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The behavioral inhibition and activation systems and function in patients with chronic pain

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ABSTRACT

Background: The behavioral inhibition system (BIS) and behavioral activation system (BAS) are two neuropsychological systems hypothesized to underlie response to cues signaling potential reward and punishment, respectively, also in patient responses to chronic pain.
Objectives: The aim of this study was to test these hypotheses by evaluating the relative contributions of BIS and BAS to the prediction of function in sample individuals with chronic musculoskeletal pain.
Methods: 253 participants were administered a battery of questionnaires. Two linear regression analyses were performed to evaluate the contributions of BIS and BAS to the prediction of impairment and psychological function, and to determine if either or both moderated the effects of pain intensity on function.
Results: After controlling for demographic factors, pain diagnosis, and characteristic pain intensity, BIS contributed significantly and independently to the prediction of pain-related physical impairment and psychological function. BAS activity had a significant and direct effect on psychological function only. No moderating effects of BIS or BAS on the association between pain intensity and function were identified.
Discussion: The findings are generally consistent with a BIS-BAS 2-factor model of chronic pain, suggesting BIS and BAS activity as potential targets for chronic pain treatment.

1. Introduction

Chronic pain is a major biopsychosocial problem worldwide. It has a negative impact on people's ability to exercise, engage in valued social and family activities, and maintain an independent lifestyle (Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006). Chronic pain also has a negative impact on psychological function domains, such as depression, anxiety, and perceived stress (Stubbs et al., 2016). However, pain does not have the same impact on everyone. The negative effects of pain are known to be influenced by a number of psychological factors, such as an individual's tendency to catastrophize about their pain (Craner, Sperry, Koball, Morrison, & Gilliam, 2017) and their trait anxiety sensitivity (Esteve, Ramírez-Maestre, & López-Martínez, 2012). Additional factors that have the potential to influence adjustment to chronic pain are the relative activation of two neurophysiological systems that have been hypothesized to facilitate approach and avoidance behaviors: the behavioral inhibition system (BIS) and behavioral activation system (BAS) (Jensen, Ehde, & Day, 2016).

Gray's Reinforcement Sensitivity Theory (Gray, 1987; Gray & McNauhton, 2000) describes the BIS and BAS as neuropsychological systems that are activated in an automatic way in the presence of environmental or internal cues. Specifically this theory hypothesizes that BIS is activated in the presence of cues indicating the potential for punishment (e.g., pain). This system underlies and facilitates avoidance-related behaviors (e.g., withdrawal), emotions (e.g., anxiety), and cognitions (e.g., catastrophizing). On the other hand, BAS is activated in the presence of cues indicating the potential for reinforcement or the disappearance/omission of an expected negative stimulus. BAS activation facilitates approach-related behaviors (e.g., more activity, impulsivity), emotions (e.g., excitement, joy), and cognitions (e.g., self-efficacy; Bjørnebekk, 2007).

Pain is associated with actual or potential tissue damage and its

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protective role often elicits attention and action, which occur by virtue of the withdrawal reflex it activates, the intrinsic unpleasantness of the pain experience, and the emotional anguish it can elicit (Woolf, 2010). A person's trait tendency for BIS or BAS to be activated in response to pain may therefore explain, at least in part, the variability observed in people's adjustment to pain, as reflected by measures of activity and psychological function (Renee & Cano, 2009). The BIS-BAS model of chronic pain (Jensen et al., 2016) proposes that pain is interpreted as an aversive or punishment-related stimulus by most people. This model therefore hypothesizes that more pain intensity would tend to result in activation of the BIS and subsequent negative psychological responses and physical impairment. In addition, and in support of this idea, significant associations between pain intensity and both impairment and distress are often found. For example, Saavedra-Hernánndez et al. (2012) showed that neck pain intensity is significant predictor of disability. Similarly, Moore et al. (2010) found that moderate and substantial pain intensity reduction resulted in improvements in many outcomes (sleep disturbance, depression, anxiety, and quality of life) such that they approached levels found in the normal (i.e., otherwise healthy) population. Thus, more pain intensity is hypothesized to result in (1) more BIS activation (2) less BAS activation behavioral activation and subsequent positive emotions (BAS inhibition).

Moreover, because pain is an aversive or punishment-related stimulus, the association between BIS and BIS-related responses (as sensitivity to punishment system) and pain is hypothesized to be stronger than the associations between BAS and BAS-related responses (as sensitivity to reward system) and pain. In support of this idea, it has been found that cues that signal the occurrence of pain are more likely to increase the focus of attention on that cue, relative to "safety cues," which result in a decreased chance that the person will experience pain (Van Damme et al., 2004) and that pain will interrupt behavior (Eccleston & Crombez, 1999).

