

Transient Arousal-Driven or Valence-Driven? The Effects of Alerting Cues on the Attentional Boost Effect in Depression-Prone Individuals

Meina Zhang

Fujian Medical University

Xuanying Li

Fujian Medical University

Qinling Xie

Fujian Medical University

Yiyuan Wang

Fujian Medical University

Lingxin Liu

Fujian Medical University

Liujing Huang

Fujian Medical University

Fajie Huang

hfj0519@fjmu.edu.cn

Fujian Medical University

Research Article

Keywords: Attentional Boost Effect, depression proneness, alerting cue, emotional valence, transient arousal, memory enhancement

Posted Date: April 23rd, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9182085/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at BMC Psychology on September 7th, 2026. See the published version at <https://doi.org/10.1186/s40359-026-05313-8>.

Abstract

Background

Depression-related negative emotional bias is widely regarded as a core cognitive mechanism underlying the maintenance of depressive states. However, it remains unclear whether such biases disrupt fundamental cognitive enhancement processes driven by transient arousal. The Attentional Boost Effect (ABE) reflects a transient, phasic arousal mechanism triggered by target detection and is thought to operate relatively independently of emotional valence. This study examined whether ABE remains intact in depression-prone individuals and how alerting cues and emotional valence modulate this effect.

Method

A 2 (Group: depression-prone vs. healthy control) $\times$ 2 (Alerting cue: Cue vs. No-Cue) $\times$ 3 (Emotional valence: positive vs. neutral vs. negative) $\times$ 2 (Stimulus type: target vs. distractor) mixed design was employed. Eighty-one university students completed a classic ABE paradigm.

Result

(1) Both depression-prone and control participants exhibited a robust ABE, suggesting that arousal-driven encoding enhancement mechanisms remain functionally preserved in subclinical depression; (2) During encoding, a positivity advantage emerged under the No-Cue condition, whereas alerting cues significantly improved detection efficiency and attenuated valence-related differences; (3) During recognition, alerting cues amplified the ABE magnitude for emotional stimuli, with a particularly pronounced enhancement for positive information.

Conclusion

Depressive negative bias did not impair the fundamental arousal-regulation pathway. Instead, cognitive processing in depression proneness showed contextual modulation. Exogenous alerting cues may function as compensatory signals, optimizing attention allocation and memory enhancement. These findings provide direct behavioral evidence distinguishing valence-driven and transient arousal-driven cognitive enhancement pathways and offer theoretical support for personalized intervention strategies based on arousal modulation.

1 Introduction

Cognitive processing abnormalities related to depression are widely regarded as core features of the disorder. Among the most robust and consistently replicated findings is a negative bias in the processing of emotional information. A large body of research has demonstrated that, in both clinically depressed patients and individuals with subclinical depressive tendencies [1], this attentional bias is primarily manifested as difficulty disengaging from negative stimuli, a tendency to interpret ambiguous situations negatively, and a memory advantage for negative events [2–4]. For example, behavioral and cognitive neuroscience evidence indicates that depressed individuals are more readily captured by negative stimuli and show greater difficulty disengaging from them [5], while also exhibiting avoidance [6] or attenuated processing of positive information [7]. This persistent negative processing bias has been closely linked to deficits in emotion regulation and impaired cognitive flexibility [8], and is considered a key mechanism underlying the maintenance of depressive states.

Although emotional processing biases in depression have been well documented, an important yet insufficiently explored question is whether such biases extend to basic cognitive mechanisms that are relatively independent of emotional valence under normal conditions. More specifically, does depression-related negative bias interfere with memory enhancement processes driven by non-emotional arousal? Addressing this question is crucial for understanding the relationship between

emotional processing systems and general cognitive regulatory systems in depression. A central challenge lies in distinguishing between different types of cognitive enhancement mechanisms.

The Attentional Boost Effect (ABE) provides an important experimental paradigm for examining this issue. In the ABE paradigm, participants are required to encode background stimuli while performing a target detection task. Findings consistently show that background stimuli presented simultaneously with target detection events are remembered better than those paired with non-target events [9]. This phenomenon runs counter to predictions derived from traditional resource competition models of attention and is typically interpreted as reflecting the enhancement of information encoding by a transient alerting response elicited by target detection. Converging evidence suggests that the ABE is associated with phasic activation of the locus coeruleus–noradrenergic (LC–NE) system. Specifically, the transient arousal response triggered by target events enhances perceptual and encoding processes that are temporally synchronized with those events [10]. Accordingly, the ABE is generally regarded as a prototypical arousal-driven memory enhancement mechanism.

