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Emotional mental imagery and the reduction of fear within the mind's eye

JOHANNA M. HOPPE



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Abstract

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Mental imagery refers to sensory-perceptual experiences in the absence of external sensory input. Emotional mental imagery (i.e., imagery with emotional content) is a key feature in many mental disorders, such as the image-based intrusive memories of trauma in posttraumatic stress disorder (PTSD). However, mental imagery can also be a vehicle for emotional change. In imaginal exposure, fear-provoking events are revisited using mental imagery. Imaginal exposure is a core component in evidence-based therapies for anxiety and PTSD. Treatment development is needed, as effects are many times insufficient, accessibility is low, and the treatment is not well-tolerated by some patients. The aim of this thesis was to increase knowledge of underlying mechanisms of imaginal exposure and improve our understanding of emotional mental imagery. The thesis explored the neural underpinnings of imaginal exposure and investigated mechanisms that could enhance its effectiveness, accessibility and tolerability. To further our knowledge of intrusive memories in PTSD (i.e., involuntary mental imagery), the characteristics of trauma memory hotspots (worst moments) collected within the first hours after trauma were explored. Study I demonstrated that imaginal exposure to mental imagery of phobic (vs. neutral) stimuli robustly activated emotion-processing brain areas. Study I also revealed that a brief 10-minute session of imaginal exposure was associated with reduced fear one week later. Study II investigated the link between vividness (clarity and liveliness) of mental imagery during imaginal exposure and reduction of fear using an experimental analogue of imaginal exposure (imaginal extinction). No evidence was found that high imagery vividness during imaginal extinction was associated with better long-term reduction in physiological fear responses than lower vividness. Study III revealed that hotspots collected soon after trauma are expressed as motion-rich sensory-perceptual experiences (mental imagery) with little detail on emotion/cognition. The contributions of this thesis involve demonstrating that mental imagery has the power to elicit emotional responses at subjective, physiological and neural levels and suggesting new avenues for treatment development. Future studies should explore the benefits of briefer imaginal exposure sessions to improve the effectiveness and accessibility of imaginal exposure. Future studies should also examine if fear reduction can be obtained with less vivid imaginal exposure, which could help attenuate distress and thereby make imaginal exposure tolerable for more patients. Lastly, the dynamic and visuospatial nature of newly formed trauma memory hotspots may help elucidate mechanisms through which tasks conducted posttrauma can prevent intrusive memories.

Keywords: Mental imagery, emotion, imaginal exposure, imaginal extinction, hotspots, intrusive memories

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*To my sweet boys
Benjamin and Edgar*

List of Papers

This thesis is based on the studies described in the following papers, referred to in the text by Roman numerals.

- I **Hoppe, J. M.**, Holmes, E. A., & Agren, T. (2021). Exploring the neural basis of fear produced by mental imagery: Imaginal exposure in individuals fearful of spiders. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences*, 376(1817), 20190690.
- II **Hoppe J. M.**, Holmes, E. A., & Agren T. (2021). Imaginal extinction and the vividness of mental imagery: Exploring the reduction of fear within the mind's eye. Manuscript.
- III **Hoppe, J. M.**, Walldén, Y. S. E., Kanstrup, M., Singh, L., Agren, T., Holmes, E. A., & Moulds, M. L. (2021). Hotspots in the immediate aftermath of trauma – Mental Imagery of Worst Moments Highlighting Time, Space and Motion. *Consciousness and Cognition*. Submitted.

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3. Hjorth, O. R., Frick, A., Gingnell, M., **Hoppe, J. M.**, Faria, V., Hultberg, S., Alaie, I., Månsson, K. N. T., Wahlstedt, K., & Jonasson, M. (2019). Expression and co-expression of serotonin and dopamine transporters in social anxiety disorder: A multitracer positron emission tomography study. *Molecular Psychiatry*, 1–10.
4. Costache, M. E., Frick, A., Månsson, K., Engman, J., Faria, V., Hjorth, O., **Hoppe, J. M.**, Gingnell, M., Frans, Ö., Björkstrand, J., Rosén, J., Alaie, I., Åhs, F., Linnman, C., Wahlstedt, K., Tillfors, M., Marteinsdottir, I., Fredrikson, M., & Furmark, T. (2020). Higher- and lower-order personality traits and cluster subtypes in social anxiety disorder. *PLoS ONE*, 15(4).
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Contributions

The contributions of Johanna M. Hoppe to the studies were as follows:

Study I. Made substantial contributions to conception and design, acquisition of data, analyses, interpretation of data, and drafting the article.

Study II. Made substantial contributions to conception and design, acquisition of data, analyses, and interpretation of data, as well as drafting the article.

Study III. Made substantial contributions to the evolution of conceptualisation, research ideas and methodology, analyses, interpretation of data, data curation, and final draft preparation.

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Abbreviations

CBT	Cognitive behavioural therapy
CS+	Conditioned stimulus
CS-	Control stimulus
ED	Emergency department
EPT	Emotion processing theory
fMRI	Functional magnetic resonance imaging
GAD	Generalised anxiety disorder
LIWC	Linguistic inquiry and word count
MCC	Midcingulate cortex
PE	Prolonged exposure
PTSD	Posttraumatic stress disorder
RCT	Randomised controlled trial
SAD	Social anxiety disorder
SCR	Skin conductance response
SPQ	Spider Phobia Questionnaire
US	Unconditioned stimulus
VAS	Visual analogue scale
VVIQ-2	Vividness of visual mental imagery-2

Introduction

In a world where problems related to mental health constitute one of the largest causes of human suffering and societal cost (World Health Organization, 2019), it could be argued that the most important objectives of psychological research are to develop more effective, accessible and tolerable psychological treatments and explore how psychopathological conditions can be prevented (Singh et al., 2020; van Os et al., 2019). A crucial step toward these goals is to unravel the underlying mechanisms of our core psychological treatment techniques (Holmes et al., 2018) and to increase our understanding of psychological processes involved in the aetiology and maintenance of psychopathology (Holmes et al., 2018).

Imaginal exposure (Wolpe, 1958) is a widely used psychological treatment technique (Foa et al., 2019). This thesis sought to contribute to a better understanding of the mechanisms underlying imaginal exposure, with the goal of informing ways to enhance its effectiveness, accessibility and tolerability. In addition, this thesis sought to characterise emotional mental imagery (Holmes & Mathews, 2005; Lang, 1979), a core feature in many psychopathological conditions and a vehicle for emotional change in imaginal exposure (Holmes & Mathews, 2010). In this introduction, I will present the central concepts of the thesis, including what mental imagery is, the link between mental imagery, emotion and psychopathology, and how mental imagery can be used within imaginal exposure to alleviate fear. After that, I will provide an overview of the theoretical assumptions on the underlying mechanisms of exposure therapy in general, and imaginal exposure specifically. Then, I will highlight some of the gaps in our knowledge about imaginal exposure, including its neural basis. Subsequently, I will present the hypotheses on how factors within imaginal exposure (e.g., duration of exposure session) could be manipulated to improve its effectiveness, accessibility and tolerability. In the final section of the introduction, I will argue for the potential benefits of studying the characteristics and aetiology of the symptoms that imaginal exposure seeks to alleviate.

What is mental imagery?

The term *mental imagery* has been described as seeing with the mind's eye, hearing with the mind's ear and so on, referring to perception-like sensory

experiences in the absence of external sensory input (Holmes & Agren, 2020; Kosslyn et al., 2001). Mental imagery is a phenomenon that most of us experience in daily life (Ji et al., 2016). This remarkable human capacity allows us to revisit our past and simulate future events and their emotional consequences, to guide our behaviour (Lang, 1979; Moulton & Kosslyn, 2009; Schacter et al., 2008). For instance, mental imagery allows us to re-experience that trip to Rome, let us ‘try out’ how it would feel to visit Venice someday and help us decide if we would fancy having pizza or fish for dinner by “tasting” them within our minds. However, mental imagery is not always elicited deliberately; it often pops into the mind spontaneously or triggered by a cue (Iyadurai et al., 2019; Pearson & Westbrook, 2015).

Mental imagery is not restricted to visual imagery, but can include all our senses (Andrade et al., 2014). In general, people find that visual and haptic (touch) stimuli are the simplest to imagine, and that the most difficult are taste and smell (Andrade et al., 2014). Notably, we can also imagine motor activity, motion (Jeannerod, 1994), and somatic sensations such as pain (Berna et al., 2012). The vividness of mental imagery – that is, its clarity and liveliness – differs substantially between individuals (Andrade et al., 2014; Marks, 1973). While some people experience highly vivid, life-like mental imagery, some individuals cannot produce (visual) mental imagery at all (i.e., *aphantasia*; Keogh & Pearson, 2018). The vividness of mental imagery also differs from time to time and across different tasks. For instance, it is easier to imagine a familiar, concrete object than an unfamiliar, abstract one (Kosslyn et al., 2006). Also, as mental imagery production requires modality-specific working memory resources, it is harder to produce visual mental imagery while performing a visuospatially demanding task (e.g., playing Tetris) or to imagine a tune while listening to music (Baddeley & Andrade, 2000).

In line with the phenomenological experience of mental imagery, empirical evidence shows that mental imagery and perception of a stimulus elicit similar physiological responses and recruit overlapping neurocircuitry, i.e., seeing an object and imagining the same object will activate similar brain areas (Pearson, 2019). Further supporting the close link between mental imagery and perceptual processes, the vividness of mental imagery is correlated with activation and volume (Bergmann et al., 2016) of visual cortices (Cui et al., 2007; Dijkstra et al., 2017). However, while perception draws its major input from the outside world, mental imagery is formed through input from memory (Kosslyn, Thompson, & Ganis, 2006). Indeed, there is a significant overlap between the neurocircuitry involved in mental imagery and episodic memory (Schacter et al., 2008).

Why is mental imagery closely linked to emotion?

Mental imagery has been shown to have a special relation with emotion (Holmes & Mathews, 2005). Specifically, emotional mental imagery, i.e., mental imagery with emotion-provoking content, elicits emotional responses to a greater extent than verbal thought (Cuthbert et al., 2003; Holmes, Mathews, et al., 2008; Holmes & Mathews, 2005; Vrana & Lang, 1990). For example, imaging an emotion-provoking scene (e.g., falling from a cliff) elicits higher emotional responses than ‘thinking’ verbally about the same scene (Mathews et al., 2013). It has been hypothesised that the intricate relationship with perception gives mental imagery a fast-track to our emotions (for review, see Ji et al., 2016). Indeed, emotional mental imagery has been observed to provoke heightened physiological responses (Agren et al., 2017; Lang et al., 1980). However, compared with the extensive research on non-emotional imagery (Pearson, 2019), the neural underpinnings of emotional mental imagery have received less attention. Therefore, it is unclear if producing mental imagery of an emotion-provoking stimulus (i.e., a phobic stimulus) employs similar neurocircuitry as when the stimulus is encountered in real life through direct perception.

Mental imagery in psychological disorders

The capacity of mental imagery to produce lifelike experiences comes at a cost. Mental imagery enables us to re-experience distressful events in our past (e.g., trauma) over and over again. It also lets us ‘experience’ adverse events that we fear will happen in the future (e.g., losing a loved one, getting ill). Thus, mental imagery gives us access to what we fear, anywhere, at any time. We probably encounter the things we fear more frequently within our minds than we do in real life. Importantly, emotional mental imagery can also promote dysfunctional behaviour. Producing mental imagery of an event (e.g., being humiliated, attaining a goal) has been observed to influence how probable a person thinks it is that the imagined scenario will actually occur (Raune et al., 2005) and how they behave (e.g., avoids public speaking, attains the goal; Renner et al., 2019). Bearing in mind the impact that mental imagery has on emotion and behaviour, it is not surprising that mental imagery is critically involved in psychopathological conditions. For example, mental imagery fuels fear and avoidance behaviour in anxiety disorders such as social anxiety disorder (SAD; e.g., image of oneself doing something embarrassing), generalised anxiety disorder (GAD; e.g., image of being involved in a serious accident) and specific phobia (e.g., image of spider attacking; Hirsch & Holmes, 2007; Holmes & Mathews, 2010; Pratt et al., 2004). Mental imagery also amplifies craving in substance abuse (Andrade et al., 2012) and promotes mood instability and suicidal behaviour in bipolar disorder (Holmes, Geddes, et al.,

2008). Furthermore, impaired ability to produce positive mental imagery of the future contributes to maintaining depressive mood (Holmes, Lang, et al., 2008; Morina et al., 2011).

