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A potential pathway to the relapse of fear? Conditioned negative stimulus evaluation (but not physiological responding) resists instructed extinction

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ABSTRACT

Relapse of fear after successful intervention is a major problem in clinical practice. However, little is known about how it is mediated. The current study investigated the effects of instructed extinction and removal of the shock electrode on electrodermal responding (Experiment 1), fear potentiated startle (Experiment 2), and a continuous self-report measure of conditional stimulus valence (Experiments 1 and 2) in human differential fear conditioning. Instructed extinction and removal of the shock electrode resulted in the immediate reduction of differential fear potentiated startle and second interval electrodermal responding, but did not affect self-reported conditional stimulus valence. A separate sample of participants (Experiment 3) who were provided with a detailed description of the experimental scenario predicted the inverse outcome, reduced differential stimulus evaluations and continued differential physiological responding, rendering it unlikely that the current results reflect on demand characteristics. These results suggest that the negative valence acquired during fear conditioning is less sensitive to cognitive interventions than are the physiological indices of human fear learning and that valence reduction requires extended exposure training. Persisting negative valence after cognitive intervention may contribute to fear relapse after successful treatment.

Epidemiological data suggests that 25 percent of the population will develop an anxiety disorder at some stage in life (Kessler, Koretz, Merikangas, & Wang, 2004). It is thus reassuring that efficacious treatments are available for these conditions with exposure based and cognitive therapies emerging as the most commonly used interventions in clinical practice (Ougrin, 2011), both receiving consistent empirical support for a number of anxiety disorders (Bisson & Andrew, 2007; Ougrin, 2011; Sánchez-Meca, Rosa-Alcàzar, Marín-Martínez, & Gómez-Conesa, 2010). In spite of this considerable success, approximately one to two thirds of successfully treated patients will relapse within eight years (Craske, 1999). This figure highlights the need for continued research into the mechanisms underlying fear acquisition, reduction, and relapse — an understanding which is essential for the development of treatments with improved long term outcomes.

Fear is a basic emotion characterized by high levels of negative affect (displeasure) and physiological arousal (Lang, 1995). Classical fear conditioning models can provide a conceptual framework to study the development and treatment of human fear (Craske, Hermans, & Vansteenwegen, 2006). In the laboratory setting, a differential fear conditioning paradigm is often used, involving the presentation of two neutral conditional stimuli and an aversive unconditional stimulus (Lipp, 2006). During acquisition training, one conditional stimulus (CS+) is paired with the aversive unconditional stimulus (e.g. electrotactile stimulus), whilst the other is presented alone (CS−; Lipp, 2006). During fear acquisition, the CS+ becomes a valid predictor of the aversive unconditional stimulus, leading to the development of increased physiological responding and decreased valence ratings to the CS+ in comparison with the CS− (De Houwer, Thomas, & Baeyens, 2001; Lipp, 2006). Extinction training involves the repeated presentation of the CS+ without the unconditional stimulus and has been suggested as an experimental analogue to exposure based interventions (Kerkhof et al., 2012).

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Extinction training is very effective in eliminating differential physiological responding between CS+ and CS− and also reduces the negative valence acquired by the CS+, however, there is evidence that negative valence is more resistant to extinction than are the physiological indices of fear learning and thus requires extended extinction training (Lipp, Oughton, & LeLievre, 2003).

A very common finding in human fear learning is that after successful extinction of differential responding, conditioned responding can reoccur in a post-extinction test session, in the absence of any re-training or re-exposure to the feared stimulus (for a review see Vervliet, Craske, & Hermans, 2013). This phenomenon is referred to as the return of fear (Rachman, 1966). To date, three mechanisms mediating the return of fear have been uncovered; spontaneous recovery: the return of fear following the mere passage of time, renewal: the return of fear following a context change, and reinstatement: the return of fear following unpredicted presentations of the unconditional stimulus (Bouton, 2002). It should be noted that as defined above (Lang, 1995), return of fear implies the recurrence of both physiological arousal and negative affect. However, under a less strict definition, the return of negative valence or physiological arousal alone could be interpreted as being a partial return of the fear response – an occurrence which could predispose the individual for full return of fear.

After observing that persisting negative valence towards the feared stimulus was correlated with higher reinstatement rates, Hermans et al. (2005) suggested that lingering negative valence could provide an additional pathway for the return of fear. Noting that negative stimuli preferentially associate with aversive outcomes (Hamm, Vaitl, & Lang, 1989) and that negative valence has been associated with escape and avoidance tendencies (Chen & Bargh, 1999), Kerkhof et al. (2012) developed this theory proposing, based on Lang's (1995) conceptualization of fear as a combination of high arousal and negative valence, that if negative valence persists after extinction, fear could return if the individual is put in a high arousal situation or state.