At the functional level, depression-related emotional bias and the arousal-based regulatory mechanism underlying the ABE may involve distinct processing pathways. Negative bias in depression is typically associated with relatively sustained, top-down abnormalities in emotional processing and regulation [11, 12], whereas the ABE primarily depends on transient, bottom-up neuromodulatory processes [13]. Although prior research has distinguished between the effects of valence and arousal on memory through different pathways [14], direct evidence remains lacking as to whether arousal-driven memory enhancement mechanisms remain intact in individuals vulnerable to depression. If depression-related bias disrupts transient arousal systems, this would suggest that the impact of depression extends beyond emotional domains to more fundamental cognitive mechanisms. Conversely, if the ABE remains stable in individuals with depressive tendencies, this would support the functional separability of distinct cognitive enhancement mechanisms.

Emotional valence itself has also been considered a key variable influencing the ABE; however, existing findings remain inconsistent. Some studies have suggested that highly arousing negative stimuli attenuate the ABE [15]. Other research has reported the presence of the ABE under both positive and neutral stimulus conditions, with no significant difference between them [16]. Still other studies have found that both positive and negative emotional stimuli may enhance the ABE [17, 18]. These mixed findings indicate that emotional valence may exert a complex modulatory influence on the ABE. Specifically, the effect of emotional attributes on the ABE may not represent a fixed effect, but rather may be moderated by individual differences or task context.

In addition, alerting state has been identified as an important factor influencing the ABE. Exogenous alerting cues can transiently elevate arousal levels and effectively orient individuals toward upcoming target events, thereby enhancing task expectancy and preparatory states, which in turn facilitate the allocation and release of attentional resources [19, 20]. Moreover, previous research has shown that manipulating alerting cues can significantly increase response speed and enhance the magnitude of the ABE [21]. However, whether alerting states further modulate the influence of emotional valence on the ABE—particularly whether such modulation produces compensatory or amplifying effects in individuals vulnerable to depression—has not yet been systematically examined.

Based on the foregoing theoretical and empirical considerations, the central question of the present study is whether depression-related negative emotional bias interferes with memory enhancement mechanisms driven by transient arousal. In other words, does the phasic alerting regulatory system underlying the ABE remain functionally intact in individuals with depressive tendencies? Furthermore, is the influence of emotional valence on the ABE modulated by exogenous alerting cues, and does such modulation exhibit distinct patterns in depression-prone individuals?

To address these questions, the present study systematically manipulated emotional valence and alerting state within the classic ABE paradigm [11]. By simultaneously examining target detection performance during encoding and ABE effects during recognition, we aimed to functionally distinguish between a “valence-driven emotional processing pathway” and a “transient arousal-driven cognitive enhancement pathway,” and to test their functional separability in individuals with depressive tendencies. If the ABE remains stable in this population, the findings would support the relative independence of arousal regulatory systems and emotional bias mechanisms. Conversely, disruption of the ABE would suggest that depression affects

more fundamental neuromodulatory pathways. Through this mechanistic dissociation, the present study seeks to provide new empirical evidence for understanding the relationship between emotional abnormalities and basic cognitive regulatory systems in depression.

2 Methods

2.1 Participants

This study was approved by the Biomedical Research Ethics Committee of Fujian Medical University (Approval No. 2025 – 212). To ensure adequate statistical power, an a priori sample size estimation was conducted using G*Power 3.1 [22]. The test family “repeated-measures ANOVA: within–between interaction” was selected, with parameters set at $f = 0.25$ (medium effect size), $\alpha = 0.05$, and $1 - \beta = 0.95$. The analysis indicated that a minimum total sample size of 18 participants (9 per group) was required.

As illustrated in Fig. 1, a total of 1,184 undergraduate students were recruited via the online survey platform “Wenjuanxing” for initial screening. The Beck Depression Inventory–II (BDI-II) was used as the screening instrument. Individuals with BDI-II scores ≥ 14 were assigned to the depression-prone group [23], whereas those with scores ≤ 13 were classified into the healthy control group. Ultimately, 83 eligible participants were enrolled in the formal experiment. Two participants whose recognition accuracy fell beyond three standard deviations from the overall sample mean were excluded from the final analyses. The final valid sample consisted of 81 participants, including 42 in the depression-prone group (20 males, 22 females) and 39 in the healthy control group (19 males, 20 females).

