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

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.

Key Words: behavioral activation system, behavioral inhibition system, chronic pain, impairment, psychological function.

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, 2016). 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 (Craber, Sperry, Koball, Morrison, & Gilliam, 2017) and their trait anxiety sensitivity (Esteve, Ramírez-Maestre, & López-Martinez, 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 & McNaughton, 2000) describes the BIS and BAS as neuropsychological systems that are activated in an automatic way in the presence of environmental or internal cues. 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). 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 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 (Rene & Cano, 2009). The BIS-BAS model of chronic pain (Jensen, Ehde, & Day, 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-Hernandez and colleagues (2012) showed that neck pain intensity is significant predictor of disability. Similarly, Moore and colleagues (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 and 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. 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 et al., 2005). The combination of these two contradictory effects may act to result in an overall weaker association between pain and BAS activation. Thus, this model 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). Jensen and colleagues (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 and colleagues (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, this would support the separable subsystems model (i.e., less influence of BIS and BAS on the effects of each on function) in this context. Figure 1 presents a graphic representation of the study hypotheses.

[PLEASE INSERT FIGURE 1 ABOUT HERE]

METHODS

Participants and procedures

The study participants were recruited from two hospital pain units (the Hospital Costa del Sol Pain Unit and the Hospital Virgen de la Victoria Pain Unit, in Spain) and from a fibromyalgia association ("Asociación de Fibromialgia y Síndrome de Fatiga Crónica de Málaga AFIBROMA", Spain). For the participants who were recruited from

the hospital pain units, physicians in the units reviewed the clinical history of each potential participant, and invited them to participate if they met the study inclusion criteria. Interested participants were contacted by telephone to schedule an assessment. To recruit participants from the fibromyalgia associations, we contacted by phone with the chairpersons of associations and described the study to them. The chairperson then informed the organizations' members about the study via email, and interested members were invited to attend a meeting with research staff to hear more about the study. Those who remained interested following the meeting were enrolled in the study and scheduled for an interview for data collection. A total of 169 individuals were recruited from the pain units, and 84 individuals were recruited from the associations.

Study inclusion criteria were: (1) being from 18 to 65 years old, (2) having a musculoskeletal pain problem for at least 3 months, (3) not having any other physical condition or illness in addition to the pain problem, and (4) not having a severe psychiatric disorder that would interfere with participation. After written informed consent was obtained, a psychologist met with the participants to obtain demographic information, pain and pain history information, and to administer the study questionnaires (described in the Measures section). The study procedures complied with the Declaration of Helsinki and received institutional review board approval by the University of Málaga Ethics Committee.

Measures

Demographic variables

Participants provided basic information about their demographics including age, sex, marital status, highest level of education achieved, and employment status.

Characteristic pain intensity

Characteristic pain intensity was assessed by asking participants to rate their current pain and worst, least, and average pain in the past two weeks on 0-10 numerical rating scales, with 0 = "*No pain*" and 10 = "*Worst pain possible.*" These ratings were then averaged into a single score representing characteristic pain intensity (Jensen, Turner, Romano, & Fischer, 1999).

Trait BIS and BAS activity

Trait BIS and BAS activity were assessed using the 20-item Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ-20; Aluja & Blanch, 2011). The SPSRQ-20 measures individual differences in trait tendency for BIS and BAS activation. Item are answered with a dichotomous "Yes" or "No" response, and are then summed to score into BIS and BAS scales (10 items each). A sample BIS item is, "Are you often worried by things that you said or did?" A sample BAS item is, "Do you like being the center of attention at a party or a social meeting?" The BAS and BIS scales demonstrated good (BAS) and excellent (BIS) internal consistency in the current sample (Cronbach´s alphas = 0.81 and 0.91, respectively).

Pain-related impairment

Pain-related impairment was assessed using the 30-item Impairment and Functioning Inventory for Patients with Chronic Pain (IFI-R; Ramírez-Maestre & Esteve, 2015). With the IFI-R, respondents are asked, first, if they performed a number of daily activities (e.g., sweeping the house, dressing by themselves, or visiting friends) in the previous week. For each activity they did not perform, they were asked to indicate, yes or no, if they did not do the activity because of pain. A pain-related impairment score is then computed by summing the activities not engaged in due to

pain; a higher score indicates more pain-related impairment. In this sample, the reliability of the impairment scale was good (Cronbach´s alpha = 0.81).