The severe impact that emotional mental imagery can have on mental health is perhaps most evident in posttraumatic stress disorder (PTSD; American Psychiatric Association, 2013). In PTSD, recurrent, involuntary mental imagery of the traumatic event can cause high emotional distress and functional impairment (Holmes et al., 2017; Iyadurai et al., 2019). This specific type of mental imagery is referred to as ‘intrusive memories’, or as ‘flashbacks’ in the most extreme form, i.e., when mental imagery of the trauma is so vivid that it is ‘as if’ reliving the traumatic event all over again (Ehlers et al., 2004; Iyadurai et al., 2019). Intrusive memories often take the form of visual mental imagery, but can also include sounds, smells, and other sensory experiences that were present during the traumatic event (e.g., Hackmann et al., 2004). The mental imagery-based nature of intrusive memories can create a strong sense of current threat and nowness.

Mental imagery not only plays a central role in the symptomatology of PTSD, but also plays a key role in its treatment. Specifically, mental imagery is used to revisit traumatic events under controlled circumstances with a treatment technique called imaginal exposure. The next section of the introduction will describe imaginal exposure and how it is used in the clinic. I will also give a basic overview of the proposed mechanisms of exposure therapy in general and what we know about imaginal exposure specifically.

Mental imagery in psychological treatments

Imaginal exposure

Imaginal exposure is a quintessential treatment technique in Cognitive Behavioural Therapy (CBT). Imaginal exposure belongs under the umbrella of exposure therapy (Craske et al., 2014; Foa & McLean, 2016). Exposure therapy entails approaching the fear-provoking stimulus (e.g., a spider) repeatedly or for prolonged durations, to form new functional associations with the stimulus (‘spiders are not dangerous’). Exposure therapy is one of our most effective psychological treatments for dysfunctional fear, including anxiety disorders, obsessive-compulsive disorder and PTSD (Craske et al., 2014; Foa & McLean, 2016; Hofmann & Smits, 2008). Exposure therapy is often performed *in vivo*, i.e., with the actual fear-provoking stimulus (e.g., a real spider). When *in vivo* exposure is not possible, mental imagery of the fear-provoking stimulus/event can be used for exposure, which is referred to as imaginal exposure. For instance, in PTSD, the patient revisits the traumatic event using mental imagery (Foa et al., 2019), and in GAD, exposure can include

mental imagery of events that the patient fears will occur in the future (e.g., losing a loved one; Dugas & Robichaud, 2007).

Mental imagery has been used for exposure since the conception of behavioural therapy (Lang, 1979; Wolpe, 1958). In the late 1950s, *Joseph Wolpe* developed one of the precursors to contemporary exposure therapy; systematic desensitisation (Wolpe, 1958). This procedure used graded exposure to mental imagery of the fear-provoking stimulus to alleviate dysfunctional fear. Based on the similar emotional responses evoked by both modalities, Wolpe proposed that mental imagery could ‘stand in’ for the perception of the fear-provoking stimulus (Wolpe, 1958). Emerging evidence indeed supports that mental imagery and perception can have interchangeable roles in the reduction of both experimentally induced (conditioned) fear (Agren et al., 2017; Reddan et al., 2018) and phobic fear (Hecker, 1990).

Imaginal exposure is generally not used as a stand-alone treatment technique. Instead, it is included in treatment protocols in conjunction with other treatment components, such as cognitive techniques and *in vivo* exposure (e.g., Dugas & Robichaud, 2007; Ehlers & Clark, 2000; Foa et al., 2019). Prolonged Exposure (PE; Foa et al., 2019) for PTSD is one of the most widely used imaginal exposure-based psychological treatment protocols. Indeed, the majority of clinical research on imaginal exposure has been conducted on PE. Hence, this treatment protocol is especially relevant to this thesis, and PE will therefore be described in more detail.

Prolonged Exposure

Prolonged Exposure is one of the gold-standard treatments for PTSD and is widely used across the globe (National Institute for Health and Care Excellence, 2018). Imaginal exposure constitutes a central component in this treatment protocol (Foa et al., 2019). During imaginal exposure, the patient is asked to revisit the traumatic event by recounting the event in the present tense while imagining the trauma. The patient is encouraged to imagine the event as *vividly* as possible, to promote emotional engagement. PE consists of multiple 90-minute sessions, including about 40–60 minutes of imaginal exposure followed by 15–20 minutes of verbal processing of trauma-related thoughts and feelings. In addition, *in vivo* exposure to trauma-relevant situations is conducted between sessions as ‘homework assignments’ (Foa et al., 2019).

Though PE is one of our most effective treatments for PTSD, treatment development is needed to improve its effectiveness, accessibility and tolerability. A substantial number of patients experience residual symptoms after treatment with PE (Bradley et al., 2005; Powers et al., 2010). In addition, PE is inaccessible to many patients (McLean & Foa, 2013; Najavits, 2015) for reasons such as that treatment requires a highly trained therapist to deliver it. Also, the long 90-minute sessions, often conducted twice per week, make treatment time-consuming for health services providing treatment and

challenging for patients to fit into daily life (e.g., work, studies, childcare etc.). Furthermore, as in many PTSD treatments, dropout rates are high (25–50%; for review, see Zhou et al., 2020) for several reasons, including stigma around mental disorders and because treatment can initially cause increased levels of distress (Eftekhari et al., 2020; Imel et al., 2013; Najavits, 2015; Niles et al., 2018). As will become clearer later in the introduction, some of the theoretical assumptions underlying PE have been challenged. Challenges to the assumptions on the mechanisms of PE open opportunities for treatment development. A good way to understand the theoretical assumptions underlying imaginal exposure and PE is to get acquainted with the experimental models from which exposure therapy was derived.

The roots of exposure therapy

Exposure therapy, including imaginal exposure, has its roots in learning theory (Pavlov, 1927). Learning theory stems from experimental studies using the fear conditioning paradigm – the experimental paradigm most widely used to study fear to this day (Lonsdorf et al., 2017). This paradigm includes an experimental model for how fear is acquired (i.e., fear acquisition), how fear can be attenuated (i.e., fear extinction) and a model for return of fear (cf. relapse). Exposure therapy is, in essence, derived from fear extinction. Therefore, extinction is by many regarded as an experimental analogue of exposure therapy (Dunsmoor et al., 2015).

The paradigm of fear conditioning usually consists of three phases. During fear acquisition, a neutral stimulus is transformed into a conditioned stimulus (CS+) by repeated pairings between the neutral stimulus and an aversive unconditioned stimulus (US; e.g., electric shock; Maren, 2001; Pavlov, 1927). If fear acquisition occurs, subsequent presentation of the CS+ alone will evoke conditioned fear responses, reflecting the formation of a ‘fear memory’ consisting of the association between the CS+ and the aversive outcome (CS+ - US). In humans, skin conductance responses (SCRs) are commonly used as an index for conditioned fear responses (Boucsein, 2012). In the second phase, conditioned fear responses to the CS+ are reduced through an extinction procedure, in which the CS+ is presented repeatedly, but with the aversive outcome withheld (e.g., no electric shocks). During extinction, the participant learns that the CS+ no longer signals threat. Notably, extinction does not ‘erase’ the fear memory (CS+ - US). Instead, it creates a competing ‘extinction’ memory (CS+ - no US) that inhibits the fear memory from being expressed (Bouton, 1993). The persistence of the fear memory is evidenced by the transient and context-specific effects of extinction (Rachman, 1989). Conditioned fear responses tend to return after successful extinction if the CS+ is presented in a different context than where extinction took place (Bouton, 1993), after an unexpected re-encounter with the US (Rescorla & Heth, 1975) or through the mere passage of time (Quirk, 2002). Therefore, the effects of

extinction are typically evaluated with one or more tests of return of fear to assess the effects of extinction in a different context (renewal), after re-exposure to the US (reinstatement), or the passage of time (spontaneous recovery; for review see Lonsdorf et al., 2017).

Consistent with the proposed mechanisms of extinction, exposure therapy is believed to allow the patient to learn that the aversive outcome erroneously expected by the patient did not occur (e.g., ‘being attacked by the spider’ or ‘not being able to handle the fear elicited by exposure to the spider’; Craske, 2015). Mirroring the transient effects of extinction in the laboratory, relapse after exposure therapy is common (Vervliet et al., 2013). For instance, fear of spiders can some times return if a spider is encountered outside of the therapy room (renewal) or if the patient experiences anxiety not related to spider fear (reinstatement). Fear can also return spontaneously as time passes (spontaneous recovery). Thus, exposure therapy is also considered to form a competing ‘extinction memory’ rather than to abolish the fear memory (for review, see Craske, 2015).

The inhibitory learning approach of extinction and Emotion Processing Theory (EPT) are two influential models in clinical exposure therapy. Both models have their roots in learning theory, but prescribe somewhat different exposure procedures to maximise the long-term effects of exposure therapy. The inhibitory learning approach considers maximising expectancy violation (i.e., prediction error) to be a key factor for long-term fear reduction and advocates the use of exposure strategies that increase expectancy violation and procedures to counteract the return of fear (Craske, 2015; Craske et al., 2008, 2014). For its part, EPT proposes that successful treatment requires (1) emotional arousal at the beginning of the session, (2) reduction of emotional responses from start to end of the exposure session (within-session fear reduction) and (3) reduction of peak emotion from one session to the next (between-session fear reduction; Foa et al., 1986). Emotion Processing Theory constitutes the theoretical framework underlying PE. That is, PE is designed to maximise treatment effects based on the theoretical assumptions of EPT (Foa et al., 2019), which will now be reviewed.

Emotion Processing Theory

The development of EPT was highly influenced by the bio-informational theory of emotional imagery postulated by Peter Lang (1979). Inspired by the work of Wolpe (1958), Lang investigated the relationship between mental imagery, emotion, and physiological arousal. The goal of this enterprise was to find ways to improve the effects of exposure therapy (Lang, 1977). He proposed that emotional mental imagery was not merely an internal ‘image’ of the actual fear stimulus (e.g., a spider). Rather, Lang proposed that emotional mental imagery activated an associative network stored in long-term memory, including information about stimulus characteristics (e.g., shape, colour, size

of a spider), physiological and behavioural responses associated with encountering the stimulus (e.g., racing heart, urge to escape), and semantic information about their meaning (e.g., danger; Ji et al., 2016; Lang, 1979). Lang suggested that phobic fear consisted of a particular type of associative network that evoked relatively stable emotional responses which needed few matching inputs to be activated (seeing a spider, racing heart), referred to as a ‘fear structure’. Lang suggested that for exposure to be effective, the fear structure needed to be activated first (retrieved from memory). Activation was signalled by increased physiological arousal and imagery vividness during exposure (Lang et al., 1980).

Consistent with the bio-informational theory, EPT proposes that psychopathological conditions, such as PTSD, are maintained by dysfunctional fear structures (e.g., harmless stimuli associated with trauma trigger dysfunctional responses, such as heightened physiological arousal, avoidance behaviour, and sense of current threat; Foa et al., 1986). Exposure therapy (both *in vivo* and imaginal exposure) is suggested to be effective as it allows *emotional processing* of the dysfunctional ‘fear structure’ by promoting (1) activation of the fear structure (confrontation with a spider) and (2) providing corrective information (e.g., ‘I can handle seeing a spider without losing my mind’). Emotional processing is suggested to be indicated by a) activation of the fear structure, apparent through both subjective and objective (e.g., physiological arousal) measures of fear, (b) reduction of fear from the start to the end of the exposure session (within-session fear reduction), and (c) a reduction of peak fear level from one exposure session to the next (between-session fear reduction; for review see Foa & McLean, 2016).

To promote emotional processing, PE incorporates these assumptions in the following ways. First, during imaginal exposure, the patient is encouraged to imagine the traumatic event as vividly as possible, to increase emotional engagement with the memory (i.e., ‘activation’). Second, PE prescribes long sessions (i.e., 90 minutes) to ensure enough time for fear to subside sufficiently within each session (within-session fear reduction). As noted earlier in the introduction, the long sessions prescribed in PE negatively impact on the accessibility of this gold-standard treatment. Also, producing vivid mental imagery of trauma can be overwhelmingly distressful for some patients.

How can we improve imaginal exposure?