On the BDI-II, the depression-prone group (20.70 ± 5.47) scored significantly higher than the healthy control group (1.13 ± 1.52), $t(81) = -21.86$, $p < 0.001$. All participants were right-handed, had normal intelligence, and reported normal or corrected-to-normal vision without color blindness or color weakness. Upon completion of the experiment, each participant received RMB 15 as compensation.

To further exclude the possibility of clinical depression or other psychiatric disorders, all participants underwent a post-experimental diagnostic verification. Two rigorously trained graduate students in clinical psychology conducted structured interviews based on the Structured Clinical Interview for DSM-5 (SCID-5). Exclusion criteria included: (1) recent major stressful or traumatic events (e.g., bereavement); (2) bipolar disorder, major depressive disorder, anxiety disorders, or other psychiatric diagnoses; (3) a personal or family history of severe psychiatric disorders such as schizophrenia; (4) regular medication use or substance use potentially affecting the central nervous system; and (5) a history of neurological disorders. The results confirmed that none of the individuals in the depression-prone group met the diagnostic criteria for clinical depression, thereby ruling out the inclusion of clinically depressed participants.

2.2 Experimental Design

The study employed a 2 (Group: healthy control vs. depression-prone) $\times$ 2 (Alerting cue: present vs. absent) $\times$ 3 (Emotional valence: positive vs. neutral vs. negative) $\times$ 2 (Stimulus type: target vs. distractor) mixed factorial design. Group was a between-subjects factor, whereas alerting cue, emotional valence, and stimulus type were within-subjects factors.

The experimental task consisted of two phases: an encoding phase and a test phase. During the encoding phase, participants performed a target detection task. Behavioral performance was assessed using target detection accuracy and reaction time, reflecting task execution under different experimental conditions. During the test phase, the Attentional Boost Effect (ABE) was examined by comparing recognition performance for previously presented words paired with targets versus those paired with distractor during encoding.

In addition, a difference score (d) representing the magnitude of the ABE was calculated as: $d = \text{recognition accuracy for target-paired stimuli} - \text{recognition accuracy for distractor-paired stimuli}$. This index was used to quantify the strength of the ABE and to further analyze the moderating effects of alerting cues, emotional valence, and group on the ABE.

2.3 Materials

A total of 480 two-character Chinese emotional words were selected from the Chinese Affective Word System developed by Wang, Zhou [24], including 160 positive words (e.g., “兴,” lively), 160 neutral words (e.g., “身,” body), and 160 negative words (e.g., “丧,” despair). All words had been normatively rated on three dimensions: stroke count, emotional valence, and arousal, ensuring comparability across emotional categories.

Specifically, one-way analyses of variance (ANOVAs) were conducted with emotional valence (positive vs. neutral vs. negative) as the independent variable and each of the three indices as dependent variables. For stroke count, the main effect of valence was not significant, $F(2, 477) = 0.21, p > 0.05$, indicating that positive (17.88 ± 2.77), neutral (17.71 ± 2.63), and negative words (17.88 ± 2.78) were evenly matched in visual complexity. For emotional valence ratings, the main effect was significant, $F(2, 477) = 3313.38, p < 0.001$. Positive words (6.75 ± 0.25) were rated significantly higher than neutral words (5.10 ± 0.56), which in turn were rated significantly higher than negative words (3.18 ± 0.30), all $ps < 0.001$. For arousal, the main effect was also significant, $F(2, 477) = 14.54, p < 0.001$. Positive (4.99 ± 0.54) and negative words (5.06 ± 0.54) were rated as significantly more arousing than neutral words (4.76 ± 0.50), all $ps < 0.001$, whereas no significant difference was found between positive and negative words ($p > 0.05$). These results indicate that the three categories of two-character words were well matched in stroke count and clearly differentiated in valence and arousal, satisfying the requirements for experimental control.

Within each emotional valence category (positive, neutral, negative), words were randomly divided into two sets for the No-Cue and Cue experimental conditions, respectively. In each experimental task, 40 two-character words were presented during the encoding phase, including 20 target detection words (paired with a red circle) and 20 distractor rejection words (paired with a green circle). In the test phase, the 40 previously studied (“old”) words were randomly intermixed with 40 new words.

In addition, to increase cognitive task complexity and reduce predictability regarding target occurrence, 804 additional two-character words were selected from the same database as filler items. These filler words were presented only during the encoding phase and were neither included in the recognition test nor entered into the final data analyses.