Psychological function

Psychological function was assessed using the 5-item World Health Organization Well-Being Index (WHO-5; Bech, 1999). With the WHO-5, respondents indicate how they have been feeling over the last two weeks on a 0 ("*At no time*") to 5 ("*All of the time*") scale. Sample items include, "I have felt calm and relaxed" and "I have felt cheerful and in good spirits." The internal consistency of the measure was excellent in the current sample (Cronbach´s alpha = 0.90).

Statistical analyses

We first computed descriptive statistics to describe the sample. We then calculated Pearson correlations coefficients between the study variables to understand their univariate associations. Next, we examined the variables and their distributions for normality, homoscedasticity and multicollinearity to ensure that they met the assumptions for the planned regression analyses study (Tabachnick & Fidell, 2007). Finally, to test the study hypotheses we performed two multiple regression analyses (Cohen, Cohen, West, & Aiken, 2003), one for each criterion variable (i.e., pain-related impairment and psychological function). Given research that has shown that socio-demographic factors and pain diagnosis can influence important pain-related outcomes (e.g., Ando, et al., 2013; Goldenberg, 2009; May, 2008), we planned to control for these factors in the analyses.

In line with it, in each analyses, we first entered demographic (age, sex) and diagnostic group (fibromyalgia, low back pain, and limb [arm, hand, leg, or foot] pain, or other, dummy coded, being "other" the reference category) as control variables. We

then entered characteristic pain intensity in step 2 and the BIS and BAS scale scores in in step 3. Finally, in step 4, we entered the BIS × Pain Intensity, BAS × Pain Intensity, and BIS × BAS interaction terms. The predictor variables (characteristic pain intensity, BIS score, BAS score) were centered prior to entry to avoid the biasing effects associated with multicollinearity that can occur when examining interaction terms. All analyses were conducted using the Statistical Package for Social Sciences (SPSS; Windows version 22.0, SPSS Inc., Chicago, IL).

RESULTS

Sample characteristics

Two hundred and fifty-three individuals participated in the study. They had a mean age of 52.51 years (SD = 9.85), and 206 (81%) were woman. Eighty-four (33%) reported a diagnosis of fibromyalgia, 75 (30%) of low back pain, 67 (26%) limb pain, and 27 (11%) other musculoskeletal pain problem. The mean pain duration was 10.06 years (SD = 12.23). Table 1 shows more details about the participants' characteristics.

[PLEASE INSERT TABLE 1 ABOUT HERE]

Descriptive analyses and correlations between variables

Mean, standard deviations, and correlations among the study variables are presented in Table 2. The sample reported a characteristic pain intensity level that was moderate to severe, with a mean values of 6.32 (SD = 1.35; possible range, 0 – 10). The strength of the zero order associations between the predictor and criterion variables ranged from small (e.g., BAS with impairment, $r = .16$, $p < .01$; BAS with psychological function, $r = .12$, $p < .05$) to strong (e.g., BIS with impairment, $r = .51$, $p < .01$; BIS with psychological function .55, $p < .01$).

With respect to assumptions testing, the skewness (range from -0.07 to 1.26) and kurtosis (range from -0.02 to -0.59) values did not exceed the standard cutoff of 3 (Tabachnick & Fidell, 2007) indicating adequately normal distributions for the study variables for the planned regression analyses. The lack of multicollinearity among the predictor variables was confirmed by variance inflation factors, as their values (range from 1.04 to 2.28 in both regression analyses) were substantially below the standard cutoff of 10 (Hair, Anderson, Tatham, & Black, 1995).

[PLEASE INSERT TABLE 2 ABOUT HERE]

Pain intensity and BIS and BAS activity as predictors of pain-related impairment

Table 3 presents the result of multiple regression analysis predicting pain-related impairment. As can be seen, and after controlling demographic variables (age and sex) and the diagnoses of the participants, we found that pain intensity contributed significantly to the prediction of pain-related impairment (R^2 change = 0.05; $p < .001$). When pain intensity was controlled, BIS activity ($\beta = 0.44$, $p < .001$), but not BAS ($\beta = 0.03$, $p = 0.671$), made an additional significant contribution to the prediction of this criterion variable. However, none of the interactions made a significant contribution to the prediction of the criterion variable.

