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Individual differences in behavioral activation and cardiac vagal control influence affective startle modification☆

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HIGHLIGHTS

- BAS scores related to both withdrawal and approach systems in startle modification.
- BAS moderated the relation of HRV and startle modification.
- Startle and HRV scores were treated as continuous variables in regression models.
- This motivation-regulation interaction underlies stress coping and stress disorders.

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ABSTRACT

The startle response (SR) has a close relationship with stress responses. Startle modification (SRM) has been widely used to study stress disorders (e.g., posttraumatic stress disorder). The framework of the behavioral inhibition and activation systems (BIS/BAS) has been thought to correspond with withdrawal and approach motivational processes underlying affective SRM and can influence stress reactivity. Vagally-mediated cardiac activity as indexed by heart rate variability (HRV) has been associated with SRM and regulatory processes during stress. In the present study, the influence of individual differences in the BIS/BAS and resting HRV on affective SRM were examined. Eighty-six subjects viewed affective pictures while acoustic SR stimuli were delivered. Individual differences in motivation were measured by the BIS/BAS scales. The magnitude of SR was assessed as electromyographic activity of the SR eyeblink during pictures of different valences. Resting HRV was derived from electrocardiography. In contrast to previous studies, the present results showed that startle inhibition and potentiation were related to BAS and HRV, but not to BIS. There was also an interaction of BAS and HRV, indicating that the relationship between HRV and SRM strengthened as BAS scores decreased. The present findings suggest that BAS may relate to both withdrawal and approach, and trait stress reactivity is influenced by BAS and cardiac vagal activity. In addition, BAS moderates the relationship between cardiac vagal activity and SRM. These findings have both theoretical and practical implications for the study of SRM, stress disorders, and health.

The startle response (SR) is considered to be closely related to the prototypical *fight-or-flight* stress response [74]. The SR consists of a series of bodily movements that disengage an organism from ongoing activities and facilitates escape from a sudden, intense stimulus [35]. In humans, SR magnitude is typically assessed by the strength of the eyeblink reflex, as indexed by electromyography (EMG) of the *orbicularis oculi* muscle [28]. The SR can be viewed as a reaction to a *threat to control* and has aversive quality.

Because of this close relationship between the SR and stress, the SR has become a valuable tool for the study of acute and prolonged stress. SR modification (SRM) provides researchers with a well-controlled paradigm to study affective and attentional phenomena, and consequently has been widely used in the study of cognitive and emotional dysfunction in psychopathology. In SRM paradigms, the SR is modulated by preceding stimuli (e.g., the *prepulse inhibition*; [45]; *fear-potentiated startle*; [14]). Integrating previous findings, Lang and colleagues have found that the SR is systematically modulated by the affective valence of foreground stimuli (e.g., [49,72]). This phenomenon has been most commonly studied in a picture viewing paradigm, in which unpleasant pictures potentiate, and pleasant pictures diminish, the acoustic SR (i.e., elicited by a sudden burst of noise), as compared to neutral images.

Since the late 1980s, research on SRM and posttraumatic stress disorder (PTSD) has been growing, a trend that can be traced to clinical

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observations of exaggerated SR in combat survivors of World War II. Individuals with PTSD exhibit increased startle potentiation during danger cues and poor startle inhibition in the presence of safety cues [48,55,64]. Situational fear is a core characteristic of PTSD, and fear learning plays a central role in inhibiting recovery from trauma exposure [29], which might account for the exaggerated SR and the atypical SRM pattern in PTSD individuals [19].

1. Neural control of affective startle modulation

The neural pathways that mediate the affective SRM have been well documented. The amygdala, the dorsal periacqueductal gray, and the bed nucleus of the stria terminalis play key roles in SR enhanced by fear (for a review, see [24]). Davis et al. [24] proposed that the acoustic SR is affectively modulated by projections from the central nucleus of the amygdale to the nucleus reticularis pontis caudalis. The prefrontal cortex (PFC) is also important in affective startle responses: PFC deficits are associated with altered affective startle modulation [43]. PFC activation has also been associated with fear extinction in SRM [61].

The amygdalar role in SRM varies with different affective cues [24]. This finding is consistent with the *motivational priming* hypothesis that different systems are activated in appetitive and aversive contexts [49]. Collectively, these notions highlight the importance of the central nucleus of the amygdala and PFC in SRM, but they suggest that the motivational systems have separate neural substrates for SRM by positive and negative emotions.

The amygdala is also the focal point of stress responses; it is where sensory inputs are integrated and where autonomic (ANS) and endocrine responses to psychological stressors are initiated [51]. The amygdala is reciprocally connected to a wide range of frontal areas, which form frontal-limbic pathways. These frontal-limbic interactions serve as the neurophysiological substrates of primary and secondary appraisal during stress response. The amygdala has extensive connections to the hypothalamus and brainstem, which initiate ANS and endocrine stress responses. Amygdala kindling has been proposed as a neural mechanism for stress sensitization in PTSD ([1]; for a review, see [50]). According to this stress sensitization theory, traumatic experiences induce the amygdala-based fear circuit, resulting in hyper-excitability (i.e., stress sensitization) among people with PTSD. Overall, a top-down process occurring in the PFC regulates a lower-order process initiated by the amygdala, resulting in SRM and stress response. Because many neural structures involved in affective SRM and stress responding overlap, individual differences in affective SRM are expected to influence stress reactivity, and vice versa.