2.4 Procedure

The experiment was programmed using E-Prime 2.0, following the paradigms described by Swallow and Jiang [9] and Huang, Guyang [21]. The experiment was conducted individually in a quiet behavioral laboratory cubicle using a 14-inch laptop computer (resolution: 1366 × 768). Participants completed the tasks using a standard keyboard according to on-screen instructions.

Each participant completed two experiments: a No-Cue condition and a Cue condition. The No-Cue experiment was always administered before the Cue experiment to control for order effects.

In the No-Cue experiment, two-character words were divided into three separate tasks based on emotional valence (positive, neutral, negative). Each task consisted of three phases: encoding, distraction, and test. Participants were allowed short breaks between tasks.

During the encoding phase, a two-character word appeared at the center of the screen with a colored circle (red or green) presented 1 cm below it. Both stimuli were displayed simultaneously for 100 ms, after which the circle disappeared while the word remained for an additional 400 ms, followed by a 500 ms blank screen. Participants were instructed to silently read each word while judging the color of the circle. They were required to press the spacebar when a red circle appeared (target detection) and withhold responses when a green circle appeared (distractor).

Participants completed 20 blocks, each containing 5 two-character words: 2 critical words and 3 filler words. Among the two critical words, one was a target detection word paired with a red circle and fixed at the third position of the sequence; the other was a distractor rejection word paired with a green circle and randomly presented at either the first or fifth position. The three filler words were all paired with green circles.

To minimize participants’ awareness of positional regularities and expectancy effects, 0–3 additional filler words were randomly inserted between blocks. All filler words were paired with green circles. The presentation sequence during encoding

was as follows: target and word presented simultaneously for 100 ms, circle disappeared while the word remained for 400 ms, followed by a 500 ms blank screen, and then the next trial commenced.

After the encoding phase, participants completed a serial subtraction-by-three task to prevent short-term memory rehearsal, and then proceeded to the test phase.

During the test phase, 40 previously studied words (20 target-paired and 20 distractor-paired) were randomly intermixed with 40 new words. Participants performed an old/new recognition task, pressing the “F” key for new words and the “J” key for old words. Words remained on the screen until a response was made.

The Cue experiment followed the same general procedure as the No-Cue experiment, with the addition of an orange circle during the encoding phase as an alerting cue. Participants were instructed not to respond to the orange circle. The appearance of the orange circle signaled that the subsequent word would be a target detection word (i.e., paired with a red circle). The cue stimulus always appeared immediately before the target word (i.e., at the second position in the sequence). The word presented simultaneously with the orange circle served as a cue word and was not included in the data analysis.

Prior to the formal experiment, all participants completed a practice session to familiarize themselves with the instructions and procedures. Practice data were not included in the final analyses. All two-character words were presented in white, 90-point bold font against a gray background, and the circles were displayed at a size equivalent to 120-point font. The overall experimental procedure is illustrated in Fig. 2.

2.5 Data Analysis

Data were analyzed according to the standard ABE procedure and consisted of three parts. First, performance on the target detection task during the encoding phase (accuracy and reaction time) was analyzed to assess participants’ processing characteristics for target words. Second, recognition accuracy during the test phase was examined to determine the effects of alerting cues, group, and emotional valence on overall recognition performance. Finally, the ABE magnitude was calculated as $d = \text{recognition accuracy for target-paired stimuli} - \text{recognition accuracy for distractor-paired stimuli}$, in order to investigate the moderating effects of these variables on the Attentional Boost Effect.

All statistical analyses were conducted using SPSS 29.0. Analytical methods included independent-samples t tests, mixed-design ANOVAs, simple effects analyses, and effect size estimation (partial η^2). To control for Type I error inflation due to multiple comparisons, the Least Significant Difference (LSD) method was used for exploratory pairwise comparisons of main effects. For simple effects analyses following significant interactions, the Šidák correction was applied. The Šidák procedure is suitable for a set of related comparisons and provides slightly greater statistical power than the Bonferroni correction while effectively controlling the familywise error rate (FWER) [25]. All comparisons in the present study followed these correction strategies to ensure statistical rigor and reliability.

3 Results

3.1 Encoding Phase: Analysis of the Target Detection Task

First, accuracy for the target detection task during the encoding phase was analyzed (see Table 1 and Figure 3). Across all conditions, target detection accuracy was significantly above chance level (0.50), $t_s > 14.38$, $p_s < 0.001$ indicating that participants were able to perform the target detection task effectively.

