Anxiety sensitivity and its impact on pain experiences and conditions: A state of the art

Sherry H. Stewart and Gordon J. G. Asmundson

1 Dalhousie University, Departments of Psychiatry and Psychology, and 2 University of Regina, Faculty of Kinesiology and Health Studies and Department of Psychology

Abstract. This paper serves as an introduction to the special issue of Cognitive Behaviour Therapy devoted to the topic of anxiety sensitivity (AS) and its impact on pain experiences and conditions. We provide a historical overview of relevant cognitive behavioural models of chronic pain, summarize recent models incorporating the AS construct, and introduce the papers in the special issue. These papers are organized into two sets – basic laboratory-based investigations and relatively more applied studies. We attempt to highlight some of the most important findings from each of these investigations and studies, in turn. Then, we consider several important conclusions derived from the set of special issue papers and the implications of these for the practice of cognitive-behavioural interventions with pain populations. Finally, we make several suggestions for directions for future investigations in this burgeoning area of cognitive behavioural research and practice. Key words: anxiety sensitivity; experimental pain; cold pressor; fear of pain; pain catastrophizing; anticipatory anxiety; fear-avoidance models; persistent headache; chronic pain

Correspondence address: Sherry H. Stewart, Ph.D., Department of Psychology, Dalhousie University, Life Sciences Centre, 1355 Oxford Street, Halifax, Nova Scotia, Canada, B3H 4J1. Tel: (902) 494-3793; Fax: (902)-494-6585. E-mail: sstewart@dal.ca

Anxiety sensitivity (AS) is a cognitive individual difference variable characterized by a fear of anxiety-related symptoms (Reiss, 1991). For example, people with high levels of AS may believe that when they are experiencing difficulty concentrating that this may portend a loss of control, or they may expect that when they experience a racing heart, this could result in a heart attack. Consistent with the predictions of Reiss’ (1991) expectancy model of anxiety, longitudinal research has now established AS as a risk factor for panic attacks and anxiety disorders (e.g., Schmidt, Lerew, & Jackson, 1999). More recently, AS has also been conceptualized as a risk factor for chronic pain (e.g., Asmundson, 1999). Indeed, studies have shown that AS is associated with acute pain experienced in the laboratory and with persistent or chronic pain conditions (e.g., Keogh & Asmundson, 2004).

It is important to place current chronic pain theories that incorporate AS into historical context. Philips (1987) expanded on the foundational work of Fordyce (1976); Lethem, Slade, Troup, and Bentley (1983); and Linton, Melin, and Götestam (1984) in a model of chronic pain that emphasized the roles of avoidance behaviour and cognitive factors in maintaining the pain condition. She noted that chronic pain patients are characterized by extensive avoidance, including avoidance of stimulation, movement, activity, and social/leisure pursuits. Philips explained that this avoidance behaviour is not only ineffective as a longer-term pain management strategy, but it actually may be harmful in terms of maintaining or exacerbating pain. She also recognized the role of a variety of fear-based cognitive factors (e.g., pain expectancies, pain-related self efficacy, memories of past aversive/painful experiences) and, thus, set the stage for later consideration of the role of AS – a cognitive variable.

Like Philips’ (1987) model, the now popular fear-avoidance model (e.g., Vlaeyen & Linton, 2000) also emphasizes the role of avoidance behaviour in maintaining chronic pain. In this...
model, it is the fear of pain (an emotional variable) that is thought to motivate pain-related avoidance and escape behaviour which, in turn, maintains exaggerated perceptions of pain through processes such as deconditioning. Others have since built upon the fear-avoidance model to incorporate the role of AS in explaining pain chronicity. Because AS reflects a general propensity to develop fears (Reiss, 1991), Asmundson, Norton, and Norton (1999) proposed that AS plays a key role in the onset and maintenance of chronic pain by amplifying the tendency to develop fear of pain. The fear of pain then elevates pain-related avoidance, leading to deconditioning and increased pain experiences. These increased pain experiences result in further avoidance and negative expectancies regarding pain, as described in earlier models. Later, as the role of “pain catastrophizing” (another cognitive variable) became increasingly recognized (see Sullivan et al., 2001), the fear-avoidance model was amended. The amended model conceptualizes cognitive variables of appraisal and expectancies of pain as feeding into pain catastrophizing which, in turn, leads to pain-related fear and associated avoidance (Norton & Asmundson, 2004). Asmundson et al. (2000) further suggested that AS (particularly the physical concerns component) promotes catastrophic cognitions regarding pain. Thus, in the amended model, AS was seen to exert its actions on pain via effects on pain catastrophizing. Additional refinements to the fear-avoidance model are outlined by Asmundson, Norton, and Vlayen (2004).