[PLEASE INSERT TABLE 3 ABOUT HERE]

Pain intensity and BIS and BAS activity as predictors of psychological function

Both BIS activity ($\beta = -0.53$, $p < .001$) and BAS activity ($\beta = 0.17$, $p < .001$) made statistically significant and independent contributions to the prediction of psychological function, once demographic variables, pain diagnosis, and pain intensity were controlled (see Table 4). However, none of the interaction terms contributed significantly to the prediction of psychological function.

[PLEASE INSERT TABLE 4 ABOUT HERE]

DISCUSSION

The primary purpose of this study was to evaluate the role that BIS and BAS may play in the physical and psychological function of individuals with chronic musculoskeletal pain as a test of the BIS-BAS model of chronic pain. The findings showed that, even after controlling for demographic factors, pain diagnosis, and characteristic pain intensity, BIS was independently and significantly associated with both pain-related impairment and psychological function. BAS was significantly and independently associated only with psychological function. Inconsistent with the study hypothesis, neither BIS nor BAS evidenced a moderating effect on the association between pain intensity and the function variables studied. These findings have important implications for understanding the potential role of BIS and BAS in adjustment to chronic pain.

In line with the BIS-BAS model of chronic pain (Jensen et al., 2016), as well as previous research (Jensen et al., 2017; Muris et al., 2007), the results indicate that the BIS has a more predominant role in the prediction of function in individuals with chronic pain than the BAS. This is reflected both by the facts that (1) the BIS scale made significant and independent contributions to the prediction of *both* function criterion variables and (2) the association between BIS and both criterion variables was stronger than between BAS and the criterion variables. Also, Gray's theory (Gray, 1987; Gray & McNauhton, 2000) posits that BIS facilitates avoidance behaviors, and avoidance behaviors have been associated with chronic pain (Crombez, Eccleston, Van Damme, Vlaeyen, & Karoly, 2012). To the extent that future research identifies a causal role for BIS activation as influencing both physical and psychological function, these

findings suggest that BIS activity may be a viable treatment target in chronic pain populations. Treatments which might decrease BIS activation (i.e., reduce avoidance behavior, maladaptive pain-related beliefs, reduce negative affect) may have benefits – at least in terms of individuals function – in people with chronic pain.

As already noted, the study findings indicated that the BAS appears to be less important as a predictor of participants function than BIS, at least with respect to predicting impairment and psychological function. However, BAS did contribute unique variance to the prediction of psychological function in the study sample, over and above that accounted for by BIS. This role for BAS (reduced but still potentially important for some function domains) is consistent with the BIS-BAS model of chronic pain (Jensen et al., 2016) as well as the findings from other research. For example, Elvemo and colleagues (2015) showed that individuals with chronic pain had significantly reduced scores on reward responsiveness, but not reward drive (both as measured by Behavioral Inhibition/Behavioral Activation Scale; Carver and White, 1994), suggesting that having chronic pain may result in a reduction in hedonic responses to rewards. Moreover, research has shown that people with chronic pain have reduced nucleus accumbens volume (Elvemo et al., 2015); this area of the brain is implicated in the processing of reward, pleasure or positive reinforcement (Malenka, Nestler, & Hyman, 2009). If the current findings are replicated, it possible that, in individuals with chronic pain, BAS plays a greater role in emotional function and responding than behavioral responding. Thus, treatments that target BAS activity such as “positive psychology” interventions (Müller et al., 2016) would be expected to impact psychological function more than physical function, and so may be particularly important for individuals who

endorse high levels of psychological dysfunction in response to pain. Research is needed to evaluate this hypothesis.

