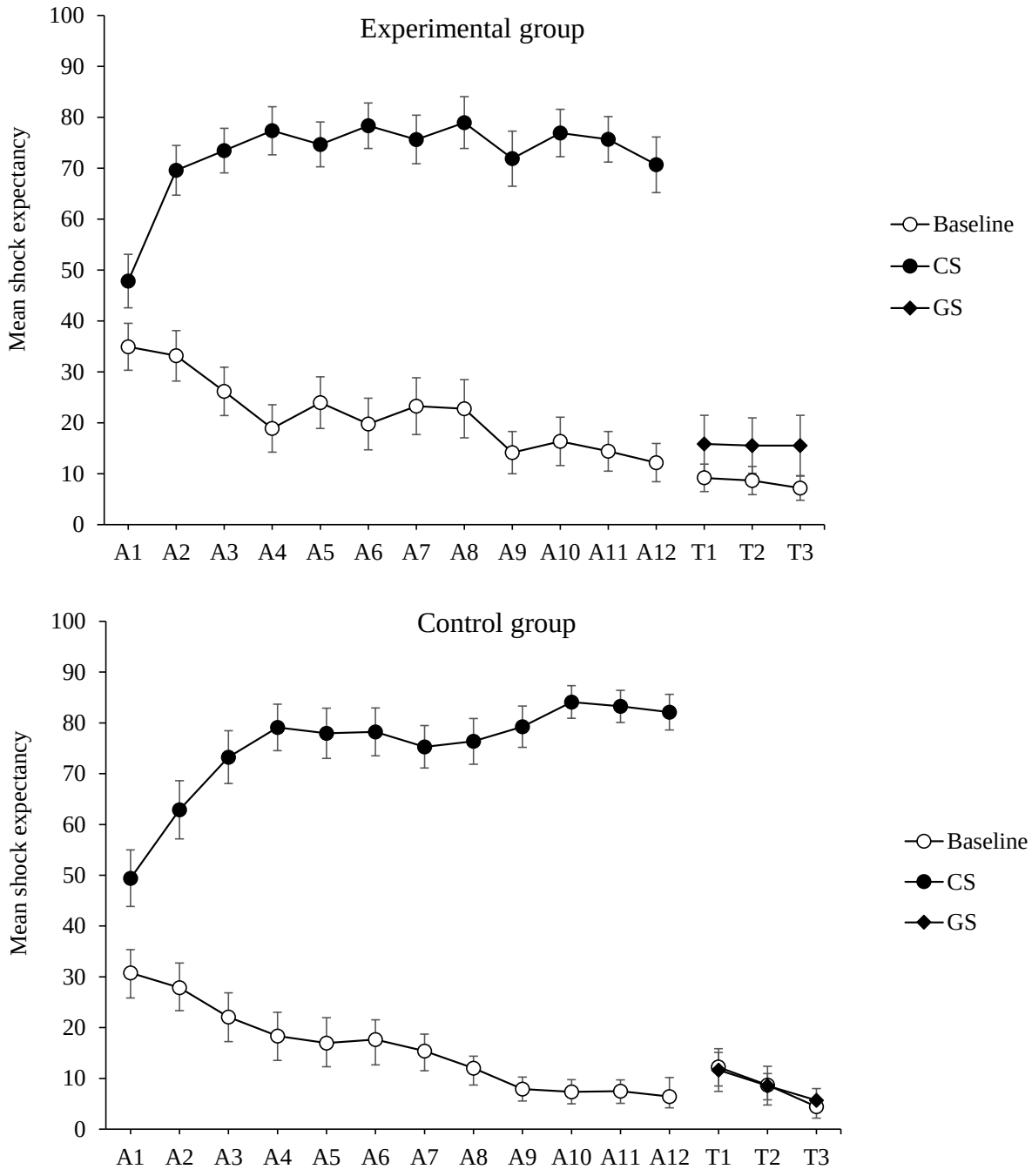

Fig. 4 Mean UCS expectancy ratings during the acquisition (A1-12) and test phase (T1-3). Error bars reflect standard errors of the mean.

Skin conductance

Four participants did not show any response at all during the experiment and were excluded from analyses. First, differential learning was examined. A 2(Group) x 2(Stimulus) x 4(Block) repeated measures ANOVA on the acquisition trials showed main effects of Group, $F(1, 46) = 5.40$, $p = .025$, $\eta p^2 = .105$, Stimulus, $F(1, 46) = 59.88$, $p < .001$, $\eta p^2 = .566$, and Block, $F(3, 138) = 40.78$, $p < .001$, $\eta p^2 = .470$. On average, SCR was higher in the control group, was higher to CS trials than during baseline trials, which indicates successful learning, and habituated over time. No other effects were found, largest $F = 1.08$, $p = .358$.

The strength of conditional and generalized responding was defined as the difference between SCR during image presentation and SCR during baseline. First, a *t*-test showed that the strength of conditional responding during the last acquisition block was comparable between the two groups, $t < 1$. Next, a *t*-test comparing the intensity of generalized responding during the test block did not reveal a between-group difference, $t < 1$. Note, however, that Figure 5 shows a sudden and substantial increase in SCR during the first baseline trial. This likely reflects an orienting response: the test phase started with four noise alone trials and the very first presentation of the shock expectancy VAS occurred during the first baseline trial. Importantly, this effect hampers the detection of between-group differences when examining the average of the three trials. Figure 5 further suggests that during test trial 2 generalized responding was stronger in the experimental group compared to the control group. This was confirmed by a *t*-test, $t(46) = 2.17$, $p = .035$, $d = .626$, providing further support for the hypothesis. This effect ceased at test trial 3, $t < 1$.

Startle EMG

Differential learning was examined using a 2(Group) x 2(Stimulus) x 4(Block) repeated measures ANOVA. There were main effects of Stimulus, $F(1, 47) = 10.58$, $p = .002$, $\eta p^2 = .184$, and Block, $F(3, 141) = 52.58$, $p < .001$, $\eta p^2 = .528$, but not of Group, $F(1, 47) = 3.00$, $p = .090$, $\eta p^2 = .060$. Startle EMG was higher to CS trials than to baseline trials, indicating successful learning, and habituated over time. No other effects were found, largest $F = 1.17$, $p = .325$.

Next, a *t*-test showed that the strength of differential responding (i.e., mean amplitude during image presentation minus mean amplitude during baseline) was comparable between groups during the last acquisition block, $t < 1$. Contrary to expectations, the intensity of differential responding was still similar between the groups during the test block, $t < 1$. Comparable to the SCR data, Figure 6 suggests that the startle amplitude during the first baseline trial was affected by an orienting response, and that groups differed in generalized responding during the second test trial. The latter observation was, however, not confirmed,

t(34) = 1.53, *p* = .137. Thus, the startle EMG data did not provide further support for the hypothesis.

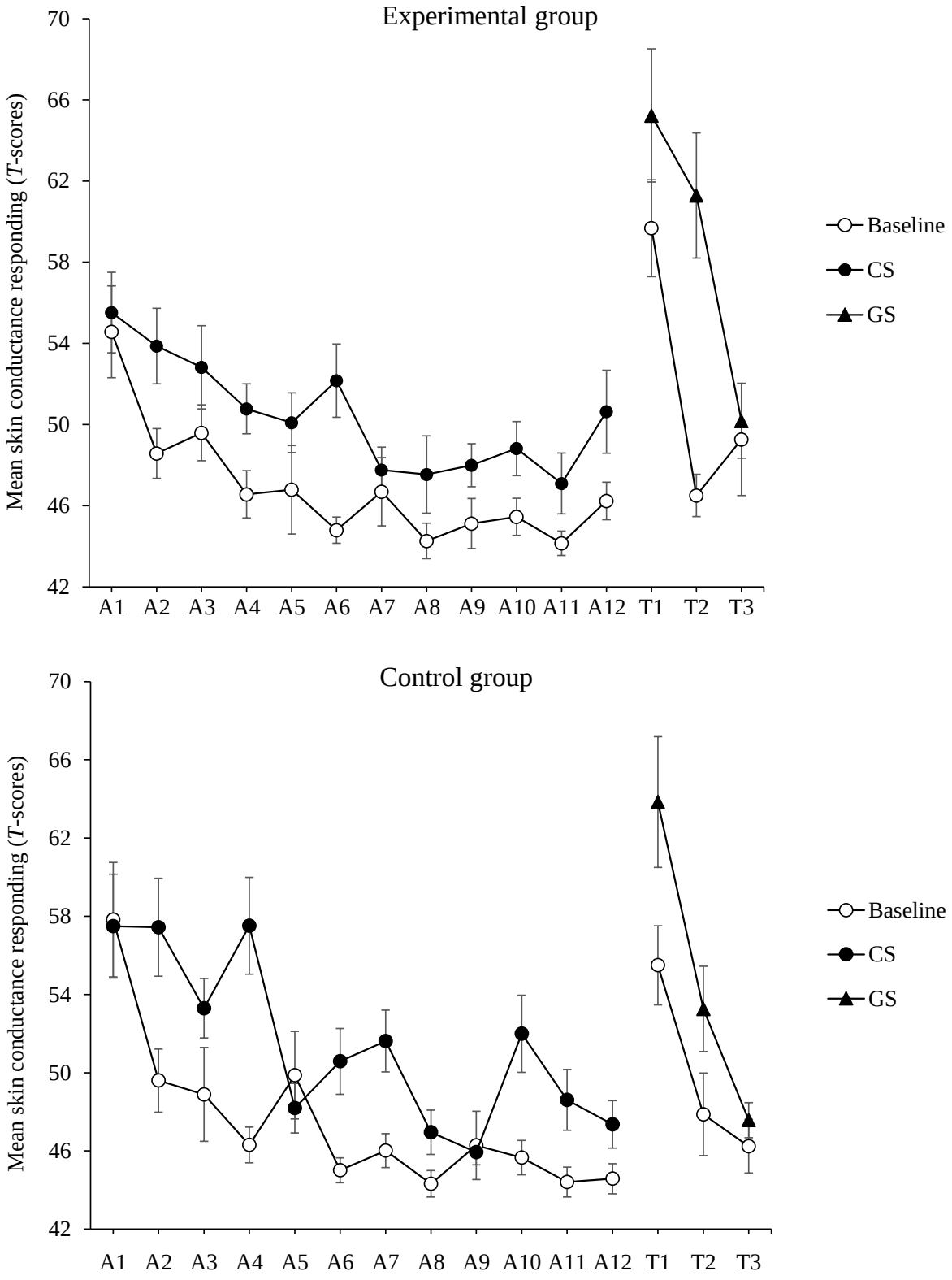

Fig. 5 Mean standardized skin conductance responses during the acquisition (A1-12) and test phase (T1-3). Error bars reflect standard errors of the mean.