Table 1 Accuracy of the target detection task under different conditions.

Group	No-Cue			Cue		
	Positive	Neutral	Negative	Positive	Neutral	Negative
	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>
Healthy Control Group	0.91(0.10)	0.84(0.17)	0.81(0.17)	0.87(0.12)	0.84(0.16)	0.85(0.15)
Depression-Prone Group	0.88(0.14)	0.81(0.19)	0.76(0.19)	0.83(0.16)	0.84(0.14)	0.83(0.15)

A 2 (Group: healthy control vs. depression-prone) $\times$ 2 (Alerting cue: No-Cue vs. Cue) $\times$ 3 (Emotional valence: positive vs. neutral vs. negative) mixed ANOVA was conducted on target detection accuracy. The main effect of emotional valence was significant, $F(2,78) = 17.94$, $p < 0.001$, $\eta_p^2 = 0.32$. Accuracy for positive words was significantly higher than for neutral ($p < 0.001$) and negative words ($p < 0.001$), and neutral words were detected more accurately than negative words ($p = 0.04$).

The interaction between alerting cue and emotional valence was significant, $F(2, 78) = 14.45$, $p < 0.001$, $\eta_p^2 = 0.27$. Simple effects analysis revealed that under the No-Cue condition, accuracy for positive words was highest, and neutral words were detected more accurately than negative words ($p = 0.007$). Under the Cue condition, no significant differences were found among the three valence categories ($ps > 0.05$). The main effect of group was not significant, $F(1, 79) = 1.09$, $p = 0.31$.

Reaction times (RTs) in the target detection task during the encoding phase is presented in Table 2.

Table 2 Reaction times in the target detection task

.Group	No-Cue			Cue		
	Positive	Neutral	Negative	Positive	Neutral	Negative
	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>
Healthy Control Group	378.36(37.39)	393.25(33.94)	393.50(34.05)	211.40(64.14)	216.03(71.68)	209.62(80.51)
Depression-Prone Group	382.52(32.71)	400.14(35.92)	409.22(31.80)	238.01(68.11)	233.24(74.36)	234.31(77.60)

The same mixed ANOVA was conducted on RTs for correct responses. The main effect of alerting cue was significant, $F(1, 79) = 531.98$, $p < 0.001$, $\eta_p^2 = 0.87$, with shorter RTs in the Cue condition than in the No-Cue condition ($p < 0.001$). The main effect of emotional valence was also significant, $F(2, 78) = 6.38$, $p < 0.001$, $\eta_p^2 = 0.14$. RTs were faster for positive words than for neutral ($p = 0.006$) and negative words ($p = 0.001$), and faster for neutral than for negative words ($p = 0.001$).

The interaction between alerting cue and emotional valence was significant, $F(2, 78) = 8.06$, $p < 0.001$, $\eta_p^2 = 0.17$. Simple effects analysis showed that in the No-Cue condition, RTs for positive words were significantly faster than for neutral ($p < 0.001$) and negative words ($p < 0.001$), with no significant difference between neutral and negative words ($p > 0.05$). In the Cue condition, no significant RT differences were observed among the three valence categories ($ps > 0.05$). Neither the main effect of group nor related interactions reached significance.

In summary, under the No-Cue condition, individuals exhibited higher detection accuracy and faster RTs for positive words. Under the Cue condition, alerting cues significantly shortened overall RTs and reduced valence-related differences. No significant group effects were observed during the encoding phase.

3.2 Test Phase: Recognition Task Results

1. Overall Recognition Accuracy

Recognition accuracy for old words under different conditions is presented in Table 3.

Table 3 Accuracy in the old/new recognition task under different conditions.

Group	Trial Type	No-Cue			Cue		
		Positive	Neutral	Negative	Positive	Neutral	Negative
		<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>
Healthy Control Group	Target	0.36(0.19)	0.37(0.17)	0.30(0.16)	0.31(0.14)	0.34(0.17)	0.30(0.19)
	Distractor	0.26(0.14)	0.18(0.17)	0.19(0.12)	0.13(0.14)	0.21(0.15)	0.17(0.15)
Depression-Prone Group	Target	0.30(0.18)	0.33(0.21)	0.28(0.20)	0.29(0.17)	0.34(0.21)	0.30(0.19)
	Distractor	0.18(0.16)	0.18(0.15)	0.19(0.13)	0.16(0.13)	0.19(0.15)	0.14(0.11)

A 2 (Alerting cue: Cue vs. No-Cue) $\times$ 2 (Group: healthy control vs. depression-prone) $\times$ 3 (Emotional valence: positive vs. neutral vs. negative) $\times$ 2 (Stimulus type: target-paired vs. distractor-paired) mixed ANOVA was conducted on recognition accuracy for old words.