2. Individual differences in affective startle modification

Individual differences in affective SRM have been extensively studied. Various forms of psychopathology such as anxiety and stress disorders are associated with aberrant SRM (for a review, see [39]). Nonclinical samples show trait differences in sensitivity to emotional cues that can influence affective startle modification. For example, high-fear subjects show greater valence modulation of SR than do low-fear ones [75], and the traits of *harm avoidance*, *extraversion*, and *neuroticism* have been shown to mediate affective SRM [20].

Among those personality dimensions that have been associated with affective startle modification, the behavioral activation and inhibition system (BIS/BAS; [36]) has been held to fit well with the motivational priming hypothesis [42]. The BIS/BAS framework offers constructs that reflect individual differences in sensitivity to affective stimuli. BIS regulates aversive motivation; it is sensitive to signals of punishment, nonreward, novelty, and innate fear stimuli, and so inhibits behaviors that can lead to negative outcomes [36,37]. In contrast, BAS promotes approach behaviors and the experience of positive feelings. Hawk and Kowmas [42]) hypothesized that high BIS sensitivity leads to stronger startle potentiation, and high BAS sensitivity produces greater startle inhibition. Using Carver & White's [15] BIS/BAS scales to test this hypotheses, it was found that high BAS was associated with larger startle inhibition, but no relationship between BIS and startle potentiation emerged. An extension of this study showed that the Drive subscale of BAS contributed the most to enhanced startle inhibition [40]. Using a different BIS/BAS measure, Caseras et al. [16] found that both high- and low-BIS groups showed startle potentiation during blood-disgust images. However, startle potentiation during fearful images was found only in the high-BIS group. These results suggest an interaction between image content and individual differences in motivation.

BIS/BAS has also been connected to trauma-related outcomes of PTSD. Theoretically, BIS-regulated aversive motivation is related to anxiety and PTSD. Significant relations have been found among BIS sensitivity, experiential avoidance (EA), and PTSD [52]. Positive associations have also emerged between PTSD symptoms and BIS scores, and negative correlations have been reported between such symptoms and the BAS Drive and Reward Responsiveness subscales [58]. In this study, EA moderated the relation between PTSD symptoms and BIS, and interacted with the BAS Fun Seeking subscale, suggesting that PTSD results from the interaction of neurobiological factors (i.e., BIS/BAS) and self-regulatory processes (i.e., EA). In addition, BIS/BAS sensitivity is also related to experiential anxiety, a key component of stress disorders [47].

However, research on BIS/BAS sensitivity and frontal asymmetry indicates that BIS/BAS may not correspond with withdrawal and approach motivational systems. Some studies show an association of BAS sensitivity with left or bilateral frontal activation, but not the predicted relationship between BIS and right frontal activation [18,41,44]. These results reveal the need to reexamine the relationships among Gray's theory, BIS/BAS self-report measures, and motivational systems. Indeed, it is inconsistent to equate BIS with withdrawal in Gray's model [53]. BAS produces both rewarding approach and active avoidance behaviors, which are both controlled by the withdrawal system [30,37]. In addition, the BAS and withdrawal systems share neural components such as the amygdala and the basal ganglia, which are involved in affective startle modulation [26,37]. These discrepancies may account for the absence of a relationship between BIS and startle potentiation in Hawk and Kowmas [42].

In sum, the literature on startle modification and BIS/BAS is conflicted. First, although BIS/BAS relates to motivational systems, BIS may not be involved in affective startle modulation. Next, the degree to which self-report BIS/BAS measures reflect Gray's theory is unclear. Finally, studies have typically dichotomized BIS/BAS scores and have not directly examined the relationship between personality variables and physiological indices.

3. Startle modulation and cardiac vagal control

The study of heart rate variability (HRV) may help clarify the motivational and self-regulatory processes of stress responses and SRM. In general, stressors induce decreased HRV, and resting HRV might be an indicator of self-regulation during stress. Thayer and Lane [69] proposed a model of *neurovisceral integration* in which vagally-(i.e., parasympathetic) mediated HRV is related to the negative feedback circuits that play a crucial role in affective regulation. A substantial body of literature supports the relationship between vagally-mediated HRV and self-regulatory processes (see [31,70] for reviews). A few studies have examined cardiac vagal control in the context of SRM: higher resting HRV is related to greater SR habituation [34] and predicts smaller SR magnitude in individuals with panic disorder [54]. In particular, Ruiz-Padial et al. [62] found that whereas valence modulation of startle was most prominent in individuals with high resting HRV, those with low resting HRV failed to show the typical pattern of valence modulation. Resting HRV levels have also been associated with fear extinction as indexed by startle potentiation [56,73]. The inverse relationship between startle