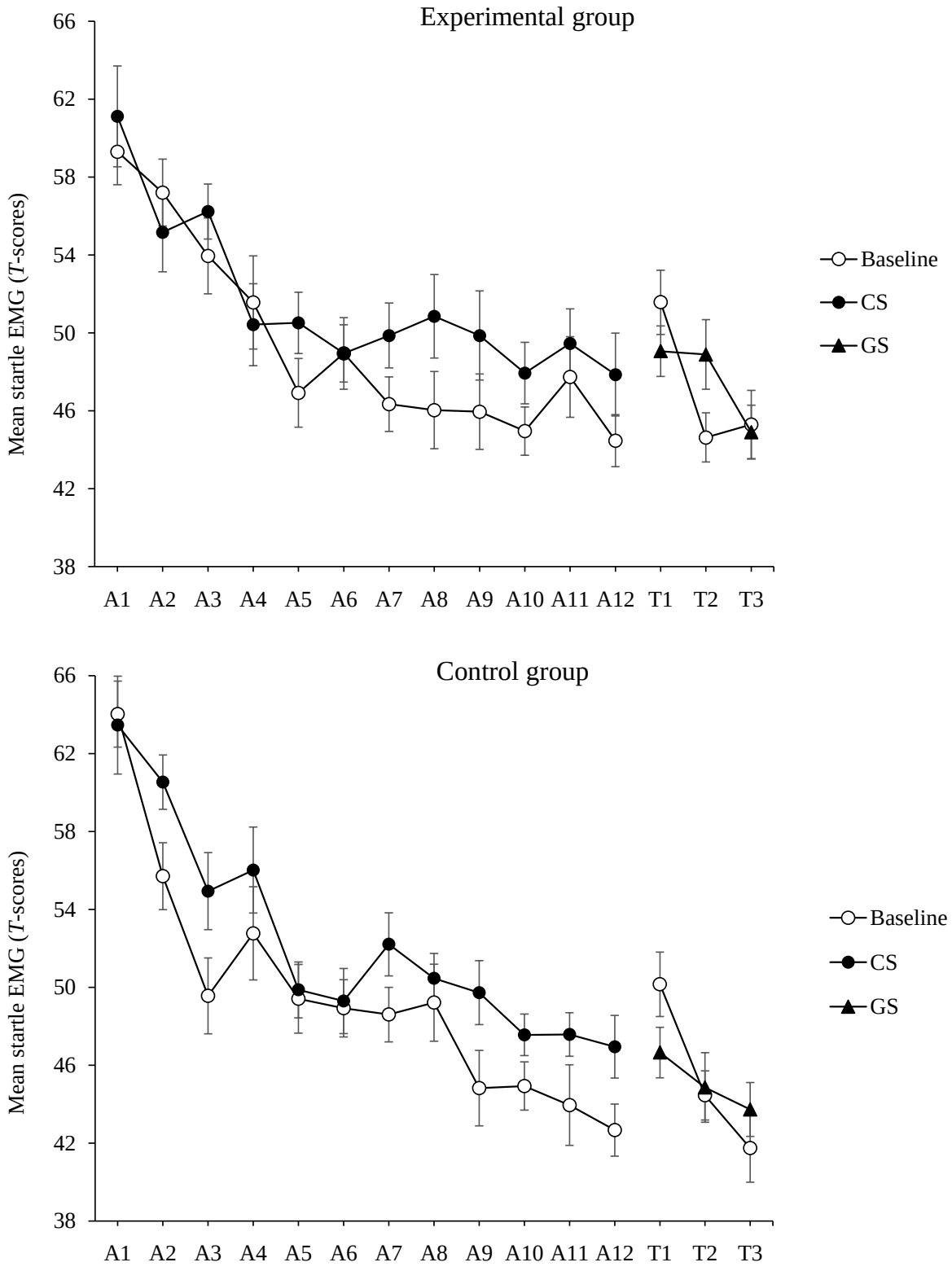

Fig. 6 Mean standardized startle EMG during the acquisition (A1-12) and test phase (T1-3). Error bars reflect standard errors of the mean.

Discussion

We set out to test whether the blurring of a danger cue representation affects discriminating it from other stimuli and thus facilitates fear generalization. Consistent with the hypotheses, EM during stimulus recall reduced stimulus discrimination speed (Experiment I), and increased fear generalization, as evidenced by shock-expectancy and elevated SCR (Experiment II). The latter finding was, however, not corroborated by the startle EMG data.

Previous research has found that EM during recall reduces self-reported memory vividness and emotionality (as reviewed by van den Hout & Engelhard, 2012). The results of Experiment I extend these findings in three ways. First, the dual task not only caused a drop in self-rated memory vividness, but also affected reaction speed in a discrimination task. Because it is unlikely that these reaction time data were affected by participants' expectations, this result precludes a demand characteristics account of the widely reported EM effects (cf. Engelhard, van Uijen, & van den Hout, 2010; van den Hout et al., 2013). Second, the reduction in memory accessibility provides a first conceptual replication of van den Hout et al. (2013). This finding supports the view that EM result in a (permanent) change of memory characteristics (e.g., Engelhard, van den Hout, Janssen, & van der Beek, 2010; van den Hout et al., 2010; Maxfield et al., 2008) Alternatively, it has been proposed that EM primarily alters one's affective evaluation (i.e., appraisal) of the memory (e.g., Gunter & Bodner, 2008; Kavanagh et al., 2001; van den Hout et al., 2001). Note, however, that these accounts may coexist. Third, relatively simple, non-idiosyncratic stimulus material was used and the dual task appeared successful in reducing the vividness of its mental representation. Two earlier investigations that requested participants to recall de novo images reported similar results (Andrade, Kavanagh, & Baddeley, 1997; van den Hout et al., 2013), but two other studies failed to provide corroborating evidence (Leer, Engelhard, Dibbets, & van den Hout, 2013; van Schie, Engelhard, & van den Hout, 2015). Methodological differences may account for this variance in findings. Leer et al. (2013) did not include an isolated encoding phase before the dual task and pre-scores were relatively low, leaving little room for improvement. Van Schie and colleagues (2015) tested the effects of *cued* recall, rather than instructed recall, and their null results may be due to suboptimal memory retrieval. Together, these findings suggest that non-idiosyncratic stimulus material can be included in the dual task paradigm, but that there are boundary conditions. This is especially relevant for future examinations of the EM effects that wish to employ a (fear) conditioning paradigm and/or require the presentation of simple and standardized stimuli.

The main finding of this investigation was that EM during danger cue recall, relative to mere recall, increases the intensity of generalized fearful responding. Although stimulus discrimination was not directly assessed in Experiment II, the drop in memory vividness ratings and the results of Experiment I strongly suggest that this effect was mediated by the ability to discriminate the GS from the CS. Reduced accessibility to the original array of stimulus attributes thus likely explains why participants showed stronger fear responses to perceptually similar stimuli. This corresponds with earlier findings that forgetting of stimulus attributes increases generalized responding (e.g., Anderson & Riccio, 2005). Indirectly, the current results suggest that *increasing* the vividness and detail of a CS representation will *reduce* fear generalization. In support of this idea, previous animal studies have documented that exposure to the training context (CS) prior to context-shock conditioning, which arguably strengthens the context's representation, reduced the generalization of conditional freezing to a (novel) test context (Biedenkapp & Rudy, 2007). Also, 'pre-test cuing treatment', i.e. exposure to the training context just before entering a (novel) test context, which likely reinstates the training context memory, has been shown to reduce generalization of conditional freezing (Zhou & Riccio, 1994). Future research can elucidate whether reinstatement of a CS representation in humans, e.g. via guided imagination, has similar effects.

An unexpected finding was that the effect on generalized responding found in the shock-expectancy and SCR-data was not present in the startle EMG data. Several explanations may account for this discrepancy. First, some scholars have identified shock-expectancy ratings and skin conductance reactivity primarily as measures of contingency learning, and startle blink responses as a more specific/affective measure of fear learning (e.g., Soeter & Kindt, 2010). Accordingly, it may be argued that the current results reflect an increase in *expectancy* generalization rather than fear generalization. However, recent data suggest that skin conductance reactivity not merely tracks learning about the strength of the CS-UCS association, but also about the estimated intensity of the UCS, which arguably reflects *threat* learning (Haesen & Vervliet, 2014). Alternatively, the effect may be short-lived. On average, expectancy ratings during GS trials were provided 4066 ms after image onset, thus reflecting UCS expectancy during the first few seconds of image presentation. SCRs typically rise 1-3 s following stimulus onset (Dawson et al., 2007). In contrast, startle probes were delivered 4 or 5 s after stimulus onset and it is possible that participants more successfully discriminate the GS from the CS after such a longer time interval. Note, however, that shock-expectancy was still increased during the second and third test trial, which is at odds with this hypothesis. Finally, statistical power may have been too low to detect between-group differences in startle

blink amplitudes. Comparing the SCR data in Figure 5 with the EMG data in Figure 6 shows that responding during test trials was quite similar for the two measures. Specifically, both graphs show that differential responding during the second test trial was stronger in the experimental group than in the control group. However, the between-group analysis on startle blink responding during this trial was carried out with n = 36 (18 per group), because noise-contaminated and non-response trials were excluded. In contrast, 48 participants (24 per group) provided useful SCR data. Still, exploratory analyses revealed that the relative increase in generalized responding during the second test trial was associated with a medium effect size for both measures (SCR: d = .626; startle EMG: d = .508), which further points towards a lack of statistical power.

