



The role of cognitions, trait anxiety and disgust sensitivity in generating faintness around blood–injury phobic stimuli

Holly A. Exeter-Kent, Andrew C. Page*

School of Psychology, University of Western Australia, 35 Stirling Highway, Crawley WA 6009, Australia

Abstract

The effects on blood–injury fear and fainting of scripts concerning pain, nausea, and anger and individual differences in trait anxiety and disgust sensitivity were investigated. Eighteen participants were high in disgust sensitivity and trait anxiety, 11 were low in disgust sensitivity but high in trait anxiety, 10 were high in disgust sensitivity but low in trait anxiety, and 16 were low in disgust sensitivity and trait anxiety. Participants were exposed to pain, nausea, and anger scripts during presentation of blood–injury slides. The ability of the scripts to increase symptoms of fear and faintness, on a state version of the Blood–Injection Symptom Scale (BISS; Page, A. C., Bennett, K. S., Carter, O., Smith, J., & Woodmore, K. (1997). Blood–Injection Symptom Scale (BISS): Assessing the structure of phobic symptomatology elicited by blood and injections. *Behaviour Research and Therapy*, 35, 457–464) were examined. Analyses indicated that individual differences in trait anxiety and disgust sensitivity interact to generate symptoms of faintness when the pain script was read. That is, disgust sensitive and trait anxious participants reported greater faintness relative to other conditions. The implications for theory and treatment of blood–injury–injection phobia are discussed.

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*Corresponding author. Tel.: +61 9 6488 3577; fax: +61 9 6488 2655.
E-mail address: andrew@psy.uwa.edu.au (A.C. Page).

1. Introduction

Blood–injury phobia is a persistent, intense and irrational fear of situations involving blood, injuries, wounds, and mutilation (Marks, 1988). Blood–injury phobia may lead to avoidance of life-threatening medical procedures (Kleinknecht & Lenz, 1989). Prevalence estimates of blood–injury phobia range between 3.1% and 4.5% (Agras, Sylvester, & Oliveau, 1969; Bienvenu & Eaton, 1998; Costello, 1982; Fredrikson, Annas, Fischer, & Wik, 1996). It is the second most frequent specific phobia for which people seek help (Kleinknecht & Thorndike, 1990). Blood–injury phobia is an atypical phobia as the majority of sufferers faint in phobic situations (Connolly, Hallam, & Marks, 1976; Öst, Sterner, & Lindahl, 1984; Thyer & Curtis, 1985) and a component of this response appears to be heritable (Page & Martin, 1998).

Blood–injury phobia has in common with other specific phobias that fear involves activation of the sympathetic nervous system (SNS). This activation is associated with the fight or flight response (Cannon, 1927), and co-ordinates the body's responses to stresses (Rhoades & Pflanzner, 1992). Unlike other specific phobias, blood–injury phobia involves a second autonomic nervous system response that entails activation of the parasympathetic nervous system (American Psychiatric Association, 1994; Öst, Lindahl, Sterner, & Jerremalm, 1984) that involves an abrupt and severe drop in blood pressure and heart rate, increases in blood glucose, cortisol, and human growth hormone and decreases of noradrenaline (Vingerhoets, 1984). As blood pressure drops, a fainting episode (or vasovagal syncope; Lewis, 1932) may occur. Thus, blood–injury phobia differs from other specific phobias in that it involves a diphasic autonomic nervous system response (Curtis & Thyer, 1983; Engel, 1978; Graham, 1961). Recent empirical support has been found for this diphasic response in blood–injury phobics (Page, 2003).

Although fear and fainting may co-exist within the same individuals (e.g., Öst, Sterner et al., 1984; Page, 1996; Thyer & Curtis, 1985), Kleinknecht (1987) illustrated that these two responses are partially independent. Kleinknecht (1987) found that some participants reported being fearful around blood–injury, but did not report fainting whereas others, who fainted, did not report fear. Interestingly, Kleinknecht and Lenz (1989) found that early episodes of fainting were more predictive of present fear, than early episodes of fear were predictive of present fainting. They suggested that fear might evolve out of several fainting episodes (rather than vice versa). This study suggested that the onset of fainting preceded the onset of fear. It may be postulated that the factors that lead to the development of fainting may interact with those that lead to fear, and in turn begs the question, what caused the initial fainting?

In response to this question, Kleinknecht and Lenz (1989) proposed that blood–injury fainters may be predisposed to react with vagal mediated responses to blood–injury stimuli, independent of fear, which is not seen in normal individuals. Others, such as Davey and colleagues have described a model that introduced disgust, in addition to fear in the presentation of phobias (Davey, 1994; Matchett & Davey, 1991; Webb & Davey, 1992). Matchett and Davey examined the relationships between fear and disgust sensitivity to different categories of animals. Disgust sensitivity measured by the Disgust Sensitivity Scale (DSS; Rozin, Fallon, & Mandell, 1984) was related to fear-evoking animals but not to those that were associated with revulsion (e.g. maggot, snail, and slug). Webb and Davey (1992) extended this research to show that introducing violent video material resulted in increased ratings of fear for animals in a high fear and high predatory category

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