

# Behavioral avoidance and self-reported fainting symptoms in blood/injury fearful individuals: An experimental test of disgust domain specificity

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## Abstract

This study examined the specificity of disgust in predicting avoidance in blood/injury (BI) phobia. Participants high ( $n = 38$ ) and low ( $n = 46$ ) in BI fear completed measures of disgust across multiple domains and severity of BI-related fear. They then completed three randomly presented behavioral avoidance tasks (BATs) that consisted of exposure to a 15" severed deer leg (BI task), a live spider (spider task), and a 'contaminated' cookie (cookie task). Fainting symptoms associated with each BAT were recorded as well. When controlling for gender and BI fear group membership, mutilation disgust contributed unique variance to avoidance on the BI task and animal disgust contributed unique variance to avoidance on the spider task. None of the disgust domains contributed unique variance to avoidance on the cookie task. For the high BI fear group, self-reported fainting symptoms were more pronounced during the BI and spider BAT than during the cookie BAT. Although mutilation disgust was significantly associated with self-reported fainting symptoms on the BI task among the high BI fear group, this relationship became nonsignificant when controlling for BI-related fear severity. Implications of the domain specificity of disgust and its relevance for understanding fainting responses in BI phobia are discussed.

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## 1. Introduction

Blood/injury (BI) phobia is a clinical diagnosis marked by persistent, intense and irrational fear of stimuli and situations involving blood, injuries, and mutilation (Marks, 1988). BI phobia is the second most common specific phobia for which people seek

treatment (Kleinknecht & Thorndike, 1990). However, for some individuals, this phobia may be severe enough to motivate avoidance and delay in seeking necessary medical care which can have serious health consequences (Kleinknecht & Lenz, 1989). In this context BI phobia may be regarded as a potentially life-threatening condition. Epidemiological research indicates that the prevalence of BI phobia ranges from 3.1 to 4.5% (Agras, Sylvester, & Oliveau, 1969; Fredrikson, Annas, Fischer, & Wik, 1996) with an age of onset around 5.5 years (Bienvenu & Eaton, 1998). Like most specific fears, development of BI phobia may occur through multiple pathways including direct conditioning,

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learning through observing others (vicarious learning), or through receiving fear-relevant information (Kleinknecht, 1994).

BI phobia is distinct from other phobias in that it is associated with a fainting response (or vasovagal syncope) observed in 75–80% of its patients (Kleinknecht & Lenz, 1989; Meade, France, & Peterson, 1996; Öst, Sterner, & Lindahl, 1984; Page, 1994) and a component of this fainting response appears to be heritable (Page & Martin, 1998). The complexity and negative impact (e.g., avoidance of blood donation) of the fainting response in BI phobia has sparked interest in the development and evaluation of potentially efficacious treatments (Öst, Lindahl, Sterner, & Jerremalm, 1984). However, empirically informed models that identify factors that contribute to the development and maintenance of the robust fainting response in BI phobia have been far more illusive. Physiologically, fainting in BI phobia is marked by initial rises in heart rate and blood pressure and their succeeding rapid drop, leading to loss of consciousness (Page, 1994). Fainting has been described as a diphasic response implying that two successive responses with opposed directions of activity are involved (Graham, Kabler, & Lunsford, 1961).

The initial phase of the diphasic response of fainting consists of sympathetic nervous system activity which leads to such symptoms as increased heart rate. There is a general consensus that the increase in sympathetic nervous system activity during the first phase is caused by fear (Curtis & Thyer, 1983). It is generally well understood that the actual fainting occurs in the second phase of the diphasic response. This second phase involves the activation of the parasympathetic nervous system, which leads to decreased blood pressure (Page, 2003). However, the mechanisms underlying the shift from sympathetic activity to the latter part of the diphasic response (parasympathetic activity and subsequent fainting) are less clear. To gain some insight into the mechanisms that contribute to fainting in BI phobia, researchers have increasingly appealed to examining the nature and specificity of emotional reactions elicited during and in anticipation of exposure to BI stimuli (de Jong & Peters, 2007; Olatunji, Lohr, Sawchuk, & Westendorf, 2005).

Although exposure to BI stimuli among phobic individuals generally produces a fear response (Kleinknecht, 1987, 1988), it has been observed to trigger disgust reactions as well (Olatunji, Williams, Lohr, & Sawchuk, et al., 2005). Disgust, as a basic emotion,

elicits a reliable physiological response, facial expression, and withdrawal/avoidance pattern (Woody & Teachman, 2000) and many studies have provided supportive evidence for its role in BI phobia. For example, studies have shown BI phobics' respond to pictures of threat-relevant stimuli with fear and disgust, with disgust being the dominant emotional response (Sawchuk, Lohr, Westendorf, Meunier & Tolin, 2002; Tolin, Lohr, Sawchuk, & Lee, 1997). Although it has been argued that disgust may be an amplified component of negative affectivity associated with phobic avoidance, as opposed to operating as a unique and independent correlate (Thorpe & Salkovskis, 1998), the findings that BI stimuli elicit both fear and disgust highlight the possibility that the physiological attributes of both emotions may be recruited in fainting responses observed in BI phobia (Page, 1994). There is some evidence that the experience of disgust involves parasympathetic activation which may facilitate decreases in blood pressure (Levenson, 1992). Consequently, disgust reactions to BI stimuli in conjunction with parasympathetic activation recruited to attenuate the initial fear-mediated sympathetic activation may result in sufficient reductions in blood pressure to initiate the fainting response (Page, 2003).

In addition to responding with disgust during exposure to threat-relevant stimuli, it has been proposed that BI phobics are characterized by heightened 'disgust sensitivity', a personality disposition marked by a general propensity towards experiencing disgust (Olatunji, Arrindell, & Lohr, 2005). In support of this notion, broad measures of disgust sensitivity have been found to be positively correlated with measures of BI phobia (Sawchuk, Lohr, Tolin, Lee, & Kleinknecht, 2000). In an experimental assessment of disgust sensitivity in BI phobia, Sawchuk, Lohr, Lee, and Tolin (1999) exposed BI phobics and nonphobics to a disgust-eliciting video depicting maggots and larvae (stimuli unrelated to their phobic concerns) and found that BI phobics rated the video as significantly more disgusting than did nonphobics. Consequently, heightened generalized disgust sensitivity has been identified as a potential risk factor in the etiology of BI phobia (Tolin, Sawchuk, & Lee, 1999). Furthermore, it has been proposed that individuals sensitive to disgust may exhibit greater fainting responses during exposure to BI stimuli (Page, 1994). In support of this notion, Page (2003) found that the diphasic blood pressure response pattern that is consistent with the fainting response was most evident among analog participants reporting fear of blood and/or injections who were also high in disgust sensitivity.

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