Given that future investigations replicate the current findings, also in clinical samples, there may be clinical implications. Defining aspects of PTSD include persistent re-experiencing of the traumatic event (e.g., intense distress after exposure to trauma reminders) and avoidance of threat-related thoughts, feelings, or external reminders (e.g., people, places, situations; APA, 2013). In this paper we argue that the blurring of danger cue representations (i.e., CSs) may extend the range of stimuli that can trigger such symptoms. We hypothesized and found that EM during recall of 'trauma' reminders intensifies the generalization of fearful responding to stimuli other than the original CS. EMDR therapists should therefore be careful when it comes to determining what memory features to include in the dual task. Specifically, trauma hotspots reflect the worst moments of the traumatic event (Holmes et al., 2005) and it seems conducive to decrease the vividness and accessibility of such memory features. The blurring of danger cue representations (i.e., cues and contexts that signal the traumatic event), however, may have harmful effects and the current findings discourage to include such memory features in the dual task.

Two issues related to this tentative recommendation deserve further attention. First, we are not aware of case reports in which EMDR treatment results in stronger fear generalization. Instead, our hypothesis was theory-driven. Note, however, that such an approach can provide valuable insights. For example, over the years, many therapists have replaced the EM by other forms of bilateral stimulation, such as listening to alternating beeps (Maxfield, 2008). However, following the advances in theory, it was postulated by van den Hout, Engelhard and colleagues (2011) that beeps may be less effective, because they hardly use working memory resources. This hypothesis was tested and confirmed, first in a student sample (van den Hout et al., 2011), and subsequently in a clinical sample (i.e., patients with PTSD; van den Hout et al., 2012). Accordingly, Dutch EMDR trainers have adjusted the EMDR guidelines for treatment and

training, and now encourage therapists to use EM rather than beeps (Beer et al., 2011). Another example relates to the processing of *positive* material and the use of EM as a 'catalyst'. During the preparation phase of EMDR, patients are often taught a stress management technique in which they are directed to imagine a place where they feel safe or calm. Importantly, the EMDR protocol instructs to elicit horizontal EM during this procedure (Shapiro, 2001). In line with working memory research, however, non-clinical research has shown that EM during recall makes such images *less vivid* and *less positive* (Engelhard, van Uijen, et al., 2010; for overview see Hornsveld, de Jongh, & ten Broeke, 2012). Therefore, the use of EM in this procedure has been seriously questioned, and is now discouraged by Dutch EMDR trainers (Beer et al., 2011). In a similar vein, the current results may ultimately change EMDR practice. However, as noticed earlier, these findings await (clinical) replication.

Another issue concerns the theoretical distinction between danger cues and hotspots, which may not be obvious in clinical cases. For example, the act of looking the perpetrator in the eyes, rather than the assault itself, may well become the image that causes peak levels of emotional distress. In this example, the danger cue *is* the trauma hotspot. Theoretically, this image does not function as a conditional stimulus that can activate the trauma memory, but represents (part of) the trauma memory itself. Accordingly, it can be expected that the blurring of such a memory does not lead to an increase in fear generalization.

A limitation of this study is that we did not include a measure of behavioral avoidance. Alongside subjective experience and physiology, behavior is core part of the emotional (fear) response (e.g., Mauss & Robinson, 2009). Because avoidance behavior is one of the diagnostic criteria for many anxiety-related disorders (including PTSD), and captures a unique component of the fear response, its measurement is worthwhile (APA, 2013; Beckers, Krypotos, Boddez, Effting, & Kindt, 2013). Future research that aims to replicate and extend the current findings may therefore include, for example, an approach-avoidance task (AAT; e.g., Krypotos, Effting, Arnoudova, Kindt, & Beckers, 2014) or the possibility to avoid potential UCS-occurrence (e.g., Lommen, Engelhard, & van den Hout, 2010). A strength of the current study is the inclusion of both subjective and physiological measures of fear, which provides some cross-validation and precludes a demand characteristics account of the findings.

In sum, the current experiments demonstrate that EM during recall of a danger cue representation causes a reduction in memory vividness, impedes the process of discriminating the danger cue from other stimuli, and intensifies generalized fearful responding. Because the blurring of memory features that are associated with the trauma hotspot is standard EMDR practice (de Jongh & ten Broeke, 2009), and these may include conditional stimuli, the current

results suggest that caution is needed. Provided that future investigations replicate the current findings, also in clinical samples, clinicians may be advised to refrain from having their clients recall conditional cues and contextual information that signal the traumatic event and make EM at the same time.

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Chapter 7

Discussion

This thesis set out to explore whether the eye movements during recall of aversive memory (1) can be used as a method of UCS deflation and (2) provide a valuable technique in the attenuation of fear renewal. In **chapter 2** we replicated the widely reported effects that the dual task reduces vividness and emotionality of autobiographical memory. We further revealed that, under the right conditions, the effects are maintained at a 24 h follow-up test. In **chapter 3** we demonstrated that eye movements during recall of a UCS representation decreases its unpleasantness and reduces conditional fear, which suggests that the dual task resembles a UCS deflation process. The experiment reported in **chapter 4** did not demonstrate that the dual task affects UCS memory emotionality/vividness, but did show a reduction in context-driven return of fear after extinction. **Chapter 5** further elucidated the underlying mechanism of this effect: we showed that UCS deflation exerts its fear-diminishing effects through reductions in the anticipated cost of the UCS, and leaves the CS-UCS association untouched. Finally, in **chapter 6** we revealed a possible risk of the dual task, i.e. that the blurring of a danger cue representation increases generalized fearful responding, and accordingly conveyed a preliminary recommendation for therapists.

This chapter presents the broader theoretical and clinical implications of these findings. As will be explained below, the current studies not only shed light on a novel route to fear reduction, but also provide new insights in how eye movements in EMDR work. These implications will be discussed as well.

Blurring aversive memory: A novel route to fear reduction?

First and foremost, the findings contribute to our understanding of whether and how the eye movement intervention can diminish (return of) conditional fear. This was the primary aim of the research. Below, we will reflect on what we have learned with respect to the possible use of the intervention as a method of UCS deflation, and its potential to reduce the return of fear after extinction. After that, we will discuss the limitations of the studies that concerned these two questions and give suggestions for future research. Finally, we will present the potential clinical implications of this work.

Can eye movements during aversive ideation be used as a method of UCS deflation?

UCS revaluation processes are defined as methods that change a person's evaluation of the UCS (Davey, 1997). Previous research has consistently shown that making eye movements during recall of a negative autobiographical memory decreases the self-rated vividness and/or

unpleasantness of the memory (see van den Hout & Engelhard, 2012). These findings suggest that the dual task resembles a UCS deflation process. However, in order to unambiguously demonstrate UCS deflation and to examine its effect on conditional (fearful) responding, a conditioning paradigm is needed. In such a paradigm the use of an unequivocal UCS with fixed duration and exact presentation timing is essential. In **chapters 3** and **4**, we employed a conditioning paradigm and used standardized, non-idiosyncratic stimulus material as UCS. In **chapter 3** we demonstrated that eye movements during recall of an aversive film clip (i.e., the UCS), relative to recall only, causes a reduction in memory emotionality, indicating UCS deflation. In **chapter 4**, however, small reductions in memory emotionality were shown in the dual task condition, but also in the control conditions. Possible explanations for the latter results are the relatively low scores at the pre-test, which left little room for decrease, and relatively quick habituation to the stimulus material in all conditions. The presence or absence of quick habituation may be related to methodological differences between the two experiments. In **chapter 3** the UCS comprised a combination of explicit film and sound clips. In contrast, in **chapter 4**, the UCS was a picture that was relatively poor in detail. It is likely that the amount of (aversive) detail is associated with the speed of stimulus habituation. Future investigations testing effects of dual-tasks in a conditioning paradigm may therefore pay special attention to this variable. In sum, we provided evidence that eye movements during aversive ideation resembles a method of UCS deflation.

Is the eye movement intervention of help in the reduction of fear renewal?

Great efforts have been made to find methods that counter the renewal of fear. The experimental studies targeting this clinical problem have typically been inspired by the inhibitory learning account of fear extinction. However, whereas previous investigations mostly focused on methods that strengthen extinction memory or facilitate its retrieval, the present thesis explored an alternative route: extinction retention through weakening of conditioning memory. A few other research groups have recently adopted a similar approach, reflecting that the exploration of this pathway is a logical and timely one (e.g., Costanzi et al., 2014; Dibbets, Poort, & Arntz, 2012; Haesen & Vervliet, 2014; Kindt, Soeter, & Vervliet, 2009; Storsve, McNally, & Richardson, 2010, 2012). Interestingly, their work stems from different disciplines (i.e., human and animal research) and includes various techniques (e.g., drug administration, habituation, imagery rescripting) and multiple outcome measures (e.g., self-report, psychophysiology), which enables cross-validation of the current findings.

The study reported in **chapter 4** demonstrates that eye movements during recall of a UCS memory attenuates context-driven return of UCS expectancy. As stated earlier, no evidence was found that UCS deflation took place in this study. Instead, the effect on UCS expectancy was attributed to reductions in the *accessibility* of the UCS memory. The only other study that aimed to (1) devaluate the UCS memory through guided imagery, and (2) test the effect on fear renewal, was by conducted by Dibbets and colleagues (2012). Healthy participants underwent fear conditioning, i.e. learned to associate a picture of a car (CS) with a picture of a mutilated child (UCS), in context "A", and underwent extinction in context "B". Finally, they saw the CS in context "A". Results were that, relative to a control condition, imagery-based rescripting of the memory of the aversive storyline (i.e., the child dying) into a more acceptable one (i.e., the child surviving and recovering from the accident) caused a devaluation of the UCS picture valence and a reduction in renewal of UCS expectancy. It may be argued that imagery rescripting not only manipulated UCS valence, but also affected the relationship between the CS and the UCS, i.e. the original UCS was replaced by another, less severe one. Therefore, reductions in UCS expectancy may have resulted from associative/ inhibitory learning rather than changes in appraisal of the UCS. This explanation, however, is at odds with the widely-reported observation that inhibitory learning *is* associated with context-driven return of UCS expectancy (e.g., Effting & Kindt, 2007; Haesen & Vervliet, 2014; Leer, Engelhard, Dibbets, & van den Hout, 2013; Leer & Engelhard, 2015; Neumann, Lipp, & Cory, 2007). Thus, the data reported in **chapter 4** together with Dibbets et al. (2012) suggest that imagery techniques can weaken conditioning memory and thereby attenuate renewal of UCS expectancy.

