Biological Psychology 125 (2017) 163–172

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

# Sigh rate during emotional transitions: More evidence for a sigh of relief

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ARTICLE INFO

*Article history:*
Received 2 February 2016
Received in revised form 8 March 2017
Accepted 9 March 2017
Available online 14 March 2017

*Keywords:*
Sighing
Relief
Emotional transitions
Salivary cortisol
Anxiety sensitivity

ABSTRACT

Evidence suggests that sighs regulate stress and emotions, e.g. by facilitating relief. This study aimed to investigate sigh rates during relief. In addition, links between sighs, anxiety sensitivity and HPA-axis activity were explored.

Healthy volunteers (N = 29) were presented cues predicting the valence of subsequent stimuli. By sequencing cues that predicted pleasant or unpleasant stimuli with or without certainty, transitions to certain pleasantness (relief) or to certain unpleasantness (control) were created and compared to no transitions. Salivary cortisol, anxiety sensitivity and respiration were measured.

Sigh frequency was significantly higher during relief than during control transitions and no transition states, and higher during control transitions than during no transition states. Sigh frequency increased with steeper cortisol declines for high anxiety sensitive persons.

Results confirm a relationship between sighs and relief. In addition, results suggest that sigh frequency is importantly related to HPA-axis activity, particularly in high anxiety sensitive persons.



## 1. Introduction

Breathing and emotions are tightly intertwined. Emotions importantly influence respiration, and vice versa, respiration affects emotional state. Because of these close interactions and the fact that breathing is under voluntary control, respiration is a popular target for treatments aiming to reduce negative emotional states, and breathing-focused therapies have been applied to various physiological and psychological disorders. Since interventions for anxiety and anxiety disorders often include breathing training (e.g. Garssen, de Ruiter, & van Dyck, 1992; Meuret, Wilhelm, Ritz, & Roth, 2003) and respiratory phenomena have been thought to be central to some anxiety disorders (Klein, 1993; Ley, 1985), research on anxiety and breathing is particularly interesting and important. Multiple reviews report a rather consistent pattern of breathing changes when anxiety or fear are induced (Boiten, Frijda, & Wientjes, 1994; Homma and Masaoka, 2008; Kreibig, 2010). Generally, anxiety elicits rapid shallow breathing; breathing frequency increases due to shorter inspiratory and expiratory time, and breathing volume decreases, resulting in increased minute ventilation and decreases in end-tidal carbon dioxide ($etCO_2$) (Boiten et al., 1994Kreibig, 2010). Fear consistently elicits increased breathing frequency and decreases in $etCO_2$, but both reports of increases and decreases in breathing volume have been reported (Kreibig, 2010).

Given the dynamic nature of the respiratory system, measures of respiratory variability could significantly add to the study of breathing during fear and anxiety. Only a few studies have investigated the effects of fear and anxiety on general measures of respiratory variability, and on sighing specifically. Increased variability in all respiratory parameters (frequency, inspiratory and expiratory time, volume and $etCO_2$) has been found during imagery of fear (Rainville, Bechara, Naqvi, & Damasio, 2006; Van Diest, Thayer, Vandeputte, Van de Woestijne, & Van den Bergh, 2006; Vlemincx, Van Diest, & Van den Bergh, 2015). Sigh frequency, specifically, increases in response to fear and anxiety; sigh frequency is high in anxious rats (Carnevali et al., 2013) and increased sigh frequencies in healthy persons are characteristic of music performance anxiety (Studer et al., 2012; Studer, Danuser, Wild, Hildebrandt, & Gomez, 2014), threat of shock (Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007) and fear imagery (Vlemincx et al., 2015). Additionally, sighing is typical of anxiety disorders, with high levels in

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http://dx.doi.org/10.1016/j.biopsycho.2017.03.005
0301-0511/

patients with chronic anxiety (Tobin et al., 1983), posttraumatic stress disorder (Blechert et al., 2007) and particularly panic disorder (Abelson, Weg, Nesse, & Curtis, 2001; Abelson, Khan, Lyubkin, & Giardino, 2008; Schwartz, Goetz, Klein, Endicott, & Gorman, 1996; Wilhelm, Gerlach, & Roth, 2001; Wilhelm, Trabert, & Roth, 2001a, [Wilhelm et al., 2001b]2001b).

The question arises as to why fear and anxiety evoke sighs and why excessive sigh frequencies are characteristic of anxiety disorders, and panic disorder particularly. Potential neurophysiological mechanisms are suggested by evidence that the generation of sighs in the pre-Bötzinger complex (Lieske, Thoby-Brisson, Telgkamp, & Ramirez, 2000; Ramirez, 2014; Tryba et al., 2008), can be modulated by brain structures involved in emotional processing. Sigh rates are influenced by disinhibition of the dorsal periaqueductal gray, which mediates defensive behavior (Davenport, Shahan, & Zhang, 2004). Sigh frequency is also modulated by limbic structures linked to emotion processing (e.g. the dorsomedial hypothalamus and the dorsal and medial paraventricular hypothalamus) (Evans, 2010; Harper, Poe, Rector, & Kristensen, 1998; Kristensen, Poe, Rector, & Harper, 1997; Reynolds, Vujisic, Davenport, & Hayward, 2008).