Challenging Emotional Processing Theory

The assumptions underlying PE have been challenged (e.g., Craske et al., 2008, 2014, 2015). For instance, Michelle Craske, a central advocate of the inhibitory learning approach, argues that extinction learning is primarily driven by expectancy violation and not by the pattern of emotional responses during the exposure session, as suggested by EPT. Supporting this notion,

research shows that long-term fear reduction is not reliably predicted by heightened emotional arousal at the start of the exposure session (i.e., activation) or by within-session fear reduction (for review, see Craske et al., 2008). Challenging the assumptions underlying EPT implicitly also challenges the need for PE to fit these assumptions. For example, less emphasis on within-session fear reduction takes away the need to schedule long exposure sessions to ensure that there is sufficient time for fear to subside during the sessions. However, it should be noted that the vast majority of research on exposure therapy, including the underlying assumptions of the inhibitory learning approach used to challenge EPT, has been conducted using exposure to observable, *in vivo* stimuli (e.g., visual/auditory; Mertens et al., 2020).

Identifying mechanisms specifically linked to imaginal exposure

Though *in vivo* and imaginal exposure likely employ similar mechanisms of change, there are certain aspects of imaginal exposure that cannot be captured by studying *in vivo* stimuli only. For instance, research on *in vivo* stimuli cannot inform us about how treatment outcome is affected by the quality (e.g., vividness) of mental imagery produced during imaginal exposure or by the characteristics of mental imagery used for exposure (e.g., prolonged vs. brief). Although the mechanisms of imaginal exposure have been studied in the context of PE (e.g., Helpman et al., 2016; Mota et al., 2015; Rauch et al., 2004), it is difficult in these studies to isolate the mechanism specifically associated with imaginal exposure relative to the other treatment components. I argue that improving imaginal exposure requires that we study imaginal exposure specifically. This includes using experimental models of exposure that use mental imagery rather than *in vivo* stimuli and that we study imaginal exposure without additional treatment protocols. Studying imaginal exposure specifically, rather than deriving knowledge about its mechanisms from studies on *in vivo* exposure and PE, would help us build a basic understanding of how fear is reduced ‘within the mind’. For instance, one of the missing pieces in our understanding of imaginal exposure is its neural basis.

What is the neural basis of imaginal exposure?

Knowledge of the neural basis of imaginal exposure is limited. Akin to clinical studies, most neuroimaging studies of imaginal exposure evaluate the effects on brain activity/volume associated with undergoing PE (e.g., Helpman, Marin, et al., 2016; Helpman, Papini, et al., 2016). As previously stated, this approach complicates interpretations of the link between findings and imaginal exposure relative to other treatment components. However, a pioneering functional magnetic resonance imaging (fMRI) study recently showed evidence that imaginal extinction (exposure to mental imagery of a conditioned stimulus) and *in vivo* extinction (exposure to the percept of conditioned

stimulus) may recruit similar neurocircuitry to reduce *conditioned fear* (Reddan et al., 2018). Despite this important finding, the neural basis of imaginal exposure to *phobic fear* has not been systematically investigated.

Knowledge of the neural mechanisms of imaginal exposure could perhaps be derived from symptom provocation studies on PTSD. However, results are inconsistent across these studies, which may in part be due to methodological limitations (e.g., Britton et al., 2005; Lanius et al., 2007; Shin et al., 2004). These neuroimaging studies use ‘script-driven imagery’. It entails constructing a personalised trauma script for each participant, which is used to invoke mental imagery of their specific trauma memory during brain imaging. The methodological limitations of using personalised scripts primarily concern that they complicate the construction of adequate control stimuli (e.g., combat situation vs. brushing one’s teeth). The tasks conducted during brain imaging will also vary across participants (e.g., war trauma vs. traffic accident). As the control condition (i.e., control stimuli) is key for the results of brain activation studies (Davis & Poldrack, 2013), script-driven imagery reduces the precision of findings. Another limitation is the use of mental imagery of long duration (30–60 s). Mental imagery can, in general, only be kept in mind for less than a second before it needs to be regenerated (Pearson et al., 2013). Thus producing mental imagery during long duration can be cognitively demanding, and neuroimaging results may be influenced by individual differences in attentional resources.

How can we measure brain activity during imaginal exposure?

One of the aims of the thesis was to explore the neural underpinnings of imaginal exposure to phobic stimuli. However, the inherently private nature of mental imagery makes it difficult to study. Due to the limitations of the methodology used in symptom-provocation studies in PTSD described above, a novel experimental analogue of imaginal exposure adapted for brain imaging was developed (Study I, Hoppe et al., 2021). The procedure was designed to more accurately capture neural activity tied to emotional processing during imaginal exposure. In addition, the procedure was designed to mirror clinical exposure therapy as closely as possible. The two key features of the novel procedure were (1) the use of non-personalised imagery scripts and (2) exposure to brief ‘flashpoint mental imagery’. The term ‘flashpoint mental imagery’ refers to evoking mental imagery of emotion-provoking content for a brief period (about 7 seconds). The rationale for using non-personalised imagery scripts was that it helps reduce variance between individuals by ensuring the same task across participants. Flashpoint mental imagery had several advantages compared with mental imagery of prolonged duration. First, it is less mentally taxing, making results less dependent on participants’ attentional resources. It also facilitates the construction of better-matched control stimuli. For example, for spider phobia, imagery including a spider can be contrasted

with identical imagery of matching complexity where the spider is replaced with a non-fear-provoking object. Moreover, brief mental imagery allows the inclusion of a greater number of trials in a brain imaging session and facilitates ‘repeated exposure’, a key mechanism of extinction. Furthermore, mirroring exposure in the clinic, the procedure uses verbal instructions to prompt imagery and includes graded exposure (i.e., exposure to progressively more fear-provoking stimuli). Notably, the clinical features of the procedure provided an opportunity to explore if a 10 minutes session of imaginal exposure to flashpoint mental imagery could influence fear responses.

Opportunities for treatment development

Can a 10-min session of imaginal exposure reduce fear?

One opportunity for treatment development concerns the duration of imaginal exposure. PE includes long sessions (90 min) to ensure sufficient time for fear to subside within the sessions (Foa et al., 2019). However, the need for within-session fear reduction for treatment success has been a subject for debate (Baker et al., 2010; Craske et al., 2008). Indeed, in recent years, Edna Foa, the creator of EPT, has acknowledged that there is limited evidence supporting that within-session fear reduction is required for treatment to be successful (Foa & McLean, 2016). Consequently, the duration of exposure sessions could potentially be shortened without compromising treatment effects. Preliminary studies show that PE using 20–30 min sessions (Nacasch et al., 2015; Van Minnen & Foa, 2006) of imaginal exposure produced effects comparable to those of standard PE (about 40-min sessions; Foa et al., 2019). Reducing the duration of imaginal exposure sessions could help increase the accessibility of PE, by making treatment less time-consuming for both healthcare services and patients.

The experimental procedure used to study neural activity during imaginal exposure provided an opportunity to explore if a 10-minute session of imaginal exposure to flashpoint mental imagery (without other treatment components) could influence fear responses. Moreover, the procedure could elucidate if flashpoint mental imagery could reduce fear. Flashpoint mental imagery could have clinical implications. For instance, it is less demanding and could help make treatment more accessible for individuals who find it hard to produce mental imagery of long durations.

What is the link between the vividness of mental imagery and the reduction of fear?

Another opportunity for treatment development is to study the impact that imagery vividness during imaginal exposure has on fear reduction.

According to EPT, activation of the ‘fear structure’ is required for exposure to be effective. The vividness of mental imagery during exposure has been suggested to promote emotional engagement and indicate the level of activation of the fear structure (Foa et al., 1986; Rauch et al., 2004). Therefore, patients undergoing imaginal exposure are encouraged to imagine the fear-provoking object or event (e.g., trauma memory) as vividly as possible (Foa et al., 2019). However, clinical studies on PE have shown mixed results (Mota et al., 2015; Rauch et al., 2004). Specifically, in these studies, imagery vividness during exposure has been found to be positively associated with fear reduction within the session. On the other hand, limited evidence was found that vividness was associated with the reduction of fear in the long-term – i.e., overall treatment outcome. Once again, as these studies are conducted on PE, the interpretation of findings is complicated by the inclusion of other treatment components.

Understanding to what extent high vividness is required for imaginal exposure to be effective could have significant clinical implications. High vividness during imaginal exposure is associated with higher levels of subjective distress (Mota et al., 2015; Rauch et al., 2004). If producing highly vivid mental imagery is not required to accrue benefits from imaginal exposure, strategies to reduce vividness could potentially be used to lower distress (Deepröse et al., 2012; Kavanagh et al., 2001) and thereby make imaginal exposure tolerable for more patients. If high vividness promotes the effects of imaginal exposure, strategies to increase vividness could be explored. Also, knowledge of the link between vividness and fear reduction could help us predict which patients are most likely to benefit from imaginal exposure. As mentioned early in the introduction, imagery vividness can be manipulated, as it depends on several factors (e.g., memory, attentional resources, imagery instructions; Baddeley & Andrade, 2000; Kosslyn, 1975; Lang et al., 1980). For instance, the vividness of visual mental imagery can be reduced by performing a concurrent task that loads the visuospatial working memory, such as playing Tetris (Bywaters et al., 2004; Engelhard et al., 2010; Kavanagh et al., 2001). Moreover, vividness can be increased by adapting the instructions given to prompt mental imagery (Lang et al., 1980).

Can we use the fear conditioning paradigm to study imaginal exposure?

To further our knowledge about the role of imagery vividness in imaginal exposure, there is a need for controlled studies, pinpointing the mechanisms driving the effects of imaginal exposure separate from other treatment components. Understanding the mechanism of *in vivo* exposure has been facilitated by the fear conditioning paradigm. However, established methods to study imaginal exposure have been lacking (Mertens et al., 2020). Imaginal

extinction is a novel experimental analogue of imaginal exposure (Agren et al., 2017). As the name suggests, imaginal extinction mimics the *in vivo* extinction procedure, except that conditioned fear is reduced through exposure to *mental imagery* of the conditioned stimulus rather than through the perception of the conditioned stimulus. In line with imaginal exposure in the clinic, imaginal extinction uses verbal instructions to prompt mental imagery. Just as *in vivo* extinction can be used to test hypotheses associated with *in vivo* exposure, imaginal extinction provides a tool to explore factors that may influence fear reduction in imaginal exposure, such as imagery vividness.

Characterising the building blocks of intrusive mental imagery after trauma

In this introduction, I have suggested that using experimental models of imaginal exposure could help us disentangle its mechanisms and find ways to improve treatment. However, treatment development could also be guided by studying the characteristics and aetiology of the symptoms that the treatment aims to alleviate. Using qualitative methods to look closer into the characteristics of symptoms could reveal information that is not easily captured in clinical and experimental studies, which often focus on quantitative outcome measures. Such information could help us generate hypotheses for how to treat symptoms, help us understand their function, and perhaps even inform us as to how to prevent them.

One of the core PTSD symptoms that imaginal exposure and PE seek to alleviate is the recurrent intrusive memories (mental imagery) of trauma. Intrusive memories are closely linked to the ‘hotspots’ (i.e., ‘worst moments’) of the trauma memory (Grey & Holmes, 2008; Holmes et al., 2005). For most PTSD patients, the contents of their intrusive memories coincide with their hotspots (e.g., 83% overlap, Grey & Holmes, 2008; 78%, Holmes, Grey, et al., 2005). Accordingly, hotspots are important treatment targets in PE and other evidence-based trauma-focused therapies for PTSD (National Institute for Health and Care Excellence, 2018). Given the close link between hotspots and intrusive memories (Grey & Holmes, 2008; Holmes et al., 2005), and the key role of hotspots in PTSD treatments (Ehlers & Clark, 2000; Foa et al., 2019; Shapiro, 1996), surprisingly few studies have investigated their characteristics and contents (Grey & Holmes, 2008; Harris & Ayers, 2012; Holmes et al., 2005; Jelinek et al., 2010; Nijdam et al., 2013). Importantly, no study has investigated the characteristics of hotspots close to the time of their encoding, i.e., in the immediate aftermath of trauma. Exploring the characteristics of newly formed hotspots could potentially inform treatment development and give us clues to how intrusive memories are formed and how to prevent them.

Aims

There is an urgent need to develop more effective, accessible, and tolerable psychological treatments and increase our understanding of psychological processes involved in the aetiology and maintenance of psychopathological conditions. Accordingly, this thesis aimed to increase our knowledge of the underlying mechanisms of imaginal exposure and investigate potential ways to enhance its effectiveness, accessibility, and tolerability. This thesis also aimed to improve our understanding of emotional mental imagery, in particular mental imagery in the immediate aftermath of trauma, which could provide insights into how intrusive memories are formed and how to prevent them.