This finding is striking in two ways. First, Davey's (1997) contemporary conditioning model holds that the fear-diminishing effect of UCS deflation is independent of changes in the CS-UCS association. This has indeed been experimentally demonstrated (e.g. Hosoba, Iwanaga, & Seiwa, 2001; Leer & Engelhard, 2015). This implies that, despite the fact that UCS expectancy is an important component of fear learning (Boddez, Baeyens, Luyten, Vansteenwegen, Hermans, & Beckers, 2013), fear reduction via UCS deflation should not be expressed through this measure. Second, other human studies that looked at the effect of UCS deflation on fear renewal did *not* find effects on UCS expectancy. For example, Haesen and Vervliet (2014) tested the effect of repeated UCS exposure. This procedure causes habituation and should therefore lead to positive reappraisal and less renewal of conditional responding. Following fear acquisition, one group received CS-only exposures (typical extinction) and another group received UCS-only exposures (habituation). Upon return to the acquisition context, renewal of conditional skin conductance responding (SCR) was eliminated in the latter

group only. UCS expectancy, however, recovered in both groups. Apparently, and in line with Davey's conditioning model, UCS deflation did not cause changes in the CS-UCS association. Research by Kindt and colleagues shows a fairly similar pattern of results (e.g., Kindt et al., 2009; Sevenster, Beckers, & Kindt, 2012; Soeter & Kindt, 2010, 2012). In a series of experiments they investigated how beta-adrenergic receptor blockage during reconsolidation of condition memory – a manipulation that is hypothesized to selectively disrupt protein synthesis of the amygdalar fear memory – affects fear renewal. This procedure does not directly target UCS valence, yet it exerts its fear-diminishing effect through non-associative learning, like UCS deflation, and should therefore not impact the CS-UCS association. Typical findings are that the manipulation blocks return of fear-potentiated startle (FPS) responding, but not of UCS expectancy and SCR, thus leaving "declarative memory" of the CS-UCS association intact. The authors suggest that FPS responses reflect *affective* learning and that SCRs and expectancy ratings reflect *contingency* learning (e.g., Soeter & Kindt, 2010; but this is somewhat controversial, see Agren, 2014). Interestingly, their findings and interpretation with regard to the SCR data deviates from Haesen and Vervliet (2014), who did not find recovery of SCR and concluded that this measure tracks the interaction between estimated UCS probability and intensity, and thus reflects *threat* learning. Anyhow, both research lines show that interventions that work via non-associative learning do not affect recovery of UCS expectancy. This idea is further supported by the study reported in **chapter 5**. Here, we assessed UCS probability and UCS cost estimates separately, and revealed that UCS deflation does not exert its effect through changes in UCS expectancy, but rather via reductions in the anticipated cost of the UCS.

The question remains: how can these findings be reconciled with the data reported in **chapter 4** and by Dibbets et al. (2012)? There may not be a clear answer to this question. First, UCS expectancy may not perfectly correlate with (i.e. is not a perfect index of) the strength of the CS-UCS association, and may also be influenced by factors other than associative learning. For example, decision making (like providing a UCS expectancy rating) can be biased by one's affective state, which in turn can be induced by the anticipated outcome (cf. the *somatic marker hypothesis*, Damasio, Everitt, & Bishop, 1996). That is, a sense of current threat may increase UCS expectancy. Accordingly, threat-devaluing techniques may decrease UCS expectancy. However, there is no a priori reason to suppose why this process would be involved in some UCS deflation processes (e.g., eye movements during UCS recall or imagery rescripting) and not in others (e.g., habituation; Haesen & Vervliet, 2014). Alternatively, reductions in the *accessibility* of the UCS memory may affect UCS expectancy. Risk assessment has been linked to the availability of threatening information (cf. the *availability heuristic*, Kahneman, Slovic,

& Tversky, 1982), and UCS occurrence may therefore be underestimated or overestimated, depending on the ease of UCS recall. Relevant in this regard is the reaction time data by van den Hout, Bartelski, and Engelhard (2013) and reported in **chapter 6** showing that eye movements during recall reduce objective memory accessibility. This explanation can thus be forward to account for the observed attenuation of UCS expectancy in **chapter 4**, and may also explain the results by Dibbets et al. (2012).

It is also interesting to consider the current findings in light of animal research on this topic. Studies on rodents and mice have investigated the effect of post-conditioning UCS habituation and post-conditioning exposure to the UCS at lower intensity on return of conditional responding (e.g., Costanzi et al., 2014; Jordan, Strasser, & McHale, 2000; Hall & Channell, 1985; Hall & Honey, 1989; Marlin & Miller, 1981; Rauhut, Thomas, & Ayres, 2001; Strosve et al., 2010; 2012). They showed that repeated exposure to loud white noise causes a reduction in *unconditional* startle responding that persists after a context manipulation (Jordan et al., 2000; Marlin & Miller, 1981; Storsve et al., 2010; 2012), indicating context-independency of stimulus habituation. However, *conditional* responses, such as orienting behavior and response suppression (Jordan et al. 2000; but see Hall & Channell, 1985 and Hall & Honey 1989), and freezing (Storsve et al., 2010; 2012) recover after return to the acquisition context. This suggests that, in rodents, UCS deflation does not reduce fear renewal. In contrast, a recent study (on mice) by Contanzi et al. (2014) demonstrated that post-conditioning exposure to the UCS (i.e., an intense electric foot shock) at lower intensity not only caused an immediate reduction in conditional freezing, but also blocked spontaneous recovery and attenuated reinstatement (relative to an extinction group that received non-reinforced CS exposures). It is unclear whether exposure the UCS at lower intensity, compared to repeated UCS exposure, is simply a more effective way to accomplish UCS deflation, or methodological differences (e.g., the intensity of the UCS during conditioning) underlie these contrasting results.

In conclusion, there is preliminary evidence that eye movements during recall of a UCS reduces fear renewal. Specifically, the results reported in **chapter 4** show that the dual task attenuates UCS expectancy. This finding is not in line with contemporary conditioning theory, which holds that UCS deflation operates without affecting the CS-UCS association, and with studies that have not found an effect on UCS expectancy (Haesen & Vervliet, 2014; Leer & Engelhard, 2015). It is, however, in line with earlier studies about the availability heuristic, which showed that risk assessment depends on the availability of threatening information (Kahneman, Slovic, & Tversky, 1982). One account of the findings in **chapter 4** is that a process other than UCS deflation took place, such as a reduction in memory accessibility. This

is not to state, however, that the dual task does not resemble a UCS deflation process. The findings in **chapter 3** suggest that it does. Together, these findings culminate in the hypothesis that eye movements during recall of a UCS can reduce (renewal of) conditional fear via multiple pathways: (1) via UCS deflation and (2) via decreases in memory accessibility. Clearly, the experiments reported in the current thesis are preliminary and additional research is needed before firm conclusions can be drawn.

Limitations and future directions

The current studies have provided new theoretical insights, but also have limitations. A first one relates to the validity of our measures. Can we conclude that the blurring of aversive memory provides a novel route to *fear* reduction? The studies included in **chapter 3**, **4**, and **5** are relevant in this regard, and will be discussed. In **chapter 3** it was shown that eye movements during recall of a *disgusting* stimulus reduces UCS expectancy, CS unpleasantness, and self-reported levels of fear to the CS. Indeed, UCS expectancy is acknowledged as an important component of the fear response and the UCS expectancy rating is considered a valid measure of fear (e.g., Beckers, Krypotos, Boddez, Effting, & Kindt, 2013; Boddez et al., 2013). However, in the context of fear learning it is assumed that the UCS is a *threatening* stimulus and UCS expectancies mimic *danger* expectancies (Boddez et al., 2013). Yet UCS expectancies in **chapter 3** (and presumably also in **chapter 4**, in which a picture of a mutilated child served as the UCS) reflect the anticipation of a stimulus that primarily elicits disgust, and not fear. One may therefore argue that the observed effect on UCS expectancy does not translate to a reduction in fear. Conversely, disgust-evoking UCSs are critically involved in several anxiety-related disorders (see Engelhard, Leer, Lange, & Olatunji, 2014), and cues predictive of such UCSs can evoke (anticipatory) fear responses. For example, about 50% of patients with obsessive-compulsive disorder suffer from fear of contamination (see Rachman, 2004). Also, individuals who suffer from fear of vomiting are characterized by high reports of fear, the presence of panic symptoms, and avoidance/safety behaviors (e.g., van Hout & Bouman, 2012). Finally, stimuli that primarily evoke disgust have been implicated in the onset and maintenance of blood-injection-injury phobia and animal phobias (e.g., Sawchuk, Lohr, Tolin, Lee, & Kleinknecht, 2000). Furthermore, as shown in **chapter 3**, self-reported fear to neutral stimuli increases following pairings of these stimuli with a disgust-evoking UCS. However, it cannot be ruled out that such ratings (partially) reflect feelings of disgust or a more general form of distress. That is, participants may not be able to accurately differentiate between these constructs. In sum, the findings reported in **chapter 3** and **4** are relevant in the context of fear

learning, but firm conclusions await replication of the effects using a UCS that primarily evokes a fear response (e.g., a loud burst of white noise or electrocutaneous stimulation).

In **chapter 5** our primary aim was not to investigate the eye movement intervention, but to test the cognitive factor(s) that may underlie the presumed effect of UCS deflation on (renewal of) conditional fear. To this end we used a fearful UCS (i.e. a 100 dB white noise tone) and measured both UCS expectancy and the anticipated cost of the UCS. These factors are held to constitute the intensity of conditional fear (Davey, 1997) and thus provide insight in *how* UCS deflation techniques, such as the blurring of aversive memory, may reduce fear. It may be argued at this point that such verbal reports reflect only one of the three fear response systems (i.e., subjective experience, next to physiological activity and overt behavior; e.g., Lang, 1968). Importantly, these three indices of fear only show weak correlations over individuals and over time (e.g., Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Moreover, reports about subjective experiences are especially prone to experimental demand. Thus, the results reported in **chapter 5** add to our understanding of how blurring of aversive memory may affect fear reduction, but replication of the findings is needed and the inclusion of physiological and behavioral indices of fear in future investigations is worthwhile.