Psychophysiologically, sighing has been hypothesized to function as a resetter, restoring a homeostatic balance when this balance has been perturbed (Vlemincx et al., 2013; Vlemincx, Van Diest, Lehrer, Aubert, & Van den Bergh, 2010). One interpretation of the resetting properties of a sigh is that a sigh facilitates behavioral and emotional transitions (Ramirez, 2014; Vlemincx et al., 2013). One example of such an emotional transition that has been linked to sighing is relief. Sighs have been related to relief from various negative emotional states. Increased sigh rates have been found during relaxation following tension (Stevenson and Ripley, 1952), relief from perceived restlessness in case studies (Hirose, 2000), reductions of negative affect in smoking withdrawal studies (McClernon, Westman, & Rose, 2004), interruptions or discontinuations of difficult or attentive tasks (Teigen, 2008; Vlemincx, Taelman, De Peuter, Van Diest, & Van Den Bergh, 2011) and cues signaling safety during stress in both animals (Soltysik and Jelen, 2005) and humans (Vlemincx et al., 2009). These findings suggest that sighing is importantly associated with relief. However, a sigh not only signals relief, but also enhances relief as evidenced by reductions in muscle tension following spontaneous sighs and increased self-reported relief following instructed deep breaths (Vlemincx, Taelman, Van Diest, & Van den Bergh, 2010; Vlemincx, Van Diest, & Van den Bergh, 2016). Given these relieving effects of a sigh, a sigh may be an adaptive regulatory mechanism during sustained stress and emotional states. In line with this, increased sigh rates are not only elicited by anxiety and fear (as mentioned above), but also more generally by both negative emotions and high arousal emotional states (Vlemincx et al., 2015), such as unpleasantness (Finesinger, 1944), desire, depression (Vlemincx et al., 2015), aggression (Carnevali, Nalivaiko, & Sgoifo, 2014) and stressful mental arithmetic (Vlemincx et al., 2011; Vlemincx, Van Diest, & Van den Bergh, 2012).

If a sigh is a regulatory mechanism inducing temporary relief during sustained stress and anxiety, one could predict that sighs are significantly related to a person's regulatory activity to stress and anxiety. Given that particularly high anxiety sensitive persons experience immediate relief following a spontaneous sigh (Vlemincx et al., 2016), the regulatory properties of sighs in stressful contexts may be especially relevant for persons with high anxiety sensitivity. Consistent with this hypothesis, important relations exist between sighing, anxiety and stress responsiveness. Hypothalamic-pituitary-adrenal (HPA) axis activity, a measure of responsiveness to contextual stress (Dickerson and Kemeny, 2004; Miller, Chen, & Zhou, 2007; Nicolson, 2008), among which novelty (Abelson, Khan, Liberzon, & Young, 2007), is a significant predictor of sigh frequency. HPA-axis activity during accommodation prior to a respiratory challenge significantly predicts sigh frequency during that accommodation period in panic disorder patients and sigh frequency in response to the respiratory challenge in both panic disorder patients and healthy controls (Abelson et al., 2008). These results suggest that high anxiety sensitive persons may show higher HPA-axis activity in a novel experimental context, in which they may sigh more frequently in efforts at regulatory coping. Thus, it seems there exist intriguing links between sighing, anxiety and regulatory activity in response to novelty. If sighing serves a regulatory function by enhancing relief when coping with stress or anxiety, it seems plausible that persons who show increased regulatory coping during stress or when exposed to novelty (e.g. those who are more sensitive to stress or anxiety), consciously or unconsciously seek relief more often, and therefore sigh more in novel or stressful contexts.

Given this hypothesis, we are particularly interested in the links between sighs, anxiety, relief and regulation in response to novelty. The primary aim of this study was to test the hypothesis that sighs are specifically linked to relief, rather than serving as general resetters that are triggered by emotional transitions of various types. Prior work has not directly compared sigh rates between different types of emotional transitions. Relief emerges as a positive emotion during a transition that is certain to occur from an aversive state to something less aversive (Deutsch, Smith, Kordts-Freudinger, & Reichardt, 2015; Roseman and Evdokas, 2004; Roseman, 1996). We sought to compare sigh frequency between relief transitions (transitions from uncertainty to the anticipation of certain pleasantness) and control transitions (transitions from uncertainty to the anticipation of certain unpleasantness) and conditions under which no transitions are occurring. We hypothesized that increased sigh rates would be specifically related to relief transitions, as compared to both a no transitions and control transitions. In addition, we explored whether "transitional sighs" in a relief paradigm are linked to HPA-axis activity in healthy persons varying on anxiety sensitivity.

## 2. Method

### 2.1. Participants

Thirty participants completed the experiment. Due to equipment failure during testing, data of one participant were excluded from analysis. Participant characteristics can be found in Table 1. Participants were screened via phone interview to assess eligibility for study participation. All participants were free of medication, major medical illness or physiological disturbances during the past three months, and had no (history of) trauma or psychiatric disease. All participants used no more than low to moderate amounts of alcohol ($\leq$10 cans of beer per week or equivalent) and tobacco (<20 cigarettes per day) and had a Body Mass Index (weight(pounds)/height$^2$(inches)*703) between 18.5 and 30. Female participants were not breast feeding and were premenopausal, had regular menstrual cycles and participated while in the mid-luteal phase. All were drug free on study day as documented by a negative urine drug test and no women were pregnant as indicated by a urine pregnancy test. The study was reviewed and approved by the University of Michigan Institutional Review Board.

### 2.2. Measures

#### 2.2.1. Self-report measures

Anxiety sensitivity was assessed by total score on the Anxiety Sensitivity Index (ASI, Taylor et al., 2007). The ASI is considered a "fear of fear" measure, as it taps fear of anxiety-related sensations, especially bodily sensations. High anxiety sensitivity is linked to