Other theorists have focused on attempting to explain the role of AS in contributing to the severity of acute pain experiences. Schmidt and Cook (1999) suggested that AS should enhance pain intensity by increasing a person’s vulnerability to experiencing anxiety which, in turn, should promote increased pain experiences. In this model, AS is seen as a distal, trait measure, and anxiety as a proximal, state measure. Similarly, Watt and Stewart (2000) suggested that AS may represent a general tendency to perceive any source of arousal as threatening. Thus, AS should amplify the experience of bodily sensations related to a wide range of somatic events, including pain.

The current special issue involves papers representing the state-of-the-art in this area of research. They are divided into two main sets – basic laboratory-based and relatively more applied studies. The first four papers consist of basic, laboratory-based investigations with non-clinical populations where the focus is on understanding the nature of the relation between AS and the pain experience. All of these laboratory-based investigations make use of experimental pain-induction methods and involve consideration of the mechanisms underlying the known association between AS and aspects of acute pain. The first paper, by Uman, Stewart, Watt, and Johnston, links AS to specific aspects of the pain experience (i.e., pain intensity and fear of pain) in a laboratory-based cold pressor investigation with university women. This investigation shows that the relationship of AS to pain intensity is mediated through the association of AS with a fearful response to the pain stimulus. The second paper, by Keogh, Barlow, Mounce, and Bond, also used the cold pressor to confirm that AS is related to experimentally-induced pain. Their findings suggest that there may be gender differences in how AS relates to pain. AS appears related to self-report measures of pain in women and to behavioural measures of pain in men. The third paper, by Tsao, Lu, Kim, and Zeltzer, describes the results of an investigation using the cold pressor and two other types of laboratory pain stimuli (thermal pain and pressure pain) to investigate underlying mechanisms relating AS to pain experience in children. Their work shows that the relation of AS to pain intensity operates indirectly through the association of AS to anticipatory anxiety while children await painful stimuli. Further, their work contributes to a fledgling body of research that extends the relation of AS and pain from adults to children. The last paper in this section, by Conrod, describes an investigation linking AS to increased anticipatory anxiety when awaiting a painful stimulus in the laboratory. Through the inclusion of a social stressor control condition, this investigation also shows that the elevated anticipatory anxiety of high AS participants is not specific to pain situations.

The second set of papers are relatively more applied than the first set in terms of the type of pain investigated (e.g., persistent headache pain), the population investigated (e.g., chronic musculoskeletal pain patients), or the
treatment focus of the investigation (e.g., an intervention study). In the first paper of this second set, Drahovzal, Stewart, and Sullivan describe the results of a survey study with undergraduates that investigated the relations between AS and pain catastrophizing and their relative contributions to various aspects of persistent headache pain. Their results suggest that although AS and pain catastrophizing are highly correlated, they are separable psychologically, and they independently contribute to the prediction of persistent headache pain. The next paper, by Carleton, Asmundson, Collimore, and Ellwanger, examined differences between chronic musculoskeletal pain patients and controls on responses to a startle probe paradigm tapping automatic and strategic attention allocation to several types of pain-schema relevant words. One result from this investigation was that chronic pain patients with high AS appear to have more difficulty disengaging attention from words indicative of physical harm. In the final paper, Watt, Stewart, Lefaivre, and Uman present on the results of a randomized controlled trial. Their findings show that high AS participants randomized to a cognitive behavioural intervention focused on reducing AS levels, displayed concomitant reductions in fear of pain.