In conclusion, the results reported in **chapter 3**, **4**, and **5** are preliminary and need replication. Moreover, to extend their external validity, future studies should include (1) a UCS that primarily evokes a fear response and (2) fear indices other than self-report, i.e. measures of behavioral avoidance (e.g., an approach-avoidance task) and physiological activity (e.g., fear-potentiated startle EMG).

A second limitation is that only nonclinical samples were tested. Therefore, it is unclear whether the findings may be generalized to clinical populations. This research strategy was chosen for several reasons. First, the human fear conditioning paradigm is a powerful and successful laboratory model for the study of (clinical) fear and anxiety (e.g., Beckers et al., 2013; Craske, Hermans, & Vansteenwegen, 2006; LeDoux, 2014), and its (face, predictive, and construct) validity has been acknowledged (e.g., Vervliet & Raes, 2013; but see recent criticisms directed at its diagnostic validity; Beckers et al., 2013). Moreover, research in fear conditioning has been successful in translation of results from preclinical work (on animals and healthy humans) to real-world fears (e.g., Vervliet, Craske, & Hermans, 2013). Thus, there is no large gap between preclinical and clinical research in the fear domain. The same rationale applies to the effects of the eye movement intervention. Working memory theory, which is supported by a large body of preclinical studies, has been validated in clinical PTSD research (Lee & Cuijpers, 2013; van den Hout et al., 2012). Finally, the use of nonclinical samples in

strictly controlled experiments comes with optimal randomization of individual differences and allows control of all relevant factors involved. As such, the mechanisms at work can be determined systematically and conclusions can be drawn about causality. In summary, the current research may best be considered in a preliminary stage of a translational research program. Its aim was to examine fundamental learning principles and therefore nonclinical samples were used. A next step is to verify the clinical relevance of the findings in clinical analogue studies.

Clinical implications

Although preliminary, the current studies may ultimately have important clinical implications. In the introduction of this thesis we explained that exposure-based techniques are central to the treatment of fear (Arch & Craske, 2009) and capitalize on weakening of the CS-UCS association. Although this approach effectively reduces conditional fear in the short-term (e.g., Hofmann & Smits, 2008; Olatunji, Cisler, & Deacon, 2010), it is associated with a continuous risk for a return of fear (e.g., Vervliet et al., 2013). The current studies have provided two important new insights in this respect. First, UCS deflation, relative to repeated CS exposure, is associated with less renewal of conditional responding. Second, eye movements during aversive ideation, a technique that is used in EMDR therapy, can be conceptualized as a UCS deflation process, and may therefore provide a value technique in the attenuation of fear renewal.

Obviously, the findings need to pass the critical test of replication and clinical validation. Nevertheless, it is worthwhile to discuss how the results may ultimately be implemented. First, the eye movement intervention may not be of use in *all* clinical cases that involve conditional fear. Because the dual task capitalizes on mental imagery, it may only affect (renewal of) conditional responding when upsetting images play an important role in the disorder. This may differ from person to person, and may depend on the type of anxiety disorder. Interestingly, recurrent intrusive imagery is not restricted to PTSD, but is also reported by patients with social phobia, agoraphobia, obsessive-compulsive disorder, spider phobia, and health anxiety (for overviews, see Engelhard, van den Hout, Janssen, & van der Beek, 2010; Hackmann & Holmes, 2004, and Hirsch & Holmes, 2007). If, however, patients do not report any intrusive images, or if the content of their images does not relate to the psychopathology, then the eye movement intervention may not be of help.

Second, we do not recommend that UCS deflation techniques, like the eye movements, should be used *instead* of traditional exposure. Rather, they could be used in combination. It is

well documented in the literature that fear can return when initially reduced UCS expectancy increases again (e.g., via spontaneous recovery, renewal, reinstatement; e.g., Vervliet et al. 2013). Based on contemporary conditioning theory (Davey, 1997) it can also be expected that fear returns when initial threat-devaluation is nullified following subsequent UCS *inflation* processes (such as repeated UCS imagination or worrying about the catastrophic outcome; e.g., Davey & Matchett, 1994). For example, a patient may show a reduction in fear of flying after devaluation of recurring mental images (e.g., of a crashing aircraft). Exposure to novel footage, e.g. via the media, may then reinstate a sense of current threat and thereby give rise to a return of fear. Only targeting one of the two processes (i.e., UCS expectancy or UCS memory valence) thus seems suboptimal. Instead, when treatment of fear involves both UCS expectancy violation *and* threat devaluation, this provides a 'buffer' against fear renewal. Specifically, low UCS expectancy in combination with low UCS memory emotionality should not result in fear renewal when UCS expectancy suddenly increases again (e.g., when a CS is encountered outside the extinction context). Likewise, it can be expected that when UCS expectancy is still low, conditional fear does not return after UCS inflation. UCS deflation may thus best be used as an add-on therapy to traditional exposure.

New insights in how eye movements in EMDR work

The experiments reported in this thesis further contribute to our understanding of *how* the eye movements in EMDR exert their beneficial effects. In the general introduction we explained that there is much evidence for a working memory theory. In short, this account holds that eye movements during aversive ideation causes a competition for working memory resources, which results in suboptimal memory recall that is associated with lower levels of distress. The experiments reported in this thesis not only provide corroborating evidence for this theory, but also provide new insights.

First, many of the previous studies used an 'instructed recall' paradigm. Specifically, in this paradigm the experimenter instructs the participant to bring to mind a negative autobiographical memory as vividly as possible, while thinking about what happened, who was present, and as it were happening right now. Next, memory vividness and emotionality are rated (cf. the procedure we used in **chapter 2**). Arguably, this procedure facilitates optimal memory retrieval and provides a useful way to study the effects of the dual task. However, in real life, memories are typically triggered by subtle cues, i.e. conditional stimuli. PTSD is characterized by recurrent (involuntary and often externally triggered) flashbacks to the traumatic event (i.e.,

intrusive remembering, reliving, and having nightmares) and avoidance of reminders cues (APA, 2013). In **chapter 6** we describe the example of an assault victim that encounters a person that perceptually resembles the perpetrator and is thereby reminded of the assault. Considering its ecological validity, it is thus interesting to know how the dual task affects levels of distress that are elicited by (conditional) stimuli that activate aversive memory through their memory-encoded association (i.e. 'cued recall'). In the experiment reported in **chapter 3** we assessed the effects of both instructed and cued recall. Participants were presented with pairings of a neutral tone stimulus and an aversive audiovisual stimulus (i.e. a highly disgusting film clip). As a result, the tone stimulus itself became evaluated as fearful and unpleasant, as evidenced by self-report measures. Next, using the instructed recall paradigm, participants visualized the content of the film clip with or without making eye movements. Findings were significantly larger drops in memory vividness and emotionality ratings in the dual task group compared to the control group. Finally, during a critical test phase, participants were represented the tone stimulus. Only the dual task group reported lower levels of fear evoked by this stimulus, and indicated that the tone stimulus was less unpleasant, compared to pre-intervention. This experiment provided the first evidence that the eye movement intervention reduces levels of subjective distress associated with cued recall.

Second, most studies investigating the working memory account have focused exclusively on measures of self-report (i.e., by means of visual analogue scales for memory vividness and emotionality). Given that subjective memory experience is an important clinical index, such endpoints should not be considered subordinate per se. However, the risk of demand characteristics is relatively high using this methodology (but see Gunter & Bodner, 2008, exp. 1). A few studies did include other measures. Engelhard, van Uijen, and van den Hout (2010) measured FPS responses during memory recall and found larger reductions from a pre-test to a post-test in the dual task condition compared to a control condition, which suggests that memory recall at the post-test was associated with lower levels of distress. This effect should be replicated. Van den Hout et al. (2013) had participants encode a complex visual display and then recall it with or without eye movements. Subsequently, participants did a reaction time task in which they indicated as quickly as possible whether fragments were part of the original image. Results were that reaction times were slowed down in the dual task condition, which indicates a reduction in (objective) memory accessibility (and possibly reflects a reduction in memory vividness). The study included in **chapter 6** provides a first conceptual replication of this finding by demonstrating that eye movements during recall of a stimulus representation, compared to recall only, slows down the process of comparing it with subsequently perceived

stimuli. The data reported in **chapter 6** thus provide corroborating evidence for the idea that the dual task's effects are not driven by mere recall, or experimental demand, and thereby further support the working memory account.

Third, working memory theory provides an explanation for drops in memory vividness and emotionality *during* the dual task. However, as put forward in **chapter 2**, competition for memory resources during the intervention can, in itself, not account for the typically observed rating reductions at the immediate/delayed post-test, or the clinical observation that EMDR affects the vividness/emotionality of memory recall *after* a treatment session. One rationale that has been advanced holds that the instant loss of memory detail is reconsolidated in long-term memory (e.g., Engelhard, 2012; Maxfield, Melnyk, & Hayman, 2008; van den Hout et al., 2010). This account capitalizes on research that has shown that when (fear) memory is retrieved from long-term memory, it enters a transitory labile state in which it can be updated before is stabilizes again (i.e., reconsolidates; Nader & Hardt, 2009, and see the work by Kindt and colleagues). Empirical data supporting a reconsolidation account of the eye movement's long-term effects, however, are scarce. In **chapter 2** we explain that immediate rating changes (i.e., within minutes after the invention) cannot be taken as evidence for the reconsolidation account. Possibly, participants find it difficult to differentiate between what they had experienced during the dual task and what they experience directly afterwards (van den Hout, Muris, Salemink, & Kindt, 2001). If rating changes persist at a *delayed* (and therefore unambiguous) post-test, then this may suggest that the memory has actually been modified. The findings thus far were mixed. One study demonstrated lasting effects of the dual task (i.e., at a 1-week follow-up test; Gunter & Bodner, 2008, exp. 2), but four studies did not (Kavanagh, Freese, Andrade, & May, 2001; Lee & Drummond, 2008; Lilley, Andrade, Turpin, Sarbin-Farrell & Holmes, 2009; Schubert, Lee, & Drummond, 2011), and these findings might reflect a chance discovery in the first study or indicate methodological shortcomings in the other studies. In **chapter 2** we addressed an important methodological variable (i.e., intervention duration) and provided the first replication of Gunter and Bodner's (2008) results. Specifically, we demonstrated that eight dual task periods of 24 s, but not four periods, reduced memory vividness/emotionality at a 24 h follow-up test. This pattern of results can account for the null-results in some of the previous studies (i.e., Kavanagh et al., 2001; Lilley et al., 2009), and the findings provide corroborating evidence that the dual task *has* long-term effects.