The results did not support an interaction effect of BIS and BAS as predictors of function in our sample of individuals with chronic musculoskeletal pain. This findings are in line with the "separable subsystems" model (Corr, 2002; Gray & McNauhton, 2000), and inconsistent with previous human experimental research in undergraduate students (Corr, 2002). However, Corr (2002) notes that the "separable subsystems" model may be more appropriate in some contexts than others.. For example, in the presence of strong appetitive/aversive stimuli, or in samples of individuals with "extreme" personality traits. The chronic pain context could potentially influence both of these characteristics. For example, chronic pain – especially when severe – can be viewed as a strong aversive stimuli. In addition, individuals with chronic pain may have "extreme personality" traits as a result of suffering for a long period of time (the mean pain duration of chronic pain in the sample of individuals who participated in this study was 10 years approximately). Thus, it remains possible that BIS X BAS interactions may emerge in samples of individuals with more mild pain, or who have experienced chronic pain for a shorter duration, consistent with the idea that BIS and BAS may work synergistically in some contexts and with some populations, but not others.

Given that both BIS and BAS made significant and independent contributions to the prediction of psychological function, it is possible that overall treatment efficacy – at least on psychological function outcome domains – could be enhanced by targeting *both* an increase in BAS and a reduction in BIS activity as underlying mechanisms (instead of just one or the other). Research to evaluate the relative effects of existing (and new) treatments on each component of BIS and BAS could identify the potential

"best combination" of treatments which maximally influence (reduce) behavioral avoidance, negative/maladaptive pain beliefs, and negative affect, and also influence (increase) approach behaviors, adaptive pain beliefs, and positive affect; such treatment combinations could potentially be more effective than treatments that target only BIS- or BAS-related domains.

We had hypothesized that BIS or BAS levels could potentially moderate the association between pain and the criterion variables studied here. However, this hypothesis was not supported by the findings; BIS and BAS appeared to have direct effects on function that did not vary as a function of pain severity. However, it remains possible that BIS might increase the vulnerability of people to the consequences of pain, and/or BAS might provide individuals with more resources to help them when faced with the challenges associated with pain, even if these effects are similar across all levels of characteristic pain intensity levels. This possibility provides further support for the need to evaluate the potential benefits of treatments which effectively target and reduce BIS activity and increase BAS activity in individuals with chronic pain.

A number of limitations should be considered when interpreting the current findings. First, we only used self-report measures in the study. Thus, it is possible that shared method variance may have influenced the findings, resulting in stronger associations between the predictors and criterion variables than would have occurred had different sources been used as sources for the study variables. Research that examines the associations between self-report measures of BIS and BAS and objective measures of patient function (e.g., actigraph measures of activity) would be useful. A second limitation is that the study design was cross-sectional. As a result, it is not possible to draw causal conclusions from the associations found. Future research is

needed determine the effects of changes in BIS or BAS (e.g., as might occur with treatments that target BIS and BAS activity) and subsequent patient function. Third, the sample included a larger number of women than men. Although the ratio of women is greater than of men in this health services, a sample with more men as well as with other type of chronic pain diagnoses is needed to evaluate the generalizability of the current findings. In addition, the most recent version of Gray´s theory includes a third system – a fight-flight-freeze system (FFFS) – that we did not evaluate here. We had a number of reasons for not including an examination of the FFFS in the current study. First, the goal of the current study was to evaluate the BIS-BAS model of chronic pain (Jensen et al., 2016), which does not take into account the FFFS, because the FFFS system is rarely stimulated in most situations; fight or flight responses do not usually occur on a daily basis. Thus, excluding this system allowed the model to keep more focused on those factors that predict day-to-day responses. In addition, like our 2-factor model (Jensen et al., 2016), none of the many other 2-factor models which incorporate the BIS and BAS or systems very much like them (Elliot, 1997; Gray and McNaughton, 2000; Harmon-jones, 2004; Watson, Wiese, Vaidya & Tellegan, 1999), also do not incorporate the FFFS as a part of their model. Moreover, scientists, including McNaughton and Corr (2008), note that the association between BIS and FFFS is very close. FFFS activation is thought to be preceded by BIS activation and they can therefore be combined into a single “punishment sensitivity” factor of personality (Corr, 2009). Thus, the distinction between the FFFS and BIS is thought be less than that between the BIS and the BAS. Future research is needed to evaluate if, and how, the FFFS and other systems may interact with the BIS and BAS to impact adjustment to chronic pain.