- I Study I investigated the neural underpinnings of imaginal exposure in individuals fearful of spiders. This study also explored if a 10-minute session of repeated exposure to flashpoint mental imagery of phobic stimuli (spiders) had an effect on fear responses at the subjective and physiological (SCR) level.
- II Study II used an experimental analogue of imaginal exposure (imaginal extinction) to investigate the link between the vividness of mental imagery and the reduction of conditioned fear as measured with skin conductance responses.
- III Study III explored the frequency, sensory-perceptual features and other mental imagery-related content in hotspots in memories of traumatic events as reported within the first hours by trauma-exposed individuals waiting in an emergency department (ED).

Methods

Functional magnetic resonance imaging

Functional magnetic resonance imaging is a non-invasive technology that can be used to measure brain function. Neural activity is associated with inflow of oxygenated blood. Through fMRI, changes in blood flow within the brain during a specific task are measured, providing an indirect measure of neural activity. The fMRI technology takes advantage of the fact that deoxygenated (oxygen-poor) and oxygenated (oxygen-rich) blood have different magnetic properties. The strength of the fMRI signal, referred to as the blood oxygen-dependent signal (BOLD signal), depends on the relative rate of oxygenated to deoxygenated blood.

Study I used fMRI to study brain activity related to emotional processing during imaginal exposure. Isolating brain activity (BOLD signal) associated with a specific task requires that the BOLD signal of interest is contrasted to an adequate control condition. Thus, in Study I, the BOLD signal during mental imagery of phobic stimuli was contrasted to the BOLD signal during neutral mental imagery to derive brain activity associated with fear created within the mind's eye, controlling for brain activity related to the production of mental imagery per se.

Skin conductance responses

Skin conductance response is one of the most widely used physiological measures to assess fear learning and extinction in humans (Lonsdorf et al., 2017). Specifically, in fear research, SCR is used to derive the activation of the sympathetic nervous system triggered by fear-provoking events/stimuli (Sequeira et al., 2009). The general principles of this method rely on the link between the activation of the sympathetic nervous system and increased secretion from the eccrine sweat glands. When facing a real or perceived threatening situation, the sympathetic nervous system mediates an increase in physiological arousal, preparing the body for fight or flight, which includes increased secretion of sweat. As sweat improves the electrical conductivity of the skin, fluctuations in sweat secretion can be detected (Boucsein, 2012). In practice, SCRs are measured by attaching electrodes to body areas rich in eccrine sweat glands, such as the palms of the hands, and transmitting a weak

electric current, which is used to measure phasic shifts in response to specific emotional events (e.g., presentation of a conditioned stimulus). Skin conductance responses were used to index physiological fear responses in Study I and Study II.

Qualitative coding

Sensory-perceptual features and contents

Study III used qualitative coding to explore the sensory-perceptual features and other contents of hotspots. A novel coding frame was developed using a mixture of inductive and deductive coding. Deductive (theory-driven) categories were designed to capture imagery-related features in hotspots and other contents linked to intrusive memories. These categories included sensory-perceptual features (e.g., visual, auditory, tactile), the presence of threat signals and narrative cohesiveness. Inductive (data-driven) categories were created based on the results from linguistic analysis and bottom-up reading of hotspot data. Such categories included motion features and valence. The hotspot coding frame is presented in the Supplementary materials of Paper III. Hotspots were coded based on the presence (1) or absence (0) of the sensory-perceptual feature or other contents (e.g., if a hotspot included visual features, the category visual feature was coded as 1).

Emotional and cognitive themes

Emotional and cognitive themes in hotspots were coded using an existing coding frame developed by Holmes et al. (2005). Emotional themes were coded using the following categories: *anger*, *fear*, *disgust*, *sadness*, *happiness*, *shame*, *guilt*, *surprise*, *helplessness*, *horror* and the state of *dissociation* (although not strictly an emotion). If a participant reported feeling trapped, for instance, this was coded under ‘helplessness’; if they used the word ‘terrified’, it was coded under ‘fear’. Cognitions were similarly coded based on the existing coding frame (Holmes et al., 2005), which consisted of seven overarching *cognitive themes*, including 21 coding categories: (1) *uncertain threat* (unease, confusion, realisation of a nonspecific threat, ongoing threat), (2) *general threat of injury and death* (self dying, self will die, self injured, self will be injured, death or injury of others), (3) *control and reasoning* (interpersonal reasoning, planning, revenge/injustice), (4) *consequences* (consequences, relief, realisation after), (5) *abandonment* (let down by others, outrage), (6) *esteem* (self-blame/criticism), and (7) *cognitive avoidance* (disbelief, dissociation). Hotspots were coded based on the presence (1) or absence (0) of emotion and cognitive themes.

Linguistic analysis

Study III used linguistic analysis to characterise the contents of hotspots. Linguistic analysis was conducted using the Linguistic Inquiry and Word Count software (LIWC 2015 for Windows, Austin, TX; Pennebaker et al., 2015; Tausczik & Pennebaker, 2010). The LIWC software reads the source data (e.g., hotspot descriptions), word by word (e.g., ‘sad’), and categorises them into predefined *word categories* (e.g., ‘affective processes’) and subcategories (e.g., ‘negative affect’). Each *word category* has a dictionary of words associated with the specific construct, which source data is matched with (‘sad’ is included in the dictionary of the word category ‘affective processes’). The output of LIWC is the percentage of words within the source data matching each *word category*. Individual words can be included in several categories (Tausczik & Pennebaker, 2010).

Self-reported measures

Vividness

Task-specific vividness ratings

Task-specific vividness, i.e., the vividness of mental imagery during a specific task, was assessed using a visual analogue scale (VAS) ranging from 1–5. The VAS for task-specific vividness in Study I used the anchor points specified in Holmes & Mathews (2005; Figure 1a). In Study II, the anchor points were updated to be consistent with the anchor points of the vividness of visual mental imagery-2 (VVIQ-2), which is one of the most used scales to assess trait vividness of visual mental imagery (Marks, 1995; Figure 1b).

a) Study I

1	2	3	4	5
Not vivid at all	Slightly vivid	Somewhat vivid	Very vivid	Extremely vivid

b) Study II

1	2	3	4	5
No image at all (only ‘knowing’ that you are thinking of the object)	Vague and dim	Moderately clear and vivid	Clear and reasonably vivid	Perfectly clear and as vivid as normal vision

Figure 1. Task-specific vividness rating scales used in Study I and Study II. a) The vividness scale used in Study I was adapted from Holmes & Mathews (2005). b) The vividness scale used in Study II included the same anchor points as the VVIQ-2 (Marks, 1995).

Measures of trait vividness

Vividness of Visual Mental Imagery-2

The VVIQ-2 is a 16-item self-rated questionnaire that measures the general ability to produce vivid visual mental imagery (i.e., trait-vividness; Marks, 1995; Marks, 1973). Vividness is rated on a 5-point Likert scale with anchor points ranging from 1 (‘no image at all, you only “knowing” that you are thinking of an object’) to 5 (‘perfectly clear and as vivid as normal vision’). The VVIQ-2 has good internal consistency ($\alpha = .85-.91$) and acceptable test-retest reliability ($r = .74$). The VVIQ-2 was translated into Swedish by JMH and back-translated into English by EAH.

Subjective fear

Task-specific subjective fear

Subjective fear during experimental procedures (Study I and Study II) was rated using a VAS, with anchor points ranging from 0 (no fear at all) to 100 (extreme fear).

The Spider Phobia Questionnaire

The Spider Phobia Questionnaire (SPQ; Klorman et al., 1974) was used to select participants with significant spider fear during recruitment for Study I.

The SPQ is a 31-item self-rated questionnaire. Items are scored as either ‘true’ or ‘false’ (i.e., max score = 31). The SPQ has high internal consistency ($\alpha = .90$) and test-retest reliability ($r = .87$; Muris & Merckelbach, 1996). In a Swedish population, mean SPQ score was 23.8 ($SD = 3.5$) in spider phobics and 3.8 ($SD = 5.0$) in non-phobic students (Fredrikson, 1983).

Hotspots sheet

Hotspot data in Study III were collected in the context of a pilot randomised controlled trial (RCT) conducted on trauma-exposed patients in an ED (Kanstrup et al., 2021). Hotspots were collected using a brief interview technique (Iyadurai et al., 2018; Kanstrup et al., 2021). Specifically, participants were asked to briefly mention their worst moment images of the traumatic event for which they were admitted to the ED (e.g., ‘seeing the lorry coming towards me’). Hotspots were described by participants verbally and written down by the researcher on a sheet of paper (‘hotspots’ sheet; Kanstrup et al., 2021) visible to the participant, so they could verify the written descriptions. The researcher asked if there were any additional hotspots, one at a time, until the participant confirmed they did not have any more hotspots. Only participants in the intervention group provided hotspot data.

Diary examples

Hotspot data (described above) constituted the primary data in Study III. However, a larger number of participants in the RCT (i.e., from both the intervention and control groups) provided examples of intrusive memories and hotspots (referred to as *diary examples*) in an intrusive memory diary. These data were analysed as a convergent measure for the hotspot data. Diary examples were provided by participants in accordance with the standardised instructions on how to fill out an intrusive memory diary that would be used to monitor intrusive memories in the overarching RCT (i.e., in Kanstrup et al., 2021). Participants were asked about both intrusive memories and hotspots. The reason for doing this was that some people begin experiencing intrusive memories in the first hours after trauma, while for others, trauma memories (e.g., hotspots) may start to intrude later on. It was not recorded if a diary example constituted an intrusive memory or/and a hotspot, precluding the possibility to distinguish between them.

Ethical statement

Ethical approvals were granted by the Swedish Ethical Review Authority for all included studies (Study I: EPN dnr: 2017/419; Study II: dnr 2019-00524). Study III was drawn from data collected in a pilot RCT and ethical approval

was embedded in the approval of the overarching study (EPN dnr: 2017-2215-31, study dnr: 2017-4750, updates 2018/416-32, 2018/1435-32, 2018/2150-32 and 2019-01328). For Study III, the Karolinska Trial Alliance monitored the RCT for adherence to good clinical practice. Written and informed consent was obtained in all three studies.

Open science

To promote transparency and contribute to future research, the data analysed in the studies included in this thesis are (Study I) or will be openly available upon publication at Open Science Framework (Study I: <https://osf.io/6dc5h/>; Study II: <https://osf.io/r4jac/>; Study III: <https://osf.io/a3vyb/>).

Summary of studies

Study I

*Exploring the neural basis of fear produced by mental imagery:
Imaginal exposure in individuals fearful of spiders*

Background and aim

Imaginal exposure is a widely used psychological treatment technique and a key component in evidence-based treatments for PTSD (National Institute for Health and Care Excellence, 2018). Still, treatment development is needed (Bradley et al., 2005; Powers et al., 2010). A better understanding of the mechanisms underlying imaginal exposure could inform ways to optimise its effects and make treatment more accessible. Study I aimed to explore the neural basis of imaginal exposure using fMRI and measures of SCRs. To this end, a novel experimental procedure was developed. The procedure consisted of repeated exposures to brief instances of mental imagery of phobic (spiders) and neutral (gloves) stimuli.

The secondary aim of the study concerned the duration of the exposure session. Imaginal exposure in the clinic is often of long duration (e.g., 40 min), making treatment time-consuming for both therapists and patients and thus less accessible. If the duration of imaginal exposure could be shortened, it could make treatment more accessible. The clinical features of the experimental procedure provided an opportunity to explore if a brief imaginal exposure session (without other treatment components typical of clinical protocols) could influence fear responses. Therefore, the secondary aim was to evaluate possible lasting effects of the experimental procedure (i.e., 10 min of repeated exposure to flashpoint mental imagery) on participants' fear responses.