Can we conclude that these data support a reconsolidation account? No, because there is still an alternative explanation for these (relatively) long-term effects. Gunter and Bodner (2008, p. 928) put forward: "By experiencing the memory in a weakened form (while being

distracted), people may change some of their beliefs about how dangerous their memories are, as well as their beliefs about their ability to cope with remembering them". In other words, distraction during aversive ideation may primarily work to foster memory acceptance. Hence, the (persistent) reductions in memory emotionality may reflect changes in memory appraisal, rather than changes in memory characteristics. Nevertheless, the *appraisal account* and the reconsolidation account may coexist: the two are not mutually exclusive. Perhaps the dual task alters memory characteristics as well as memory appraisals, and it may well be that the former facilitates the latter.

Future research may focus on testing these two accounts. This is relevant, because insight into the mechanism(s) underlying the eye movement's long-term effects has important theoretical and clinical implications. A theoretical implication relates to whether or not the dual task involves a *unique* intervention. If the eye movements merely change memory appraisals, then the mechanism that drives its long-term effects is not different from the one that is held to underlie the effects of (imaginal) exposure. Specifically, exposure is thought to initiate a search for meaning, which allows the processing of sensory information into a more meaningful form, so that the traumatic event can be integrated into one's autobiographical memory knowledge base (e.g., ; Engelhard, Arntz, & Kindt, 2011; Engelhard & van der Ploeg, 2015). The appraisal account thus holds that the eye movements primarily facilitate this process by making imaginal exposure more palatable. A clinical implication is that changes in memory appraisal should be achieved more rapidly following the dual task compared to mere imaginal exposure. Also, (imaginal) exposure to feared stimuli typically elicits negative reactions that can trigger drop-out. A key strategy to deal with such reactions may be stepwise exposure (e.g., Kavanagh et al., 2001). In line with the appraisal account, the execution of eye movements can be conceptualized as the first steps of such an approach, thus increasing short-term treatment benefits and decreasing drop-out. Alternatively, the eye movements may not (only) change memory appraisal, but rather cause a permanent loss of memory detail (cf. the reconsolidation account). This account thus holds that the dual task is driven by a mechanism that is qualitatively different from the one supposedly driving the effects of imaginal exposure. If the eye movements exert their long-term effect primarily through reductions in memory detail, then such changes should be closely monitored during treatment. Based on this account, it can further be predicted that the dual task as currently used in EMDR (i.e., including images that are associated with the trauma hotspot) not only results in the blurring of intrusive images, but may also cause a permanent loss of detail of *useful* mental images (such as mental representations of danger cues). Obviously, such an outcome would be undesirable. Preliminary support for this

hypothesis was provided in **chapter 6**, and replication of these findings is thus urgently needed. Ultimately, clinicians may be advised to be alert in determining what memory features to include in the dual task.

Conclusion

To summarize, the experiments reported in this thesis contribute to our understanding of how blurring of aversive memory may provide a novel route to fear reduction. Main findings are that the execution of eye movements during aversive ideation can be conceptualized as a method of UCS deflation, and that such methods have the potential of attenuating fear renewal. We further showed that eye movements have lasting effects on self-report measures of memory vividness and emotionality, indicating long-term changes in memory appraisals/detail. Finally, we revealed a possible risk of the dual task: blurring of a conditional stimulus representation may actually *increase* generalization of fearful responding to previously non-threat-related stimuli. If replicated and clinically validated, this result should serve a warning to therapists.

The currents findings are preliminary and limited in some respects. Future research should aim to replicate the results, also with behavioral and psychophysiological indices of fear. A next step is clinical validation. The observed results have also raised important new questions. Can the dual task's fear-diminishing effect be attributed to the process of UCS deflation, or to reductions in memory accessibility, or both? Do the eye movements involve a unique mechanism, or do they primarily facilitate imaginal exposure? Ultimately, the answering of these empirical questions may help to advance the treatment of pathological fear.

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Summary in Dutch

(Samenvatting in het Nederlands)

Achtergrond

Bijna één op de vijf Nederlanders krijgt eens in het leven te kampen met een angststoornis. Per definitie veroorzaakt dit lijden of sociale dan wel beroepsmatige problemen. De meest effectieve behandeling op dit moment is cognitieve gedragstherapie (CGT). Echter, ondanks het succes en de brede toepassing van CGT is er bij een aanzienlijk deel van de patiënten die in eerste instantie opknapt sprake van terugval, oftewel een terugkeer van angst. Onderzoek naar optimalisatie van de behandeling is dus essentieel. Het doel van dit proefschrift was het exploreren van een nieuwe weg tot vermindering van angstterugkeer: vervaging van akelige herinneringen.

Hoe ontstaat angst?

Angst is een emotie die wordt uitgelokt door situaties die geassocieerd zijn met gevaar. Angst is in het beginsel adaptief: lichaam en geest worden in staat van paraatheid gebracht om de dreigende situatie te lijf te gaan dan wel te ontvluchten (de zogeheten 'fight-or-flight response'). Volgens de breed gedragen conditioneringstheorie is angst een 'geconditioneerde' respons. Dat wil zeggen: angst is aangeleerd. Een hond, bijvoorbeeld, is op zichzelf een neutrale stimulus. Pas na associatie met een biologisch belangrijke 'ongeconditioneerde' stimulus (*unconditional stimulus*; afgekort UCS) zoals een hondenbeet zal het als cue voor dreigend gevaar gaan fungeren (als 'geconditioneerde' stimulus of *conditional stimulus*; afgekort CS). In vakjargon: na vorming van een CS-UCS associatie lokt de CS angst uit.

Angst wordt pathologisch wanneer het generaliseert naar objectief veilige situaties of stimuli en daarmee het normale functioneren belemmert of significant lijden veroorzaakt. Zo neemt angst een pathologische vorm aan bij iemand die niet meer over straat durft uit angst een hond tegen te komen. Pathologische angst komt eveneens tot stand via associatief leren. Toch ontwikkelt slechts een minderheid van de mensen een angststoornis na een traumatische ervaring. Ook heeft niet iedereen met een angststoornis een traumatische gebeurtenis meegemaakt. Het direct leren van een CS-UCS associatie is dus voldoende noch noodzakelijk voor het ontstaan van angst. Hoe valt dit te verklaren? Volgens het conditioneringsmodel van Davey (1997) is angst een interactie tussen (I) de waargenomen kans dat een stimulus (zoals het zien van een hond) wordt gevolgd door de gevreesde uitkomst (zoals een hondenbeet) en (II) de subjectieve ernst van die uitkomst (zoals de ernst van de verwondingen je denkt eraan over te houden). Met andere woorden:

Angst = Kans x Ernst

Een eerste implicatie van dit model is dat elke factor die van invloed is op de (waargenomen) kans op de gevreesde uitkomst een effect heeft op de intensiteit van de angstrespons. Persoonlijke ervaring is slechts één manier om over de associatie tussen de CS en de UCS te leren. Het zien of anderszins afleiden van een dergelijk verband is soms voldoende. Zo kan herhaaldelijk berichtgeving over gecrashte vliegtuigen aanleiding geven tot vliegangst. Omgekeerd geldt hetzelfde: neemt de waargenomen kans op een catastrofe af, dan daalt ook de angst. Conform dit model zorgt blootstelling aan de CS, zonder dat de UCS volgt, voor een daling in angst. Dit leerprincipe is zeer robuust en vormt een basistechniek in CGT (bekend als *exposure*). Bovendien kan op basis van dit leerprincipe voorspeld worden dat mensen die zichzelf na een akelige ervaring opnieuw blootstellen aan de CS (bv. opnieuw een hond aaien of een vliegtuig instappen), hun waargenomen kans op de gevreesde uitkomst verkleinen en minder waarschijnlijk een angststoornis ontwikkelen.

Een tweede implicatie is dat de intensiteit van de angstrespons wordt beïnvloed door elke factor die een effect heeft op de subjectieve ernst van de gevreesde uitkomst. Met andere woorden: herwaardering van de UCS verandert de intensiteit van de angstrespons. Experimenteel onderzoek waarin de ernst van de UCS wordt gemanipuleerd, bevestigt deze veronderstelling. Onafhankelijk van de kans op een UCS leidt een daling in de subjectieve ernst, UCS *deflatie* genaamd, tot een zwakkere angstrespons. Een stijging in de subjectieve ernst, oftewel UCS *inflatie*, leidt tot een sterkere angstrespons. Dit leerprincipe verklaart deels waarom slechts een minderheid van de mensen klachten behoudt na een traumatische ervaring. Zo blijkt uit onderzoek dat piekeren over negatieve gevolgen geconditioneerde angst in stand kan houden.

Waarom keert angst vaak terug na uitdoving?

Exposure is gebaseerd op de conditioneringstheorie en richt zich op het ontkrachten van de geleerde CS-UCS associatie. Dit resulteert in de vorming van een nieuwe associatie, “CS leidt niet tot UCS”, dat ook wel het *extinctiegeheugen* wordt genoemd. Uit fundamenteel onderzoek blijkt dat dit nieuwe geheugenspoor de competitie aangaat met het originele angstgeheugen. Dat wil zeggen, na behandeling wordt bij confrontatie met een CS óf het angstgeheugen óf het extinctiegeheugen geactiveerd. Zolang het extinctiegeheugen de competitie wint, blijven angstresponsen achterwege. Dat angst na uitdoving vaak terugkeert, wordt vanuit dit model dus verklaard doordat het angstgeheugen opnieuw de overhand krijgt. De vraag is hoe dit kan en hoe angstterugkeer voorkomen kan worden.

Uit talrijke experimenten blijkt dat de activatie van het extinctiegeheugen bijzonder contextgevoelig is. Activatie van het angstgeheugen is dat in veel mindere mate. Confrontatie met een CS buiten de extinctiecontext leidt daarom steevast tot angstterugkeer. Om die reden zijn methoden bedacht die zich richten op het versterken van het extinctiegeheugen dan wel het faciliteren van de toegankelijkheid van het extinctiegeheugen. Voorbeelden zijn het opschroeven van het aantal leermomenten en het uitvoeren van *exposure* in meerdere contexten. Een alternatief is het gebruik van een zogeheten *reminder cue* die doet terugdenken aan de behandeling. Alhoewel er enige evidentie is voor de effectiviteit van deze methoden, kan niet worden ontkend dat ze een gemeenschappelijk nadeel hebben: het angstgeheugen blijft intact en daardoor ligt angstterugkeer altijd op de loer.