Despite the study's limitations, the findings provide new information regarding the role that BIS and BAS have as predictors of function in in individuals with chronic pain. The results are generally consistent with a model that argues that both BIS and BAS may explain differential responses to pain, and that BIS may play a larger role than BAS (Jensen et al., 2016). The findings also suggest that BAS may be only meaningfully important with respect to psychological function, while BIS may play roles in both impairment and psychological function. Additional research is needed to evaluate the generalizability of these findings, as well as to study the potential causal role that BIS and BAS may play in adjustment to chronic pain. In addition, based on these findings, further research could analyze in detail how, and through what mechanisms, BIS and BAS are related to psychological function and emotional regulation in patients with chronic pain. In the same way, they could evaluate how the systems interact in the activity patterns of this type of patients (excessive avoidance or excessive persistent). Also, we recommend that future researchers incorporate the evaluation of additional subsystems when possible (e.g., as measures of these are developed) for understanding, and treating, chronic pain and its negative impact.

REFERENCES

Aluja, A. & Blanch, A. (2011) Neuropsychological Behavioral Inhibition System (BIS) and Behavioral Approach System (BAS) Assessment: A Shortened Sensitivity to Punishment and Sensitivity to Reward Questionnaire Version (SPSRQ–20). *Journal of Personality Assessment, 93*(6), 628-636.

Ando, S., Yamasaki, S., Shimodera, S., Sasaki, T., Oshima, N., Furukama, T., Asukai, N., Kasai, K., Moni, Y., Inoue, S., Okazaki, Y., & Nishida, A. (2013). A greater number of somatic pain sites is associated with poor mental health in adolescents: a cross-sectional study, *BMC Psychiatry, 13*(30), 1-8.

Becerra-Garcia, J. A., & Robles, M. (2014). Behavioral approach system activity and self-reported somatic symptoms in fibromyalgia: an exploratory study. *International Journal of Rheumatic Diseases, 17*, 89–92.

Bech, P. (1999). Health-related quality of life measurements in the assessment of pain clinic results. *Acta Anaesthesiol Scand, 43:* 893–896.

Bjørnebekk, G. (2007). Reinforcement sensitivity theory and major motivational and self-regulatory processes in children. *Personality and Individual Differences, 43*, 1980-1990.

Breivik, H, Collett, B, Ventafridda, V., Cohen, R., & Gallacher, D. (2006). Survey of chronic pain in Europe: Prevalence, impact on daily life, and treatment. *European Journal of Pain, 10,* 287–333.

Carver, C.S., & White, T.L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology, 67*, 319-333.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences*. United State: Lawrence Erlbaum Associates.

Corr, P. (2002). J. A. Gray's reinforcement sensitivity theory: tests of the joint subsystems hypothesis of anxiety and impulsivity. *Personality and Individual Differences, 33,* 511-532.

Craner, J. R., Sperry, J. A., Koball, A. M., Morrison, E. J. & Gilliam, W. P. (2017) Unique Contributions of Acceptance and Catastrophizing on Chronic Pain Adaptation. *International Society of Behavioral Medicine, 24*(4), 542-551.

Crombez, G., Eccleston, C., Van Damme, S., Vlaeyen, J.W., & Karoly, P. (2012). Fear-avoidance model of chronic pain: the next generation. *Clinical Journal of Pain*, 28, 475-83.

Eccleston, C. & Crombez, G. (1999). Pain Demands Attention: A Cognitive-Affective Model of the Interruptive Function of Pain. *Psychological Bulletin, 125*, 356-366.

Elliot, A.J. (1997). Integrating the ''classic'' and ''contemporary'' approaches to achivement motivation: A hierarchical model of approach and avoidance motivation. In M. Maeher and P. Pintrich, (Eds), *Advances in Motivation and Achievement.* (143-179). Greenwich, CT: JAI Press.

Elvemo, N. A, Landrø, N. I, Borchgrevink, P. C, & Haberg, A. K. (2015). Reward responsiveness in patients with chronic pain. *European Journal of Pain, 19*(10), 1537-43.

Esteve, R., Ramírez-Maestre, C. & López-Martínez, A. E. (2012). Experiential avoidance and anxiety sensitivity as dispositional variables and their relationship to the adjustment to chronic pain. *European Journal of Pain, 16,* 718-726.