Methods

Thirty participants (age: $M = 24.0$ years, $SD = 5.6$ years; 8 men) fearful of spiders (≥ 19 on the SPQ; Klorman et al., 1974), underwent the experimental procedure, which included repeated exposure to flashpoint mental imagery of phobic (spiders) and neutral (gloves) stimuli. Participant characteristics are

presented in Table 1. Functional magnetic resonance imaging was used to assess neural activity and SCRs to measure physiological arousal. The study used a within-subject experimental design, in which each participant performed the experimental procedure twice, approximately nine days apart ($M = 9.5$, $SD = 1.8$ days). The first session was conducted at Uppsala University Hospital, where both fMRI data and SCRs were measured throughout the procedure. Session 2 was carried out at the Department of Psychology, Uppsala University, where SCRs, but no fMRI data, were collected (Figure 2). The experimental procedure consisted of graded exposure (i.e., exposure to progressively more fear-provoking stimuli) to brief ‘flashpoint’ mental imagery (7 s; Figure 3) of 13 different situations, including spiders, interleaved with exposure to 13 corresponding situations including a neutral stimulus (a glove; e.g., phobic: ‘see a spider in front of you’; neutral: ‘see a glove in front of you’; Table 2). Recorded verbal instructions prompted mental imagery. Analyses of brain data focused on contrasting phobic mental imagery to neutral mental imagery (phobic > neutral), pinpointing brain activity associated with fear produced by mental imagery.

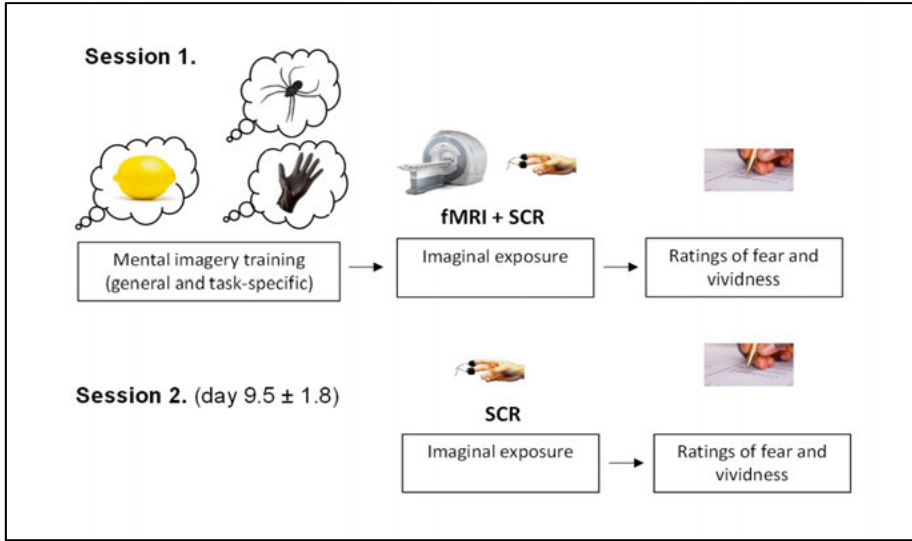


Figure 2. Overview of Study I, including methods used to assess phobic imagery in each session.

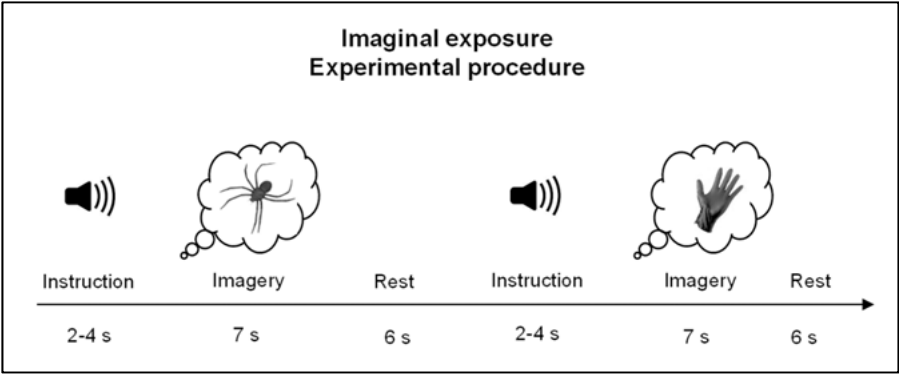


Figure 3. Overview of the experimental procedure.

Table 1. Participant characteristics

	Participants (N=30)		Women (N=22)		Men (N=8)	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Age	24.0	5.6	24.7	6.0	22.0	3.5
SPQ	23.5	2.7	23.2	2.6	24.2	3.0
STAI-T	34.8	5.7	34.9	5.1	34.9	7.7
Psi-Q	7.6	1.0	7.6	1.0	7.8	1.1

Note. SPQ: Spider Phobia Questionnaire; STAI-T: Spielberger State-Trait Anxiety Inventory; Psi-Q: Plymouth Sensory Imagery Questionnaire. No significant gender differences were observed on any measures (all $p > .05$).

Table 2. Situations used for mental imagery production during the procedure

Stimulus category		
Trial	Phobic	Neutral
1	See a spider in front of you	See a glove in front of you
2	A spider is climbing down a thread	A glove is hanging off a hook
3	Look carefully at a spider's eyes and teeth	Look carefully at the texture of a glove
4	Look carefully at a spider's legs	Look carefully at the thumb of a glove
5	You touch a spider	You pick up a glove
6	A spider moves towards you	Someone hands you a glove
7	A spider is laying eggs	Sand is poured from a glove
8	A spider jumps towards you	Someone throws a glove at you
9	A spider is crawling over your hand	A glove is pulled over your hand
10	It is raining spiders	It is raining gloves
11	A spider crawls in under your shirt	Someone puts a glove under your shirt
12	A tarantula is eating a mouse	A mouse is crawling into a glove
13	A spider is crawling into your ear	A glove is placed on your ear

Note. The situations have been translated from Swedish.

Results

Mental imagery of phobic stimuli (spiders), compared with neutral stimuli, activated more emotion-processing brain regions, including the amygdala, midcingulate cortex (MCC) and insula (Figure 4, Table 3). Subjective fear and SCRs were significantly higher during mental imagery of phobic stimuli compared with neutral stimuli, in both sessions. Subjective fear and SCRs to mental imagery of phobic stimuli were significantly reduced from session 1 to session 2 (Figure 5).

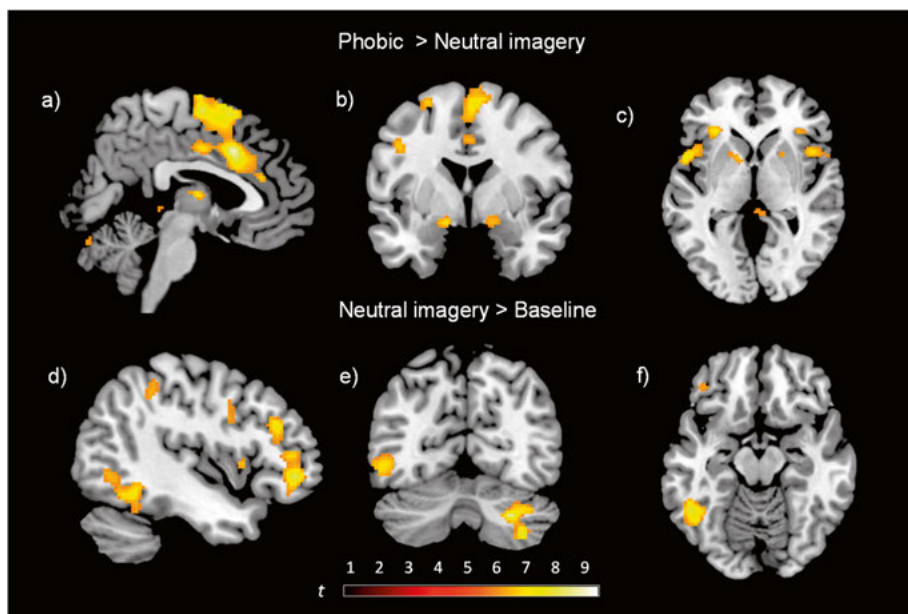


Figure 4. Brain activity during mental imagery of phobic stimuli. Images show results from whole brain analysis overlaid on a standard brain. Images are displayed at $P < .05$ family-wise error-corrected statistical threshold. The top panel depicts increased activity during imaginal exposure to phobic stimuli (phobic > neutral imagery) including in **a)** the left midcingulate gyrus, supplementary motor area, **b)** the bilateral amygdala, and **c)** the bilateral insula. The bottom panel shows activity in areas associated with mental imagery production (neutral imagery > baseline) including **d)** the left dorsolateral and orbitofrontal cortex, **e)** the right cerebellum, and **f)** the left inferior temporal gyrus.

Table 3. Neural activity during exposure to phobic imagery (phobic > neutral imagery)

	MNI coordinates			<i>p</i> FWE	<i>Z</i>	Voxels
	x	y	z			
Frontal lobe						
Middle frontal gyrus L	-30	46	18	.003	5.31	91
	-32	32	36	.030	4.76	5
Inferior frontal gyrus L (triangular)	-42	12	24	.009	5.03	64
Precentral R	42	-6	44	.014	4.94	26
Precentral L	-36	-10	50	.002	5.34	147
	-26	0	64	.007	5.10	44
Limbic areas						
Midcingulate cortex L (extending into supplementary motor area)	-4	16	36	<.001	6.38	1883
Insula L	-30	24	8	<.001	6.12	488
Insula R	46	12	-2	.003	5.31	194
Amygdala L (extending into putamen, pallidum, hippocampus, olfactory, and caudate)	-16	-2	-14	.004	5.23	109
Amygdala R	16	2	-14	.007	5.09	43
Basal ganglia						
Thalamus L	-2	-10	10	.001	5.52	74
Putamen R	22	12	0	.029	4.76	9
Parietal lobe						
Precuneus R	10	-38	6	.012	4.98	32
Precuneus L	-12	-72	54	.033	4.73	6
Supramarginal	-62	-38	26	.039	4.69	10
Cerebellum						
R	26	-62	-28	<.001	5.97	432
	16	-60	-46	.001	5.45	81
	12	-70	-22	.017	4.90	42
	6	-84	-22	.018	4.88	24
L	-38	-56	-30	.004	5.21	115
NA	20	-16	-12	.014	4.93	12

Note. L = left hemisphere; R = right hemisphere. NA = region not defined in the Automated Anatomical Labeling brain atlas.

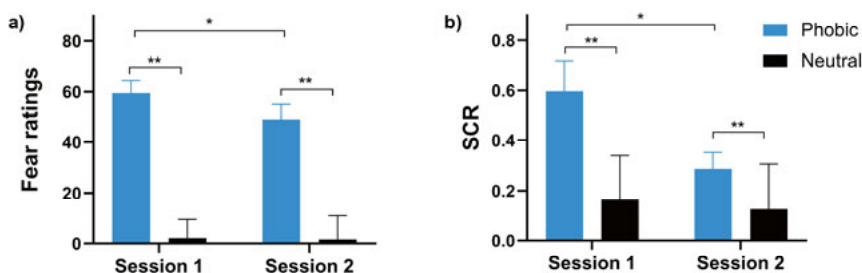


Figure 5. Subjective fear and skin conductance responses (SCRs) to mental imagery of phobic and neutral stimuli. Bar charts depict a) subjective fear ratings (N=29) and b) mean SCRs (N=27) to mental imagery of phobic stimuli (spiders) and neutral stimuli (gloves) during the experimental procedure in sessions 1 and 2. Error bars represent standard errors of mean. * $p < .05$, ** $p < .001$.

Discussion

This was the first experimental study that systematically investigated neural processing during imaginal exposure to phobic stimuli. Results showed that generating mental imagery of a phobic stimulus recruited brain areas associated with fear processing. Notably, even though no direct comparisons were conducted, imaginal exposure to phobic stimuli (i.e., mental imagery of spiders) activated similar brain areas as those previously found to be activated by *in vivo* exposure (i.e., the direct perception of spiders). The results highlight the power that mental imagery has on emotion. That is, mental imagery of a fear-provoking stimulus alone, i.e., in the absence of the actual stimulus, can elicit fear responses that can be observed at the subjective, physiological and neural level. Relevant for treatment development, the results revealed that a single 10-minute session of brief exposures to mental imagery of phobic stimuli could promote reductions in subjective and physiological fear responses. The results of the study were in line with emerging evidence suggesting that the duration of imaginal exposure could potentially be reduced without compromising treatment effects (Nacasch et al., 2015).