Een nieuw perspectief

In dit proefschrift onderzochten we een nieuwe methode om de terugkeer van angst te reduceren. Onze hoofdvraag luidde: Leidt aanpassing van het angstgeheugen tot een vermindering van angstterugkeer? We veronderstelden (I) dat angstreductie via UCS deflatie leidt tot een aanpassing in het angstgeheugen (namelijk een lagere emotionele lading van de UCS representatie), maar niet tot een verandering in de CS-UCS associatiesterkte, en daarom (II) dat angstreductie via UCS deflatie contextonafhankelijk is.

Een cruciale vraag op dit punt is: Welke UCS deflatietechnieken zijn bewezen effectief én hebben klinische toepasbaarheid? Experimenteel onderzoek laat zien dat herhaaldelijke blootstelling aan een UCS, bijvoorbeeld een zeer luide toon of een elektrische prikkel, leidt tot gewenning en een minder negatieve evaluatie van de UCS. Dit effect wordt sneller bereikt wanneer de UCS tijdens de interventie wordt aangeboden op een intensiteit die lager is dan tijdens conditionering. Deze methoden leiden aantoonbaar tot angstreductie. Echter, in de klinische praktijk is het vrijwel onmogelijk om een patiënt direct bloot te stellen aan de gevreesde uitkomst. Dit is reden om naar alternatieve technieken te kijken.

In theorie zou het afleren van angst, net als het aanleren van angst, bereikt kunnen worden zonder directe blootstelling aan de gevreesde stimulus en catastrofale consequentie. Een potentieel bruikbare techniek die recentelijk veel wetenschappelijke aandacht heeft gekregen, is het vervagen van akelige herinneringen. Deze techniek vormt een belangrijke schakel in *eye movement desensitization and reprocessing* (EMDR), een van de meest effectieve en meest onderzochte therapieën voor posttraumatische stressstoornis. Tijdens een EMDR-sessie visualiseert de patiënt het actueel meest beladen herinneringsbeeld van de traumatische ervaring. Tegelijkertijd maakt de patiënt horizontale oogbewegingen door de heen-en-weer

bewegende wijsvinger van de therapeut te volgen. Ondanks de wat vreemde eerste indruk die deze techniek wekt, blijkt uit recent onderzoek dat de oogbewegingen daadwerkelijk toegevoegde waarden hebben. Bovendien is er sinds enkele jaren meer duidelijkheid over hoe dit kan. Volgens de werkgeheugentheorie belasten de oogbewegingen het werkgeheugen, net als het ophalen van een akelige herinnering. Worden beide taken simultaan uitgevoerd, dan raakt het werkgeheugen overbelast. Hierdoor wordt de opgehaalde herinnering minder helder en minder onaangenaam. Daarna wordt deze nieuwe, mildere herinnering weer opgeslagen in het langetermijngeheugen, in plaats van de oude, intensere herinnering.

Zowel experimenteel als klinisch onderzoek laat zien dat de zogenaamde duale taak de emotionele lading van negatieve herinneringen doet afnemen. De oogbewegingen zorgen ervoor dat de *subjectieve* ernst van de gebeurtenis, oftewel de negatieve beleving van de herinnering daaraan, vermindert. In theorie kan de duale taak dus fungeren als UCS deflatietechniek en daarom worden ingezet om de terugkeer van angst te reduceren. In dit proefschrift stelden we ons ten doel deze assumpties te onderzoeken. De hieronder beschreven experimenten zijn uitgevoerd in steekproeven uit een gezonde studentenpopulatie.

De bevindingen in dit proefschrift

Hoofdstuk 2 onderzocht de werking van de oogbewegingen. Meer specifiek toetsten we of de directe dalingen in helderheid en onaangenaamheid, die herhaaldelijk zijn gerapporteerd in de literatuur, standhouden na een tijdsinterval van 24 uur. Participanten werd gevraagd om twee negatieve autobiografische herinneringen te selecteren (bv. het getuige zijn van een ernstig ongeluk). Tijdens een pre-test werden beide herinneringen gescoord op helderheid en onaangenaamheid. Vervolgens werd een van de herinneringen gevisualiseerd mét horizontale oogbewegingen. In de controleconditie werd de andere herinnering gevisualiseerd zónder oogbewegingen. Naast het effect van de oogbewegingen bestuurden we het effect van interventieduur: visualisatie (in beide condities) besloeg voor de helft van de participanten vier periodes en voor de andere helft acht periodes. Tijdens een post-test scoorden de participanten beide herinneringen opnieuw op helderheid en onaangenaamheid. Eén dag later kwamen ze terug naar het laboratorium voor een follow-up meting en scoorden ze de herinneringen voor een laatste maal en op eenzelfde wijze. De resultaten lieten zien dat, onafhankelijk van interventieduur, oogbewegingen tijdens visualisatie zorgen voor directe daling in de helderheid van de herinnering, maar niet in onaangenaamheid. De daling in helderheid hield stand tot de follow-up meting, maar enkel in de groep die acht periodes visualiseerde. Daarnaast bleek in de groep die acht periodes visualiseerde dat (ondanks het uitblijven van een directe daling)

onaangenaamheidscores waren gedaald van de pre-test naar de follow-up. Deze bevindingen suggereren dat de duale taak zorgt voor een permanente verandering in de beleving van de herinnering, mits de interventieduur lang genoeg is.

Hoofdstuk 3 onderzocht of het maken van oogbewegingen tijdens visualisatie van een UCS herinneringsbeeld leidt tot herwaardering en een daling in geconditioneerde angst. Participanten werden geconditioneerd via een computertaak. Telkens na het horen van een toon (CS) zagen ze een walgelijk filmfragment (UCS; een hevig brakende jongeman). Deze procedure resulteerde in een stijging in de gerapporteerde angst voor de CS. Vervolgens vroegen we de participanten om de filmfragmenten te visualiseren. De helft van hen maakte tegelijkertijd oogbewegingen, de andere helft deed dit niet. Ten slotte hoorden alle participanten opnieuw de CS en rapporteerden ze hun angstniveau. De resultaten lieten zien dat de oogbewegingen zorgden voor een scherpe daling in de helderheid en onaangenaamheid van de herinnering aan de filmfragmenten. Daarnaast daalde de angst voor de CS, maar alleen in de groep die oogbewegingen maakte. Deze bevindingen zijn in lijn met Davey's conditioneringsmodel en suggereren dat de duale taak kan fungeren als UCS deflatietechniek.

Hoofdstuk 4 onderzocht of de duale taak ingezet kan worden om de terugkeer van angst te verminderen. Via een computer werden participanten onderworpen aan een zogeheten ABA-angstterugkeer taak. Participanten leerden de relatie tussen een CS (een geometrisch figuur) en een UCS (een afbeelding van een verminkt, dood kind) in context "A" (tegen een gele achtergrond). In een extinctiefase werd deze relatie afgeleerd door herhaaldelijke blootstelling aan de CS zónder de UCS. Dit vond plaats in context "B" (tegen een blauwe achtergrond). Ten slotte, tijdens een testfase, werd de CS opnieuw in context "A" getoond. Deze laatste contextswitch leidt zonder interventie tot een terugkeer van angst. We toetsten of het maken van oogbewegingen tijdens visualisatie van de UCS, voorafgaand aan de testfase, zou zorgen voor een daling in de helderheid en onaangenaamheid van de UCS representatie en in een vermindering in angstterugkeer. De resultaten lieten een scherpe daling in helderheid- en onaangenaamheidscores zien. Dit was echter ook het geval in de controlegroep die geen oogbewegingen maakten. Dit experiment leverde dus geen bewijs dat de oogbewegingen resulteren in UCS deflatie. Verder bleek dat in de groep die enkel de UCS visualiseerde angst terugkeerde in de testfase. In de groep die tijdens visualisatie van de UCS oogbewegingen maakte, was dit niet het geval: de duale taak had dus wél het gewenste effect.

Hoofdstuk 5 onderzocht hoe UCS deflatie precies samenhangt met angstreductie en of UCS deflatie leidt tot verminderde angstterugkeer. In dit experiment splitsten we de meting van angst op in (I) de verwachting van de UCS en (II) de geschatte ernst van de UCS. In theorie zou

UCS herwaardering enkel de laatstgenoemde factor moeten beïnvloeden. Participanten weden opnieuw onderworpen aan een ABA-angstterugkeer taak. Tijdens acquisitie in context "A" leerden ze de relatie tussen een CS (een geometrisch figuur) en een UCS (een zeer luide ruistoon; 100 dB). Tijdens de interventiefase (eveneens in context "A") werd in een experimentele groep de ruistoon herhaaldelijk aangeboden, waarbij het volume stapsgewijs daalde tot 70 dB. Een controlegroep kreeg eveneens herhaaldelijk de ruistoon aangeboden, maar hier bleef het volume 100 dB. De resultaten lieten zien dat de experimentele groep de ruistoon een stuk minder onaangenaam vond na deze interventie. In de controlegroep was slechts een kleine daling zichtbaar (ten gevolge van gewenning). Vervolgens werden beide groepen opnieuw en herhaaldelijk blootgesteld aan de CS, maar nu zonder de UCS en in context "B". Ten slotte werd in een testfase de CS aangeboden in context "A". Tijdens elke CS werd de geschatte kans op een UCS en de geschatte ernst van de UCS gemeten. De resultaten lieten zien dat beide interventies geen effect hadden op de kansschatting. Echter, zoals verwacht, daalde de geschatte ernst van de UCS in de experimentele groep sterker dan in de controlegroep, waar geen daling plaatsvond. Deze effecten bleven behouden na terugkeer naar acquisitiecontext "A". Dit bevestigt de hypothese dat UCS deflatie leidt tot contextonafhankelijke angstreductie.