Goldenberg, D.L. (2009). Diagnosis and Differential Diagnosis of Fibromyalgia. *The American Journal of Medicine, 122*, S14–S21.

Gray, J. A. (1987). *The psychology of fear and stress*. Londres: Cambridge University Press.

Gray, J. A, & McNauhton (2000). N. *The neuropsychology of anxiety: an enquiry into the functions of the septo-hippocampal system*. Oxford: Oxford University Press.

Hair, J.F., Anderson, R.E., Tatham, R.L., Black, W.C. (1995). *Multivariate data analysis.* New York: Macmillan.

Jensen, M. P, Ehde, D. M, & Day, M. A. (2016). The behavioral activation and inhibition systems: implications for understanding and treating chronic pain. *Journal of Pain, 17*(5), 529e1-e18.

Harmon-Jones, E. (2004). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology, 67*, 51-76.

Jensen, M. P, Sole, E., Castarlenas, E., Racine, M., Roy, R., Miro, J & Cane, D. (2017). Behavioral inhibition, maladaptative pain cognitions, and function in patients with chronic pain. *Scandinavian Journal of Pain, 17*, 41-48

Jensen, M. P, Tan, G., Chua, S. M, & BSoc, M. H. C. (2015). Pain intensity, headache frequency, and the behavioural activation and inhibition systems. *Clinical Journal os Pain, 31*(12), 1068- 1074.

Jensen, M. P., Turner, P., Romano, J. M., & Fischer, L. D. (1999). Comparative reliability and validity of chronic pain intensity measures. *Pain, 83*,157-162.

Malenka, R.C., Nestler, E.J., & Hyman, S.E. (2009). *Molecular Neuropharmacology: A Foundation for Clinical Neuroscience*. New York: McGraw-Hill Medical.

May, A. (2008). Chronic pain may change the structure of the brain. *Pain, 137*, 7-15

Moore, R.A., Straube, S., Paine, J., Phillips, C.J., Derry, S., & McQuay, H.J. (2010). Fibromyalgia: Moderate and substantial pain intensity reduction predicts improvement in other outcomes and substantial quality of life gain. *Pain, 149*, 360-364.

Müller, R., Gertz, K.J., Molton, I.R., Terrill, A.L., Bombardier, C.H., Ehde, D.M., & Jensen, M.P. (2016). Effects of a tailored positive psychology intervention on well-being and pain in individuals with chronic pain and a physical disability: A feasibility trial. *Clinical Journal of Pain*, 32, 32-44.

Muris, P., Meesters, C., de Kanter, E., & Timmerman, P.E. (2005). Behavioural inhibition and behavioural activation system scales for children: relationships with Eysenc´s personality traits and psychopathological symptoms. *Personality and Individual Differences, 38*, 831–841.

Muris, P., Meesters, C., van den Hout, A., Wessels, S., Franken, I., & Rassin, E. (2007). Personality and temperament correlates of pain catastrophizing in young adolescents. *Child Psychiatry Hum Dev, 38*(3), 171-81.

Quartana, P. J., Campbell, C. M., & Edwards, R. R. (2009) Pain catastrophizing: a critical review. *Expert Review of Neurotherapeutics, 9*, 745–58.

Ramírez-Maestre C, & Esteve, R. (2015). A new version of the Impairment and Functioning Inventory for Patients with Chronic Pain (IFI-R). *The Journal of Injury, Function and Rehabilitation, 7*, 455-465.

Renee, L., & Cano, A. (2009). Comorbid Chronic Pain and Depression: Who Is at Risk? *The Journal of Pain, 10*(6), 619-627.

Saavedra-Hernánndez, M., Castro-Sánchez, A.M., Cuesta-Vargas, A.I., Cleland, J.A., Fernández-de-las-Peñas, C., & Arroyo-Morales, M. (2012). The contribution of previous episodes of pain, pain intensity, physical impairment, and pain-related fear to disability in patients with chronic mechanical neck pain. *American Journal of Physical Medicine & Rehabilitation, 91*,1070-1076.