The human fear conditioning paradigm can also be used to examine the influence of cognition on the extinction of fear learning. Following, Mower's (1938) initial observation that electrodermal responding could be 'switched on and off' with signals informing the participants when an aversive unconditional stimulus was to be expected, researchers have used the instructed extinction paradigm as an experimental analogue for cognitive interventions to reduce fear. Instructed extinction involves informing one group of participants at the end of acquisition training that the aversive unconditional stimulus will no longer be presented, whilst a control group receives the same level of interaction with the experimenter, but is not informed. Frequently, the instruction that no further unconditional stimuli will be presented is accompanied by removal of the unconditional stimulus electrode (Hugdahl, 1978; Hugdahl & Öhman, 1977; see Sevenster, Beckers, & Kindt, 2012, for mere instruction effects). This manipulation has been shown to reduce the differential electrodermal responding acquired during fear conditioning unless the conditional stimuli used are pictures of snakes or spiders as fear conditioned to these stimuli seems to be encapsulated from cognition (for a recent review see Mallan, Lipp, & Cochrane, 2013). However, electrodermal responding is not selectively sensitive to fear learning, showing the same pattern of responding regardless of whether the conditional stimulus is paired with an aversive or a non-aversive unconditional stimulus (Lipp & Vaitl, 1990). Fear potentiated startle is said to be a more selective index of conditioned fear (Hamm & Weike, 2005), but it is currently not clear whether instructed extinction also affects fear learning as indexed by fear potentiated startle, or the negative valence acquired during fear conditioning.

To date, two studies have assessed the effect of instructed extinction on conditioned fear as indexed by fear potentiated startle and have reached different conclusions. Whereas Mallan, Sax, and Lipp (2009) report that, like differential electrodermal responding, instructed extinction abolishes differential fear potentiated startle, Sevenster et al. (2012) report a dissociation between electrodermal responding and fear potentiated startle. In this study, instruction effects on differential electrodermal responses were immediate, i.e., evident on the very first trial of extinction training, whereas differential startle potentiation persisted for the first two trials of extinction. It should be noted, however, that relative to the non-instructed control group, extinction of fear as indexed by fear potentiated startle was accelerated considerably, as differential fear potentiated startle was absent after the first two extinction trials in the instructed group, but persisted across the first ten extinction trials in controls. Based on the latter finding it seems reasonable to conclude that conditioned fear as indexed by both physiological indices is subject to instructed extinction.

Whether instructed extinction affects the negative valence acquired by a CS+ during acquisition training is less clear. Lipp and Edwards (2002) and Rowles, Lipp, and Mallan (2012) included post-extinction assessments of conditional stimulus valence which seemed to be unaffected by instruction. Equivalent differential evaluation of CS+ and CS− was evident in all groups regardless of the nature of the conditional stimuli used or the instructions provided. However, as conditional stimulus valence was assessed after the completion of extinction training, it is not clear whether the differential conditional stimulus evaluations reflect on insensitivity to instruction or the renewal of fear due to a context change (Bouton, 2002; Vansteenwegen, Dirikx, Hermans, Vervliet, & Eelen, 2006). Lipp et al. (2003; Experiment 2) did not find an effect of instructed extinction on conditional stimulus valence using a continuous assessment during extinction training, however, these results need to be considered with care due to fast extinction in the control group and no instruction effect on electrodermal responses.

The effect of instructed extinction on acquired conditional stimulus valence has also been examined in studies of evaluative conditioning which can inform studies of fear learning. In evaluative conditioning, pleasant and unpleasant pictures rather than aversive electrotactile stimuli are used as unconditional stimuli and conditional stimulus valence can be assessed immediately after instruction and during extinction training. Using such a paradigm, Lipp, Mallan, Libera, and Tan (2010) failed to find an effect of instructed extinction on measures of conditional stimulus valence, immediate or delayed, although participants reported reduced expectancy of the unconditional stimuli immediately after instruction. Gast and De Houwer (2013) found valence measures to be sensitive to instructed extinction in their first, but not in their second experiment. However, the instructed extinction effect in Experiment 1 was not significant for participants who could correctly report the stimulus contingencies used during evaluative conditioning training. Taken together, results from evaluative conditioning studies seem to suggest no effect of instructed extinction on conditional stimulus valence, at least in participants who show evidence of learning during the initial training. It is unclear, however, whether these findings would transfer to fear conditioning that is acquired using a biologically significant aversive unconditional stimulus, such as an electrotactile stimulus. Such an unconditional stimulus is likely to convey significantly higher levels of negative valence and emotional arousal than the presentation of an unpleasant picture.

To assess the effects of instructed extinction on electrodermal responses, fear potentiated startle, and conditional stimulus valence, two differential fear conditioning experiments were conducted using neutral faces as conditional stimuli and an aversive