Study II

Imaginal extinction and the vividness of mental imagery: Exploring the reduction of fear within the mind's eye

Background and aim

Vivid mental imagery during imaginal exposure is generally assumed to promote fear reduction. However, vividness can increase distress, which is not well-tolerated by some patients. Importantly, it is currently not clear to what extent fear reduction depends on the vividness of mental imagery produced by the patient during imaginal exposure. Study II examined the link between the vividness of mental imagery and the reduction of fear using an experimental analogue of imaginal exposure: imaginal extinction (Agren et al., 2017). In imaginal extinction, conditioned fear, as measured with skin conductance, is reduced through exposure to mental imagery of the conditioned stimulus. Study II investigated if the reduction of conditioned fear (i.e., SCRs) was moderated by how vividly participants could imagine the conditioned stimulus during imaginal extinction (i.e., task-specific vividness). The impact of vividness during imaginal extinction on remaining fear responses 24 h later was also explored. The study also examined if the impact of imagery vividness on fear response depended on the perceptual complexity of the conditioned stimulus. For this purpose and to defend against a possible ceiling and floor effect on vividness (i.e., stimuli being too easy/hard to visualise vividly), participants were allocated to undergo the experimental procedure with either complex or simple stimuli.

Methods

Forty-eight participants (age: $M = 25.3$ years; $SD = 5.8$ years; 22 women and 26 men) were included in analyses (Figure 6). The study took place during three consecutive days, with fear conditioning to visual stimuli on day 1, imaginal extinction on day 2, and a reinstatement procedure, again to visual stimuli, on day 3 (Figure 7b). Skin conductance response was used to measure fear response (on days 1, 2 and 3; primary outcome measure). Fear responses within the extinction session were compared between participants reporting higher versus lower task-specific vividness, defined by a median split. Participants were allocated to undergo all experimental phases (days 1–3) with either complex or simple stimuli (Figure 7a). The study was registered at ClinicalTrials.gov (id: NCT03989518).

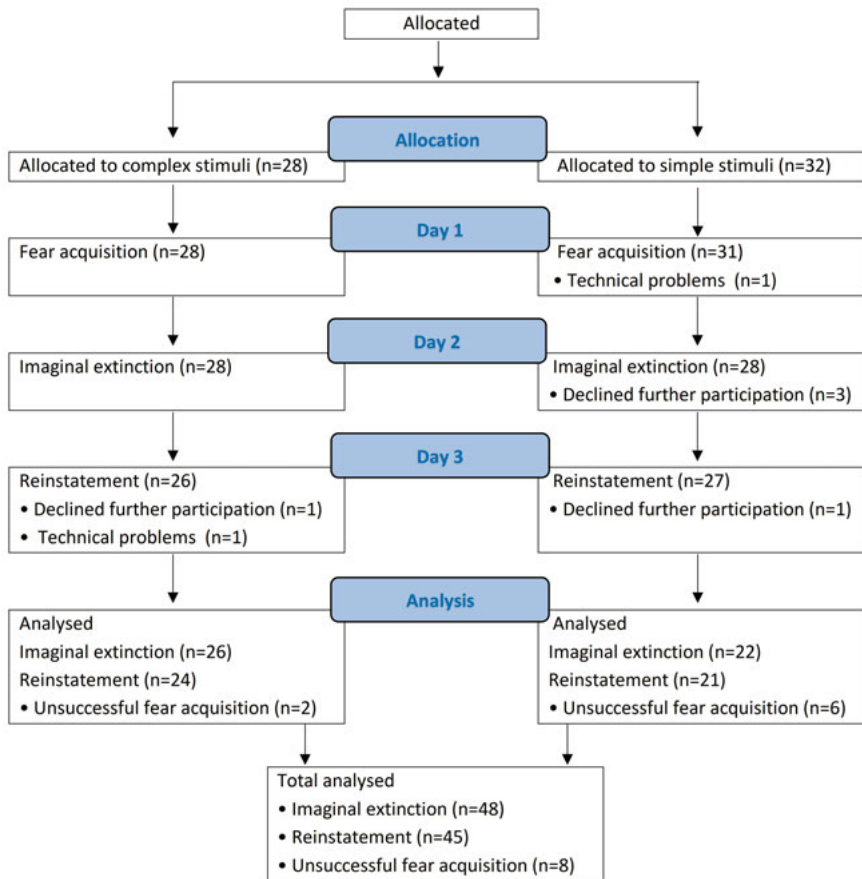


Figure 6. Participant flowchart for the study. The flowchart shows an overview of the allocation to experimental conditions (complex vs. simple stimuli) and attrition across experimental phases (acquisition, imaginal extinction and reinstatement). For inclusion in analyses, participants had to meet the criteria for successful fear acquisition (mean SCR CS+ > mean SCR CS-) and undergo the imaginal extinction procedure.

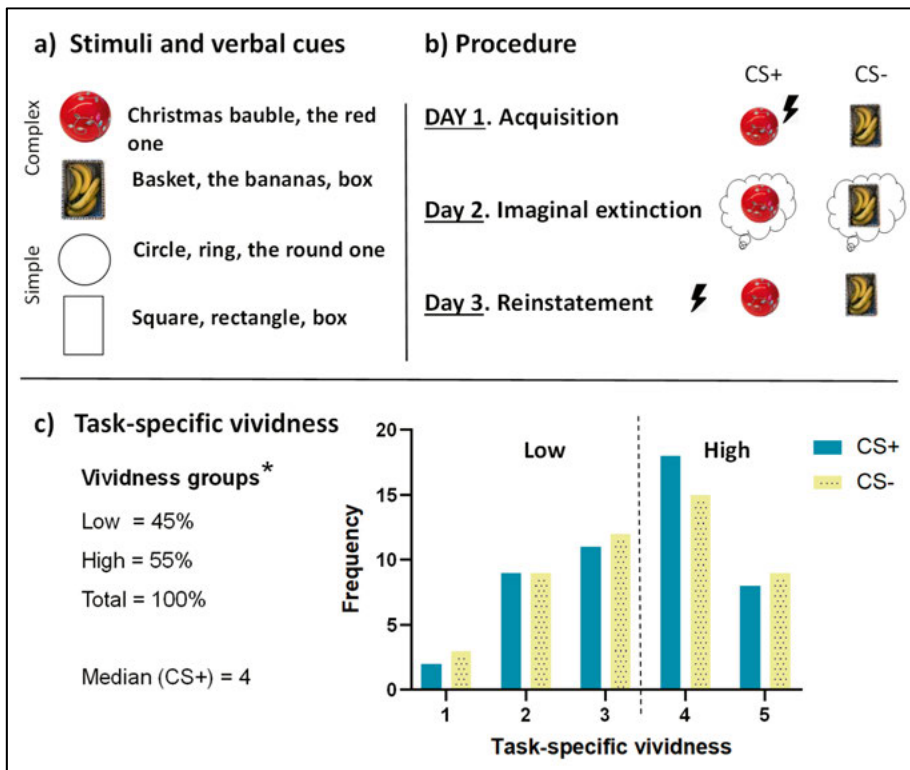


Figure 7. Overview of stimuli, experimental design and task-specific vividness. The Top-left panel depicts (a): Experimental stimuli and corresponding verbal cues used to prompt imagery of each stimulus. The experimental stimuli representing the conditioned stimulus (CS+) and the control stimulus (CS-) within each condition (simple vs. complex) were counterbalanced across participants. Top-right panel (b): Overview of the experimental procedure, using complex stimuli as an example. Each experimental phase was performed 24 h after the previous phase. Bottom panel (c): Distribution of ratings of task-specific vividness during imaginal extinction across all participants. *Participants were divided into two groups (high vs. low), based on a median split of task-specific vividness ratings of the CS+.

Results

Analysis of variance showed successful fear extinction across the entire sample (Figure 8a). Follow-up analyses of imaginal extinction revealed that only the high vividness group showed successful fear extinction, as reflected by a significant stimulus \times trial interaction (Figure 8b). When fear responses were measured 24 hours later, no significant differences in remaining fear responses were observed between high and low vividness groups, as measured with the reinstatement procedure (Figure 8c). Stimulus complexity did not moderate the effect of vividness on conditioned responses during either imaginal extinction or reinstatement.

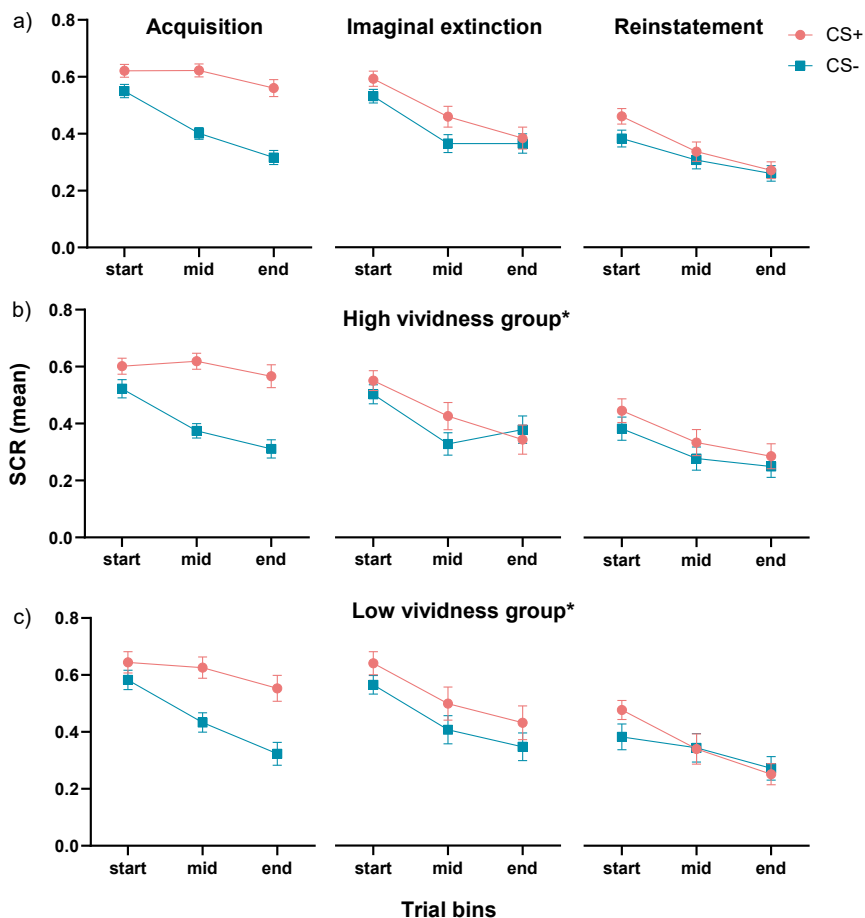


Figure 8. Skin conductance responses (SCRs) across experimental phases for all participants and within each vividness group. Mean SCRs to experimental stimuli (CS+ and CS-) within all experimental phases (start, mid, end). SCRs are displayed for (a) all participants and for (b) high and (c) low vividness groups. *Vividness groups are based on task-specific vividness ratings of the CS+. SCRs are root-transformed and range-corrected. Error bars represent standard errors.

Discussion

Study II examined the link between the vividness of mental imagery and the reduction of fear (SCR) in imaginal extinction. Findings suggested that high vividness may be advantageous for within-session fear reduction, but may not influence fear responses in the longer term. Considering that long-term effects are the ultimate goal of imaginal exposure, the findings suggested that high imagery vividness may not be vital for overall treatment outcome. The results

showed a pattern not dissimilar to previous findings in clinical studies on PE, which found consistent evidence of an association between vividness and reduction in subjective distress within the imaginal exposure session, but not between in session vividness and overall treatment outcome (Mota et al., 2015; Rauch et al., 2004). The results of Study II are preliminary. In order to inform treatment development, the results need to be extended from conditioned fear to naturally occurring fear, and from physiological fear responses (SCR) to subjective fear. Extending the findings is essential, as clarifying the link between vividness and long-term fear reduction could improve the tolerability of imaginal exposure. If similar long-term fear reduction can be obtained with less vivid imaginal exposure, using strategies to reduce vividness during exposure (e.g., concurrent visuospatial tasks) could lower distress and make treatment tolerable for more patients.

Study III

Hotspots in the immediate aftermath of trauma – Mental Imagery of Worst Moments Highlighting Time, Space and Motion.