There are several common conclusions that emerge from the results of this set of papers. The first involves the intervening or mediating role of more proximal state anxiety-type constructs in explaining the previously-established relationship between the more distal trait variable of AS and the pain experience. The findings of Uman et al., Tsao et al., and Conrod – all consistent with the theoretical predictions of Schmidt and Cook (1999) – suggest that fear of pain and anxiety when anticipating painful stimulation should be targets of intervention in cognitive behavioural interventions for high AS chronic pain patients. This suggestion is consistent with emerging clinical interventions for chronic pain (Asmundson, Vlaeyen, & Crombez, 2004).

A second conclusion is that the indirect relationship of AS to increased pain experience, via increased state anxiety, generalizes across a variety of different pain induction methods. Although most studies in the first section of this special issue made use of the traditional cold pressor task, Tsao et al. extended these findings to thermal and pressure pain and Conrod extended them to pain induced by mild electric shock. We can have more confidence in this set of findings knowing that they generalize across a wide range of types of pain experience.

The findings also converge in suggesting that the role of AS in pain experiences and conditions may be more complex than originally conceptualized. On the one hand, the results of Watt et al. are consistent with the position of Asmundson et al. (1999) that AS drives the fear of pain, because an intervention focused on AS-reduction resulted in concomitant reductions in fear of pain. On the other hand, the findings of Drahovzal et al. are not entirely consistent with predictions of the amended Vlaeyen-Linton fear-avoidance model that AS exerts its effects on pain via its effects on pain catastrophizing. If this were the case, pain catastrophizing would have been the only significant predictor of persistent headache when both AS and pain catastrophizing were simultaneously entered as predictors in the regression. Instead, AS must have some additional influences on persistent pain that are not mediated through effects on pain catastrophizing. These effects might be mediated through other cognitive processes, such as the difficulties disengaging attention from general physical threat cues identified by Carleton et al.

There are several interesting avenues for future research that emanate from the results of the present set of papers. For example, more research is needed on the underlying mechanisms to explain how elevated state anxiety contributes to increased pain in high AS individuals. One possibility is that elevated anticipatory anxiety contributes to increased physiological arousal (e.g., increased muscle tension) which contributes to an increased pain experience. Conrod’s study tested this possibility and did not provide support for an intervening role of increased physiological arousal. Future research might consider the role of cognitive variables, including attention (see Carleton et al.).

The question of whether the relation of AS to pain experiences and conditions varies by gender also deserves further study. One study in the special issue that directly tested for gender effects found important differences in the relation of AS to manifestations of pain in women versus men (i.e., Keogh et al.).
However, two other studies that directly tested for gender effects failed to provide support for any differences in the relation of AS to pain constructs in males versus females (i.e., Conrod; Tsao et al.). Future research should systematically examine the conditions under which gender moderation effects are and are not observed. For example, do such gender differences emerge with development?

Finally, the Watt et al. study suggests novel avenues for intervention research. Watt et al. showed that by intervening at the level of AS via cognitive-behavioural methods, one could also impact fear of pain in a non-clinical sample. Important next steps would be to determine whether these results extend to the treatment of chronic pain patients; apply to a wider range of outcomes (e.g., perceived disability, pain self-efficacy, pain catastrophizing); and persist at longer-term follow-up. Moreover, future investigations should compare Watt et al.’s new ‘AS-reduction approach’ to ‘graded in vivo exposure treatment’ which involves exposure to movements and tasks that chronic pain patients have avoided due to fear of (re)injury (see de Jong et al., 2005). Watt et al.’s recommended focus on AS in treatment fits well with recent findings (Greenberg & Burns, 2003) that pain anxiety is better viewed as a manifestation of AS than as a specific phobia of pain (since the latter position would favour a focus on reducing avoidance behaviour, as in graded in vivo exposure treatment). Only future randomized controlled trials will tell which of these approaches produces the greatest benefit in the cognitive behavioural treatment of chronic pain, or whether these two approaches can be usefully combined.

**References**