With respect to the relationship between BIS and BAS, a "separable subsystems" model (Corr, 2002; Gray & McNauhton, 2000) hypothesizes that the BIS and BAS work mostly independently. That is, individuals with greater BIS activity, compared with those with a less BIS activity, should be most sensitive to signals of punishment, regardless of their level of BAS activation; and individuals with greater BAS activity, relative to a less activity, should be most sensitive to signals of reward, regardless of their level of BIS activation. Thus, pain is thought to be a cue that directly activates the BIS and pain's impact on patient dysfunction (e.g., negative emotions and disability) is hypothesized to be mediated by BIS, at least in part, regardless of the level of BAS activity (Jensen et al., 2016). If pain influences BAS, then any of pain's negative effects on positive function (e.g., positive emotions and life engagement) would be expected to be mediated by BAS activity, separately and distinctly from any effects on BIS.

On the other hand, a more recent "joint subsystems hypothesis" (Corr, 2002) postulates that BIS and BAS have the potential to influence each other's effects on both reward-mediated and punishment-mediated behavior. That is, these systems may work synergistically, such that the impact of one on function is influenced by the relative activation of the other. With this model, dysfunction is hypothesized to be greatest in people with both high BIS activation and lower BAS activation and vice versa (Corr, 2002). In support of this model, Corr (2002) found a significant BIS (Anxiety) x BAS (Impulsivity) interaction in reactions to experimental manipulations of punishment in a sample of volunteers recruited from a university population. However, to our knowledge, the potential moderating effects of BIS and BAS activation on their effects on patient function have not yet been examined in the context of chronic pain.

The BIS-BAS model of chronic pain (Jensen, Ehde, & Day, 2016) hypothesizes that the two systems are distinct but not completely independent; thus, this model would hypothesize that significant BIS X BAS interactions predicting function might be found in some contexts but not others. Even though pain is hypothesized activate primarily BIS, it may also influence BAS to some degree, via two mechanisms. First, because BIS activation is hypothesized to inhibit BAS to some degree (but not completely), and vice versa, an increase in pain would be expected to inhibit BAS indirectly, via its effects on BIS. Second, because in some situations, pain may activate aggressive responses (a BAS "approach" response), an increase in pain has the potential to result in an increase in BAS activity in some settings and with some individuals (i.e., Muris, Meesters, de Kanter, & Timmerman, 2005). The combination of these two contradictory effects may act to result in an overall weaker association between pain and BAS activation. Thus, the BIS-BAS model of pain hypothesizes that experience of pain would result in (1) more behavioral inhibition and subsequent negative psychological function and (2) less behavioral activation and subsequent positive emotions. A greater tendency for engaging in approach behaviors, feeling of excitement and joy, and believing that one is capable of controlling pain is hypothesized to inhibit (although not necessarily completely eliminate) a tendency to avoid activities, experience fear, or have thoughts of helplessness. With respect to a possible BIS X BAS interaction effect, the BIS-BAS model of chronic pain hypothesizes that such interaction is possible in some contexts, but unlikely to emerge across all contexts.

Existing research provides preliminary support for a BIS-BAS model of chronic pain (Jensen et al., 2016). For example, Jensen et al. (2017) found that patients with chronic pain scoring high in a tendency for BIS activation report more depressive symptoms. BIS has also been shown to moderate the associations between pain-related cognitions and psychological function. Specifically, individuals with chronic pain who endorse more BIS responding evidence stronger associations between kinesiophobia and depressive symptoms than those who endorse less BIS responding (Jensen et al., 2017). Moreover, a trait tendency towards BIS activation has been shown to be associated positively with pain catastrophizing (Muris et al., 2007) which is known to be associated with negative affect and disability in individuals with chronic pain (Quartana, Campbell, & Edwards, 2009). Also in support of the BIS-BAS model of chronic pain, Jensen, Tan, Chua, and BSoc (2015) showed that a higher frequency of severe headaches was associated with higher trait BIS and lower trait BAS scale scores in a sample of undergraduate students, with the association between BIS and pain stronger than that between BAS and pain. Consistent with this idea, Becerra-Garcia and Robles (2014) found that BAS was lower in patients with fibromyalgia, relative to a healthy control group. In addition, it has demonstrated that people with chronic pain have a reduced hedonic response to rewards, and this reduction is associated with smaller nucleus accumbens volume that is responsible of reward processing (Elvemo, Landrø, Borchgrevink, & Haberg, 2015).

In part because of the fact that the BIS-BAS model of chronic pain is relatively new, research testing the model to determine its utility remains preliminary; more research is needed to evaluate the explanatory power of the model, and adapt it as needed based on empirical findings. Given these considerations, the aim of current study was to increase our understanding of the role that BIS and BAS responding may play in the physical and psychological function of individuals with chronic musculoskeletal pain. Based on the BIS-BAS model, we hypothesized that BIS activation and BAS activation would make significant and direct contributions to the prediction of physical impairment and psychological function (positive association with BIS and negative association with BAS), when controlling for demographic factors, pain diagnosis, and characteristic pain intensity. In addition, we hypothesized that BIS and BAS would moderate the association between pain intensity and the study criterion variables, such that those with more BIS and less BAS would evidence stronger associations between pain intensity and function. Finally, we examined the possible interaction between BIS and BAS as a predictor of function. A significant interaction would support the joint subsystems model (i.e., greater influence of BIS and BAS on the effects of each on function) with respect to chronic pain. On the other hand, if a significant BIS X BAS interaction did not emerge,