Stubbs, B., Koyanagi, A., Thompsone, T., Veronese, N., Carvalho, A. F., Solomi, M., Mugisha, J., Schofield, P., Cosco, T., Wilson, N., & Vancampfort, D. (2016). The epidemiology of back pain and its relationship with depression, psychosis, anxiety, sleep disturbances, and stress sensitivity: Data from 43 low- and middle-income countries. *General Hospital Psychiatry, 43*, 63–70.

Tabachnick, B.G., & Fidell, L.S. (2007). *Using multivariate statistics* (5th ed.). Boston, MA: Pearson Education.

Van Damme, S., Lorenz, J., Eccleston, C., Koster, E.H.W., De Clercq, A., & Crombez, G. (2004). Fear-conditioned cues of impending pain facilitate attentional engagement. *Clinical Neurophysiology, 34,* 33-39.

Watson, D., Wiese, D., Vaidya, J., & Tellegan, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology, 76*, 820-838.

Woolf, C.J. (2010). What is this thing called pain? *The Journal of Clinical Investigation, 120*(11), 3742-3744.

Table 1.

Description of the study sample (N = 253).

Variable	Percent (N)
Marital Status	
Single	10% (25)
Married	58% (147)
Cohabiting	13% (32)
Divorced	13% (34)
Widowed	6% (15)
Highest level of education completed*	
Fewer than 6 years of education	15% (38)
Primary education	38% (95)
Secondary education	34% (85)
High school	13% (33)
Employment status*	
Working full- or part-time	39% (98)
Homemaker	15% (39)
Unemployed	20% (50)
Retired	25% (62)
Student	1% (3)

*Missing values in highest level of education completed (n = 2) and employment status (n = 1).

Table 2

Mean, standard deviations and correlations between variables of study

Variables	Mean (SD)	1	2	3	4
1. Pain intensity	6.52 (1.35)	-			
2. BIS (SPSRQ-20)	17.84 (7.64)	.15*	-		
3. BAS (SPSRQ-20)	12.46 (3.69)	-.14*	.36**	-	
4. Impairment (IFI-R)	9.75 (3.59)	.30**	.51**	.16*	-
5. Psychological Function (WHO-5)	11.11 (5.47)	-.32**	-.55**	-.12*	-.39**

Note: SPSRQ-20, 20-item Sensitivity to Punishment and Sensitivity to Reward Questionnaire; IFI-R, Impairment and Functioning Inventory for Patients with Chronic Pain; WHO-5, 5-item World Health Organization Well-Being Index.

*p < 0.05; **p < 0.01

Table 3.

Results of multiple regression analysis predicting pain-related impairment.

Step and variables	Total R^2	ΔR^2	*F* for model	$F\Delta$	β
1. Control variables	0.11	0.11	6.04*	6.04*	
Age					0.06
FM Diagnosis					0.14
LBP Diagnosis					-0.03
Limb Pain					-0.22
Sex					-0.06
2. Pain intensity (centered)	0.16	0.05	8.06*	16.27*	0.25*
3. BIS and BAS	0.33	0.17	15.51*	31.75*	
BIS scale (centered)					0.44*
BAS scale (centered)					0.03
4. Interactions	0.34	0.01	11.77*	1.53	
BIS × Pain intensity					0.12
BAS × Pain intensity					0.23
BIS × BAS					-0.61

Note: FM = Fibromyalgia; LBP = Low Back Pain

*p < 0.01

Table 4.

Results of multiple regression analysis predicting psychological function.

Step and variables	Total R^2	ΔR^2	*F* for model	$F\Delta$	β
1. Control variables	0.17	0.17	9.99*	9.99*	
Age					0.06
FM Diagnosis					-0.08
LBP Diagnosis					0.09
Limb pain					0.35
Sex					-0.08
2. Pain intensity (centered)	0.22	0.05	11.32*	15.11*	-0.24*
3. BIS and BAS	0.42	0.20	21.99*	42.44*	
BIS (centered)					-0.53*
BAS (centered)					0.17*
4. Interactions	0.44	0.02	16.05*	2.79	
BIS × Pain intensity					0.32
BAS× Pain intensity					-0.21
BIS × BAS					0.41

Note: FM = Fibromyalgia; LBP = Low Back Pain

*p < 0.01