Background and aim

Intrusive memories are a hallmark symptom of PTSD (American Psychiatric Association, 2013) that often take the form of vivid mental imagery of the traumatic event (Iyadurai et al., 2019). In most cases, it is the ‘hotspots’, e.g., the worst moments, in the trauma memory that become intrusive (Grey & Holmes, 2008; Holmes, Grey, et al., 2005). Besides being closely linked to intrusive memories, hotspots are important treatment targets in PE (Foa et al., 2019) and other evidence-based trauma-focused therapies for PTSD (Ehlers & Clark, 2000; Shapiro, 1996). However, knowledge about the characteristics of hotspots is limited, and no study has examined hotspots reported in the immediate aftermath of trauma, i.e., close to the time of their encoding. Increasing our knowledge of newly formed hotspots could give us clues to how intrusive memories are formed and how to prevent them. The aim of Study III was to investigate hotspots collected within the first hours posttrauma, in terms of their frequency (i.e., number of hotspots within a single event trauma memory), sensory-perceptual features and other contents, and emotional and cognitive themes.

Methods

Hotspot data ($n=21$) were collected in the context of an RCT conducted on trauma-exposed individuals in an ED (Kanstrup et al., 2021).

Hotspot data were recorded within 72 h after the traumatic event ($M = 11.1$, $Mdn = 3.0$, $SD = 14.2$, range = 0.75–56.7 h). Hotspots were collected using a brief interview technique (Iyadurai et al., 2018) conducted at the baseline of the RCT. The characteristics and contents of hotspots were analysed using linguistic analysis and qualitative content coding.

Diary examples (i.e., examples of intrusive memories and hotspots) provided by a larger number of participants in the overarching RCT were analysed as a convergent measure (i.e., for comparisons with results from the primary hotspot data). Thus, corresponding analyses conducted in hotspot data were carried out on diary examples ($n=39$). Notably, 20 of the 39 participants also provided hotspot data.

Linguistic analysis was conducted with the Linguistic inquiry and word count software (LIWC 2015; Tausczik & Pennebaker, 2010). LIWC was used

to calculate percentages of words associated with predefined word categories of interest (e.g., affective processes, cognitive processes, perceptual features). The mean number of words per hotspot was also extracted (Table 4).

Qualitative coding was used to explore hotspots in terms of their sensory-perceptual features and other contents. To this end, a novel coding frame was developed for this study (Hotspots coding frame is presented in the Supplementary materials of Paper III). Emotional and cognitive themes were examined using an existing coding frame described in Holmes et al. (2005).

Training procedures. To promote protocol fidelity, research assistants delivering the intervention/control condition in the pilot RCT (which included collecting hotspots) received formalised training and continuous feedback and monitoring (Kanstrup et al., 2021).

Results

Participants reported a mean of 3.0 ($Mdn = 3.0$, $SD = 1.1$, range 1–5) hotspots, each described using a mean of 7.8 words. Results from linguistic analysis and qualitative coding are presented in Table 4 and Table 5, respectively. Linguistic analysis revealed that hotspots primarily contained words related to time, space, motion and perceptual processes. Approximately 97% of hotspots contained sensory features and 59% contained motion features. Adopting the coding frame of Holmes et al. (2005), two hotspots were found to include cognitive themes. No direct references to emotions were identified in any hotspots.

Discussion

This study was the first to systematically investigate hotspots reported within the first hours after a traumatic event. Although preliminary, the findings indicated that newly formed hotspots were motion-rich sensory-perceptual experiences (mental imagery), with little detail on emotion/cognition. These findings were consistent with the notion that traumatic events are predominantly encoded as sensory-perceptual memories (Brewin, 2014), which are later re-experienced as mental imagery rather than verbal thought (Ehlers et al., 2004). The key finding that hotspots were to a large degree dynamic and visuospatially situated raised questions about the functional aspects of hotspots and associated intrusions (e.g., preparedness for action), as well as the mechanisms via which visuospatial interference tasks (e.g., Tetris gameplay using mental rotation) may reduce the number of subsequent intrusive memories.

Table 4. Percentages of words within hotspots/diary examples matching LIWC word categories of interest in linguistic analysis

<i>LIWC¹ word category</i>	<i>Hotspots</i>	<i>Diary examples</i>	<i>Example words</i>
	%	%	
Relativity	28.9	30.8	
Motion	8.9	9.4	falling
Space	18.1	20.0	front
Time	3.2	2.8	fast
Perception	10.6	8.7	
See	6.3	5.7	look
Hear	1.5	0.4	hear
Feel	2.7	2.5	press
Biological processes	5.3	6.2	
Body	4.4	5.1	finger
Health	0.2	0.0	bleeding
Sexual	0.0	0.0	-
Ingestion	0.8	1.1	water
Time orientation			
Time – Past	1.1	0.6	thrown
Time – Present	7.8	4.1	runs
Time – Future	2.1	1.3	then
Affective processes	1.7	3.2	
Positive emotion	0.2	0.2	lucky
Negative emotion	1.5	3.0	wrong
Anxiety	0.0	0.0	-
Anger	0.4	1.1	-
Sadness	0.2	0.4	-
Cognitive processes	4.0	1.5	
Insight	0.2	0.0	thinking
Causation	0.8	0.4	because
Discrepancy	0.2	0.2	-
Tentative	1.5	0.2	anything
Certainty	0.4	0.4	correct
Differentiation	0.8	0.4	isn't

Note. Analysis of hotspot data included 63 discrete hotspots recorded from 21 participants. Analysis of diary examples included 91 discrete examples of intrusive Analyses were conducted using the ¹ Linguistic inquiry and word count (LIWC 2015) software. % = percentage of words within hotspots/diary examples matching LIWC word categories and subcategories. The last column provides examples of words within 'hotspots'/diary example categorised into the respective LIWC word categories.

Table 5. Frequency of sensory-perceptual features, content and emotional themes in hotspots and diary examples

	<i>Hotspots</i>		<i>Diary examples</i>	
	n	%	n	%
Sensory features	61	96.8	91	100
Visual	48	76.2	66	72.5
Proprioceptive – kinaesthetic	14	22.2	24	26.4
Auditory	6	9.5	3	3.3
Tactile	5	7.9	14	15.4
No sensory features	2	3.2	0	0.0
Content features				
Narrative cohesiveness	17	81.0	22	56.4
Content conveys threat	45	71.4	59	64.8
Any motion features	37	58.7	46	50.5
- Motion – flying and falling	7	11.1	11	12.1
- Motion – other	30	47.6	35	38.5
First sign of threat	15	23.8	27	29.7
Attempted action	9	14.3	11	12.1
Outside body perspective	1	1.6	2	2.2
Body/biology	16	25.4	21	23.1
Emotional and cognitive themes				
Cognition	2	3.2	0	0.00
Emotion	0	0.0	0	0.00

Note. Analysis of hotspot data included 63 discrete hotspots recorded from 21 participants. Analysis of diary examples included 91 discrete examples of intrusive memories and hotspots from 39 participants, of whom 20 also provided hotspot data.

n = number of hotspots/diary examples with feature or theme.

% = percentage of the total number of hotspots/diary examples with feature or theme.

General discussion

Main findings

Study I demonstrated that imaginal exposure to phobic stimuli robustly activated emotion-processing brain areas, including the MCC, insula and amygdala. Study I also revealed that a single 10-minute session of imaginal exposure to flashpoint mental imagery was associated with a reduction in fear responses at both the subjective and the physiological level when assessed approximately one week later ($M = 9.5$ days, $SD = 1.8$). In **Study II**, no evidence was found that vividness of mental imagery during imaginal extinction moderated the reduction of conditioned fear (SCR) in the long term (24 h), i.e., higher vividness was not associated with greater long-term fear reduction. **Study III** revealed that the vast majority of trauma memory hotspots, reported within the first hours after trauma, were described as motion-rich sensory-perceptual experiences (mental imagery) with little detail on emotion/cognition.

Discussion

I opened the introduction by highlighting what I believe are among the most important challenges of psychological research. These were to develop more effective, accessible, and tolerable psychological treatments and to find ways to prevent psychopathological conditions. A crucial step towards these goals is to unravel the underlying mechanisms of our core psychological treatment techniques and increase our understanding of psychological processes involved in the aetiology and maintenance of psychopathology (Holmes et al., 2018). Accordingly, this thesis sought to increase our knowledge of the underlying mechanisms of imaginal exposure and improve our understanding of emotional mental imagery, including the building blocks of intrusive memories (i.e., hotspots). Specifically, **Study I** explored the neural basis of imaginal exposure and investigated if a 10-minute imaginal exposure session affected fear responses. **Study II** investigated the link between the vividness of mental imagery and the reduction of fear within the mind's eye, using an experimental analogue of imaginal exposure (i.e., imaginal extinction). Lastly, **Study III** explored the characteristics of hotspots in trauma memories collected within the first hours posttrauma.

The neural basis of imaginal exposure

Study I advanced our knowledge of the neural underpinnings of imaginal exposure by providing first evidence that exposure to mental imagery of phobic stimuli robustly activates fear-processing brain areas. Notably, the brain areas activated during *imaginal* exposure (e.g., MCC, insula and amygdala) were surprisingly similar to the areas previously shown to be most consistently associated with *in vivo* exposure to phobic stimuli (e.g., pictures of spiders; Ipser et al., 2013). The findings thus added to emerging evidence showing substantial overlap in the neurocircuitry employed by mental imagery and actual perception (Pearson, 2019), and extended these findings by showing that the overlap also applies to neural processing of phobic fear. Further highlighting the power that mental imagery has on emotion, mental imagery of phobic stimuli (spiders) elicited both intense subjective fear and heightened physiological responses.

Opportunities for psychological treatment development

Study I explored if a short (10-minute) session of imaginal exposure had an effect on fear responses. This was motivated by the potential benefits of reducing the duration of exposure sessions, to increase the accessibility of imaginal exposure-based treatments (e.g., PE). The results showed that the short session was indeed followed by a reduction in both subjective and physiological fear responses, as measured approximately one week later. This finding holds promise for the development of shorter and more accessible treatments. The results also add to other preliminary findings suggesting that shorter imaginal exposure sessions, compared with the 40-minute sessions included in PE, can be effective without compromising treatment effects (Nacasch et al., 2015; Van Minnen & Foa, 2006). In addition to the potential usefulness of short imaginal exposure sessions to reduce fear, the fact that fear was reduced despite using flashpoint imagery (brief mental imagery with hotspot-like emotion-provoking content) rather than imaginal exposure of prolonged duration also opens new avenues for treatment development, which are discussed under clinical implications, below.

Study II investigated another aspect of imaginal exposure that has the potential to improve imaginal exposure, namely the vividness of mental imagery. What made vividness an interesting research target was that it can be manipulated (increased or decreased), which could be harnessed in therapy to improve its effectivity or tolerability. The ultimate goal of psychological treatment is to achieve effects that last in the long term (rather than reducing fear within the exposure session). Thus, the most important finding related to treatment development in Study II was that no evidence was found that higher levels of vividness produced better long-term fear reduction. This finding is in good agreement with results from clinical treatment studies on PE, which

also failed to find robust evidence of a link between vividness and overall treatment outcome (cf. long-term fear reduction; Mota et al., 2015; Rauch et al., 2004). Findings are preliminary and need to be further investigated in naturally occurring fear. Still, these results pave the way for future studies to explore if less vivid imaginal exposure could lower distress and thereby make imaginal exposure tolerable for more patients. Interestingly, in line with the results from Study II, post-hoc analyses of the association between vividness and long-term fear reduction in Study I showed no relationship between vividness during the exposure session and the reduction of fear one week later.

Notably, in Study I, a within-session reduction of fear was not observed, most likely because the procedure used exposure to progressively more fear-provoking stimuli, boosting fear responses throughout the exposure session (e.g., the final exposure trial was ‘a spider crawls into your ear’). Despite the absence of within-session fear reduction, both subjective and physiological fear responses were significantly attenuated when measured approximately one week later. These results are in accord with the assumption of the inhibitory learning approach that within-session fear reduction may not be necessary to attain beneficial treatment effects in the long term (e.g., Craske et al., 2008) and that this applies not only to *in vivo*, but also imaginal exposure. I think we should consider these findings good news, as improving the accessibility of treatments is facilitated by allowing shorter imaginal exposure sessions without compromising treatment effects.