Hoofdstuk 6 onderzocht een potentieel risico van de oogbewegingen: vervaging van bepaalde geheugenbeelden kan juist leiden tot méér angst. Tijdens een EMDR-sessie visualiseert de patiënt een akelig herinneringsbeeld en maakt tegelijkertijd horizontale oogbewegingen. Een slachtoffer van een gewelddadige overval denkt bijvoorbeeld aan het moment waarop een mes tegen de keel werd gedrukt. Door de oogbewegingen wordt dit herinneringsbeeld vager en dragelijker. Als typische vervolgstap vraagt de therapeut: "Wat komt er nu in je op?" Dit kunnen nieuwe gedachten, gevoelens of herinneringsbeelden zijn, zoals het uiterlijk van de dader of kenmerken van de omgeving waarin de overval plaatsvond. De patiënt focust zich dan op dit gerelateerde herinneringsbeeld en maakt opnieuw oogbewegingen. Deze vervolgstap heeft aanzienlijk minder wetenschappelijke aandacht gekregen en werkt mogelijk averechts. We veronderstelden (I) dat het vervagen van een CS representatie (zoals het uiterlijk van de dader in bovenstaand voorbeeld) ervoor zorgt dat de CS en perceptueel gelijkende stimuli (bijvoorbeeld iemand die sterk lijkt op de dader) moeilijker uit elkaar worden gehouden, en (II) dat dit ertoe leidt dat ook perceptueel gelijkende stimuli angst kunnen oproepen. Vanzelfsprekend zou een dergelijke uitkomst onwenselijk zijn. We toetsten de hypothesen in twee experimenten. In het eerste experiment observeerden de participanten een 'target' foto van het gezicht van een jongeman. Vlak daarna visualiseerden ze deze foto met of zonder oogbewegingen. Enkel de groep die oogbewegingen maakte,

rapporteerde een scherpe daling in de helderheid van het herinneringsbeeld. Ten slotte kregen alle participanten achtereenvolgens een serie gezichten te zien. Deze foto's leken sterk op de target foto, maar waren nét anders. De participanten kregen de opdracht om zo snel mogelijk aan te geven of de getoonde gezichten 'hetzelfde' of 'anders' waren dan het gevisualiseerde gezicht. De resultaten lieten aanzienlijk tragere reactietijden zien in de groep die oogbewegingen maakte: deze groep participanten had dus meer moeite met het maken van onderscheid tussen de target foto en perceptueel gelijkende foto's. In het tweede experiment werd de target foto herhaaldelijk aangeboden en telkens direct gevolgd door een elektrische prikkel. Vervolgens visualiseerde een experimentele groep de foto met oogbewegingen. Een controlegroep deed dit zonder oogbewegingen. Ten slotte kregen alle participanten een foto van een gezicht te zien dat sterk leek op het gezicht op de target foto, maar nét anders was. De resultaten lieten zien dat participanten in de experimentele groep bij dit gezicht een sterkere verwachting van de elektrische prikkel hadden en meer zweetsecretie vertoonden vergeleken met de controlegroep. Samenvattend demonstreren de twee experimenten dat het maken van oogbewegingen tijdens visualisatie van een CS leidt tot een verstoorde discriminatie tussen de CS en perceptueel gelijkende stimuli en tot een sterkere generalisatie van geconditioneerde angst. Deze bevingen suggereren dat het belangrijk is om selectief te zijn bij het kiezen van herinneringsbeelden, in EMDR en ter preventie van angstterugkeer.

Samenvatting en implicaties van de bevindingen

Hoofdstuk 7 vat de bevindingen van dit proefschrift samen en bespreekt de bredere theoretische en klinische implicaties. We lieten zien dat herwaardering van een UCS – een factor die in belangrijke mate bepalend is voor de intensiteit van de angstrespons – behouden blijft wanneer men de 'behandelcontext' verlaat (hoofdstuk 5). Dit biedt perspectief voor de preventie van angstterugkeer. Voorts toonden we aan dat het maken van oogbewegingen tijdens visualisatie van een UCS leidt tot vermindering van geconditioneerde angst (hoofdstuk 3) en de terugkeer daarvan (hoofdstuk 4). Bewijs voor het veronderstelde onderliggende mechanisme, UCS herwaardering, werd echter niet geleverd in hoofdstuk 4. Mogelijk werken de oogbewegingen deels via een ander mechanisme, namelijk via een verandering in de *toegankelijkheid* van de UCS representatie. Ondersteuning voor deze hypothese werd gevonden in hoofdstuk 6. Deze resultaten suggereren dat angstreductie via het aanpassen van het angstgeheugen op de lange termijn voordelen biedt ten opzichte van traditionele *exposure* die resulteert in de vorming van een extinctiegeheugen.

De experimenten uit hoofdstukken 2 en 6 bieden aanvullend inzicht met betrekking tot hoe de oogbewegingen precies werken en hoe deze het best toegepast kunnen worden. In voorgaand onderzoek werd de beleving van een herinnering doorgaans direct (dus binnen minuten) na de interventie gepeild en het is lastig te bepalen wat deze metingen precies weerspiegelen. Hoofdstuk 2 toont aan dat de oogbewegingen resulteren in een *blijvende* verandering in de beleving van de herinnering. Dit is belangrijk omdat dit suggereert dat de oogbewegingen kunnen bijdragen aan een permanente daling van geconditioneerde angst. De bevindingen uit hoofdstuk 6, ten slotte, zijn alarmerend: het vervagen of minder toegankelijk maken van de herinnering aan een CS leidt mogelijk tot een breder scala aan stimuli dat een angstrespons uitlokt. Het lijkt daarom verstandig om selectief te zijn wanneer het aankomt op het opnemen van een herinneringsbeeld in de duale taak, zowel in EMDR therapie als ter preventie van de terugkeer van angst.

Kortom, dit proefschrift laat zien dat het maken van oogbewegingen tijdens visualisatie van een akelig herinneringsbeeld kan worden ingezet als techniek ter reductie van angst en ter preventie van angstterugkeer. In voorgaand onderzoek werd het angstgeheugen doorgaans ongemoeid gelaten, met een voortdurend risico op angstterugkeer tot gevolg. Het huidige onderzoek richtte zich op aanpassing van het angstgeheugen en deze aanpak lijkt effectief.

Deze bevindingen zijn nieuw en dienen met gepaste voorzichtigheid te worden geïnterpreteerd. Allereerst is replicatie van de effecten cruciaal ter vaststelling van de betrouwbaarheid. Een tweede punt van aandacht betreft de (construct)validiteit. De gevonden effecten van de duale taak op geconditioneerde angst in hoofdstukken 3 en 4, bijvoorbeeld, werden afgeleid uit zelfrapportagematen. Deze resultaten zijn belangrijk omdat subjectief ervaren angst een relevante klinische maat betreft. Tegelijkertijd is het risico op zogeheten *demand characteristics* (de neiging van participanten om in te gaan op het verwachtingspatroon van de onderzoeker) bij zelfrapportage relatief hoog. Het is de vraag of het effect van de duale taak zich ook manifesteert op een angstmaat die hier minder gevoelig voor is, zoals de oogknipperreflex. Ten slotte dient opgemerkt te worden dat het huidige onderzoek zich in een ‘preklinisch’ stadium bevindt van een onderzoekslijn waarin de toetsing van fundamentele leerprincipes vooraf gaat aan onderzoek en toepassing in de klinische praktijk. Een toekomstige vervolgstap is toetsing van de hypothesen in (sub)klinische populaties.

Ondanks het prille karakter van de huidige bevindingen is het goed om vast vooruit te blikken op de klinische implicaties. Ten eerste zouden UCS deflatietechnieken de traditionele *exposure* behandeling van angst *niet* moeten vervangen. Immers, *exposure* is bijzonder effectief in het bijstellen van de overschatting van de kans op gevaar. Wanneer de overschatte kans op

gevaar ongecorrigeerd blijft, kan negatieve herwaardering van een gevreesde uitkomst evengoed gepaard gaan met angstterugkeer. Een patiënt met vliegangst die minder angstig is na vervaging van een doembeeld, maar niet geleerd heeft dat vliegen doorgaans veilig is, durft na het zien van nieuwe heftige beelden in de media waarschijnlijk opnieuw niet te vliegen. Idealiter worden *exposure* oefeningen dus gecombineerd met UCS deflatietechnieken. Een tweede punt van aandacht is dat UCS deflatie via de duale taak enkel praktisch nut heeft wanneer de desbetreffende patiënt te kampen heeft met akelige herinneringsbeelden, die logischerwijs in verband staan met de psychopathologie. Interessant in dit kader is dat akelige herinneringsbeelden niet alleen een prominente rol spelen in posttraumatische stressstoornis, maar eveneens in sociale fobie, agorafobie, obsessieve-compulsieve stoornis, spinnenfobie en hypochondrie.

Conclusie

Een aanzienlijk deel van de patiënten met een angststoornis valt terug na behandeling. Er is veel bekend over de factoren die hierbij een rol spelen, maar methoden om angstterugkeer te voorkomen zijn tot op heden ontoereikend. In dit proefschrift toetsten we een nieuwe methode: angstreductie via vervaging van het angstgeheugen. Dit onderzoek biedt het eerste bewijs dat deze techniek kan bijdragen aan de preventie van angstterugkeer.

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About the author

Biography

Arne Leer was born on December 10, 1985 in Amsterdam, the Netherlands. His parents had a broad interest in science (his father obtained a PhD degree in mathematics and natural sciences in 1985) and encouraged him to follow his curiosity. He obtained a Bachelor's degree in neuropsychology from Utrecht University in 2007. After that, he attended a 2-year Research Master Program (Psychological Health Research, Utrecht University). Under the supervision of Prof. dr. Monique Smeets and Prof. dr. Marcel van den Hout he examined whether neutral odors can come to elicit fear (yes they can!). This research stimulated his interest in fear conditioning and experimental psychopathology. After obtaining his Master's degree in 2009, he took a quick detour to the Utrecht Fire Department, and then returned to Utrecht University to start working as a research assistant. He was a PhD student at this university from 2011 to 2015 under the supervision of Prof. dr. Iris Engelhard and Prof. dr. Marcel van den Hout. During this period he received a Short Stay PhD Fellowship from Utrecht University to work at the Center for Excellence on Generalization in Health and Psychopathology at the University of Leuven.

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