Characterising the building blocks of intrusive mental imagery after trauma

Given the link between hotspots and intrusive memories (Grey & Holmes, 2008; Holmes et al., 2005) and the central role of hotspots in evidence-based PTSD treatments (Ehlers & Clark, 2000; Foa et al., 2019), a deeper understanding of hotspots is vital both for treatment development and to increase our understanding of the aetiology of PTSD. Study III showed that recently encoded trauma memory hotspots were expressed as sensory-based experiences, dominated by visuospatial features. These findings are consistent with the notion that moments in traumatic events are predominantly encoded as sensory-perceptual memories (Brewin, 2014), which are later re-experienced in the form of mental imagery rather than as verbal thoughts (Iyadurai et al., 2019). The findings in Study III are also consistent with theoretical assumptions on the potential mechanisms through which a novel intervention may prevent intrusive memories (Horsch et al., 2017; Iyadurai et al., 2018; Kanstrup et al., 2021; Singh et al., 2020). In this intervention, it is hypothesised that playing the visuospatially demanding game Tetris (using mental rotation), shortly after trauma, while concurrently holding mental imagery of the hotspot in mind, interferes with the consolidation of the visuospatial

components (i.e., mental imagery) of the trauma memory (Singh et al., 2020). However, the prevalence of visuospatial components (i.e., mental imagery features) in newly formed hotspots had never been investigated. Study III is the first study to show that the hotspots reported in the immediate aftermath of trauma are indeed rich in visuospatial features. Results also revealed that hotspots could be described in just a few words ($M = 7.8$ words). The brevity is in line with the idea that hotspots are short memories of specific moments of a trauma (Iyadurai et al., 2019).

Another important finding of Study III was that motion/motor imagery was one of the most prominent features in hotspots. That is, many of the hotspots described by trauma-exposed individuals contained mental imagery of motion present during the traumatic event ('Seeing a lorry come towards me', 'Seeing the dog chew on the hand') or sensations of movement of their own bodies ('I'm flying through the air toward the tree', 'Trying to pull the hand away, dog lunges'). The high prevalence of motion also raises questions about the mechanisms underlying the novel intervention targeting intrusive memories. Tetris is not only a visuospatial task. It is also a dynamic, visuomotor task (i.e., includes motion and requires motor activity; Agren, Hoppe, et al., under review). Just as producing mental imagery of an object and seeing the same object (e.g., a spider) recruit similar neurocircuitry (Pearson, 2019), so does imagining action and executing action (e.g., running; Munzert et al., 2009). As a concurrent task's interference with mental imagery is modality-specific, it suggests that the motion/motor aspects of Tetris may contribute to the effects of the novel intervention.

The dynamic and visuospatial characteristics of hotspots also raise questions about the potential function of hotspots and intrusions. It could be hypothesised that the motion-related sensory information in hotspots could aid the individual to better avoid future threats by facilitating better *action preparation*. Preparedness for action has indeed been suggested to be the primary function of mental imagery (Lang, 1979). For instance, mental imagery facilitates predictions and actions by letting us simulate (voluntarily or involuntarily) future scenarios (based on past experiences) using the same processes for simulation (e.g., sensory-perceptual, motor systems) that would be engaged in the actual scenario (Moulton & Kosslyn, 2009). As stated by Peter Lang (1979) about mental imagery: '...the image is not a stimulus in the head to which we respond; it is itself an active response process.' Thus, the dynamic and visuospatial nature of a hotspot may in some way both yield rich information about a threat and help prepare for action to avoid the threat.

Clinical implications

The most prominent clinical implications of the studies included in this thesis are associated with the duration of the imaginal exposure session (Study I) and the vividness of mental imagery (Study II).

Shorter imaginal exposure sessions could help make treatment, such as PE, more efficient and accessible by making it easier to fit into the often tight schedules of both therapists and patients. Moreover, there may be several clinical implications of exposure to flashpoint mental imagery. The term flashpoint mental imagery was coined to describe brief mental imagery with emotion-provoking contents (cf. hotspots). Such flashes of mental imagery hotspots are common across mental disorders (e.g., Hirsch & Holmes, 2007; Pratt et al., 2004; examples could entail an image of being humiliated in SAD or an image of contamination in obsessive-compulsive disorder). Brief sessions of flashpoint mental images have the potential to become a transdiagnostic intervention that may help ameliorate dysfunctional mental imagery. Just as in exposure therapy, the contents of flashpoint mental imagery can be adapted to match the specific contents of a patient's hotspots. Indeed, the contents of flashpoint mental imagery in Study I were inspired by mental imagery hotspots previously reported by individuals fearful of spiders (Pratt et al., 2004). Brief sessions of flashpoint mental imagery could be used as an additional treatment component in exposure therapy, to enhance extinction learning and counteract the return of fear (Craske et al., 2014). As suggested in the inhibitory learning approach, the return of fear can be counteracted by using certain strategies, such as including exposure to multiple feared cues (deepened extinction), occasional exposure to aversive outcomes (occasionally reinforced extinction), and exposure to multiple contexts (Craske et al., 2014). Flashpoint mental imagery could facilitate the use of these strategies, as mental imagery allows (imaginal) exposure to all sorts of fear-provoking stimuli, situations, and contexts, which is often not the case for *in vivo* exposure, for practical reasons. For instance, flashpoint mental imagery allows exposure to *more numerous* (imagined) instances of the feared object/event (e.g., different types of spiders) within the therapy session, as well as contextual modifications (encountering a spider at home/on the lawn/in the shower), which may help counteract the return of fear due to spontaneous recovery, reinstatement or renewal.

Findings from Study II, together with results from previous studies (Mota et al., 2015; Rauch et al., 2004), suggest that reducing vividness could be used to lessen distress during imaginal exposure without compromising the long-term effects of imaginal exposure. If this is the case, using strategies to reduce vividness could improve the tolerability of imaginal exposure. Vividness can be reduced by using a concurrent visuospatial task, as it competes with mental imagery for limited visuospatial working memory resources (Baddeley & Andrade, 2000; Kavanagh et al., 2001; Singh et al., 2020).

A less obvious, yet significant, clinical implication that I would like to suggest concerns communicating knowledge about the nature of emotional mental imagery to patients. Just as patients undergoing CBT are often educated about how their thoughts and behaviours affect their emotions and vice versa, I argue that many patients have a lot to gain by getting more acquainted with

the nature of mental imagery. Indeed, as previously mentioned, mental imagery is even more closely related to emotion than verbal thought (e.g., Holmes & Mathews, 2005) and has a significant influence on how we behave (e.g., Renner et al., 2019). Just as making patients aware that having a negative thought does not always accurately represent reality, psychoeducation about mental imagery and its effects on emotion, arousal, and behaviour may help patients cope with/reappraise mental imagery-based symptoms and take charge over dysfunctional behaviour. I also believe that sharing research results directly with patients can be informative, useful, and empowering. The findings of Study I could be used to illustrate the impact that mental imagery has on emotion to patients experiencing distressing mental imagery. Indeed, sharing information about the characteristics of hotspots that may be present shortly after trauma could help trauma-exposed individuals to recognise and cope with this type of mental imagery in functional ways.

Limitations

There are several limitations that should be taken into consideration when interpreting the findings of this thesis.

I have argued that treatment development would benefit from studying imaginal exposure with experimental models, as they increase experimental control and allow the study of imaginal exposure specifically, independent of other treatment components. However, while experimental models increase internal validity, the external validity of findings may be compromised. For instance, while a 24 h gap between extinction and a test of return of fear is considered as standard procedure to study the long-term effect of extinction learning within the fear conditioning field (Lonsdorf et al., 2017), in a clinical context, months or years are more in line with the timeframes considered to reflect long-term effect. Therefore, it is critical that the findings in this thesis are extended to longer follow-up times. Furthermore, even though using SCR as an index for fear responses is common within the fear conditioning paradigm, physiological responses (e.g., SCR) are not equivalent to subjective fear (Barrett, 2017). Indeed, to highlight the difference between physiological responses and subjective emotions, the term ‘threat’ response is increasingly used when referring to physiological measures (LeDoux, 2014). It is, therefore, key that the findings from Study II are extended from experimental, conditioned fear to actual phobic fear.

Considering the individual studies separately, the following limitations should be noted. Study I did not include an *in vivo* condition, which precluded direct comparisons between neural activity during *in vivo* and imaginal exposure. Study II used relatively simple stimuli compared with the imagery targeted in imaginal exposure in the clinic. The limitations of Study III included that the way in which questions were worded may have influenced findings. Specifically, given that data were collected in the context of a pilot RCT, the

measures and procedures employed to collect information about hotspots were primarily developed to obtain details about imagery. It is possible that this may have shaped participants' responses by emphasising imagery aspects (e.g., sensory impressions), and inadvertently steering them away from describing verbal thoughts or emotions. Also, the study design did not allow comparisons between the characteristics of hotspots relative to non-trauma memories.

Lastly, studying non-observable processes within the mind is challenging, involving reliance on self-reported ratings of imagery vividness. However, accumulating evidence indicates that people generally have good metacognitive knowledge of their imagery ability, as shown by a positive correlation between subjective vividness ratings and non-subjective measures of imagery 'strength' (e.g., priming effect of mental imagery on perception; Pearson et al., 2011; Rademaker & Pearson, 2012).

Future directions

The findings presented in this thesis raise several research questions that could be investigated in future studies.

Study I did not include an *in vivo* condition. An essential next step to unravel the neural underpinnings of imaginal exposure would be to compare neural processing when fear is elicited through mental imagery versus direct perception of the fear-provoking stimulus (i.e., imaginal versus *in vivo* exposure). Doing so would also allow comparisons of the effects of a brief exposure session when using visual phobic stimuli versus when using imagined phobic stimuli. Assessing brain activity at the follow-up session would also provide information about changes in brain activity associated with successful imaginal exposure. Many of the situations used for flashpoint imagery in Study I contain motion (e.g., 'a spider jumps towards you'). Considering the prominent role of motion in Study III, future studies extending these findings should continue to use dynamic (rather than static) mental imagery. For instance, *in vivo* stimuli could consist of visual 'film clips' including situations corresponding to those used for mental imagery.

Another future direction is to explore the clinical viability of flashpoint imagery in imaginal exposure, e.g., dose-response effects, generalisability, tolerability, and long-term effects. Future studies could also explore if flashpoint mental imagery could be used as an additional component in psychological treatments to ameliorate dysfunctional mental imagery, boost treatment effects, or counteract relapse.

The high dropout rates of PE emphasise the need to explore ways to prevent patients from discontinuing treatment prematurely (Eftekhari et al., 2020; Najavits, 2015). The findings from Study II suggest that reducing vividness could potentially help patients who experience imaginal exposure as

overwhelmingly distressful to stay in treatment. Future studies should evaluate the minimal level of vividness necessary for imaginal exposure to be effective. Exploring optimal/minimal vividness levels could be done in experimental studies by manipulating vividness during exposure, e.g., by including concurrent visuospatial tasks (e.g., Lau-Zhu et al., 2017) or by enrolling participants who cannot deliberately evoke mental imagery (i.e., participants with *aphantasia*; Keogh & Pearson, 2018). Future studies should explore the effects of visuospatial tasks during imaginal exposure on distress levels and treatment outcome. Another interesting future direction would be to test the usefulness of reducing the vividness of the trauma memory *before* starting treatment with PE. Emerging evidence indicates that the intervention using a visuospatial task to prevent intrusive memories after trauma can also be used to reduce the frequency, emotional response, and vividness of longstanding intrusive trauma memories (Iyadurai et al., 2020; Kanstrup et al., 2020; Kessler et al., 2018). This intervention is brief and does not require that the patient talks or thinks verbally about the trauma in detail, thus limiting emotional distress. In short, the intervention entails briefly bringing the contents of the intrusive memory into the mind (often the same as the hotspots targeted in imaginal exposure) and subsequently playing Tetris (using mental rotation) for about 20 minutes (Singh et al., 2020). Thus, future studies should explore if undergoing this simple intervention prior to PE could take the edge of the imagery vividness and distress associated with hotspots and thereby help more patients accept treatment and stay in it.

The abundance of motion features in newly formed hotspots observed in Study III prompts future research to explore the functional aspects of hotspots and intrusive memories. Importantly, longitudinal studies are needed to examine the relationship between the characteristics of hotspots and the development of intrusive memories in PTSD.

Concluding remarks

The significance of the studies in this thesis involves both novel insights into the nature of emotional mental imagery, potential ways to improve imaginal exposure, and how mental imagery is manifested in hotspots within the first hours posttrauma. The studies in this thesis used several different methods, ranging from brain imaging to qualitative coding, seeking to understand the complex phenomena that emotional mental imagery constitutes. I believe that mental imagery is one of the most remarkable capacities of the human mind, and that we only just begun to grasp what emotional mental imagery entails and the many ways in which it can be harnessed in psychological treatments.

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