The main effect of stimulus type was significant, $F(1, 79) = 208.07, p < 0.001, \eta_p^2 = 0.73$. Recognition accuracy was significantly higher for target-paired words than for distractor-paired words, indicating a robust Attentional Boost Effect (ABE).

The main effect of emotional valence reached marginal significance, $F(2,78) = 3.04, p = 0.05, \eta_p^2 = 0.07$, with neutral words recognized more accurately than negative words ($p = 0.02$). The main effect of alerting cue showed a marginal trend, $F(1, 79) = 3.21, p = 0.08, \eta_p^2 = 0.39$, with higher recognition accuracy in the No-Cue condition compared to the Cue condition.

The three-way interaction among alerting cue, emotional valence, and stimulus type approached significance, $F(2,78) = 3.02, p = 0.05, \eta_p^2 = 0.07$. Simple effects analyses showed that both groups exhibited significant ABE effects under both Cue and No-Cue conditions (all $ps < 0.001$). For distractor-paired words, recognition accuracy for positive and negative words was higher in the No-Cue than in the Cue condition ($p < 0.001; p = 0.02$). In the Cue condition, neutral words were recognized more accurately than positive ($p = 0.02$) and negative words ($p = 0.03$).

For target-paired words in the No-Cue condition, neutral words were recognized more accurately than negative words ($p = 0.03$), whereas no significant differences were found between positive and neutral ($p = 0.76$) or positive and negative words ($p = 0.26$). Other higher-order interactions were not significant.

Overall, participants demonstrated a robust ABE across conditions, with higher recognition accuracy for target-paired than distractor-paired words. Recognition performance varied as a function of alerting cue, emotional valence, and stimulus type. In the No-Cue condition, recognition accuracy for positive and negative words was higher than in the Cue condition, suggesting that emotional valence exerted a stronger influence on memory when no alerting cue was present. Neutral words exhibited a recognition advantage in certain conditions, and alerting cues may have reduced emotional modulation by attenuating emotion-related attentional biases.

2. Analysis of ABE Magnitude

To more directly quantify the magnitude of the Attentional Boost Effect (ABE), an ABE index was computed for each condition by subtracting distractor-paired recognition accuracy from target-paired recognition accuracy ($d = \text{target-paired accuracy} - \text{distractor-paired accuracy}$). The descriptive statistics are presented in Table 4 and illustrated in Figure 4.

Table 4 Magnitude of the Attentional Boost Effect in the recognition task.

Group	No-Cue			Cue		
	Positive	Neutral	Negative	Positive	Neutral	Negative
	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>
Healthy Control Group	0.10(0.17)	0.19(0.14)	0.11(0.13)	0.17(0.15)	0.13(0.14)	0.13(0.15)
Depression-Prone Group	0.11(0.16)	0.15(0.17)	0.10(0.20)	0.13(0.14)	0.15(0.17)	0.16(0.19)

The ABE indices were submitted to a 2 (Alerting cue: Cue vs. No-Cue) $\times$ 2 (Group: healthy control vs. depression-prone) $\times$ 3 (Emotional valence: positive vs. neutral vs. negative) mixed-design ANOVA. The results revealed a significant interaction between alerting cue and emotional valence, $F(2, 78) = 3.02, p = 0.05, \eta_p^2 = 0.07$.

Simple-effects analyses indicated that in the No-Cue condition, the ABE magnitude for neutral words was significantly greater than that for both positive ($p = 0.04$) and negative words ($p = 0.01$), whereas no significant difference was observed between positive and negative words ($p = 0.99$). In contrast, under the Cue condition, no significant differences in ABE magnitude were found among the three emotional valence categories ($ps > 0.05$).

For positive words, the ABE magnitude differed significantly between Cue and No-Cue conditions ($p = 0.04$), with a larger ABE observed in the Cue condition. For negative words, a marginally significant trend suggested a greater ABE magnitude in the Cue condition compared to the No-Cue condition ($p = 0.08$). No significant cue-related difference was observed for neutral words ($p = 0.20$).

The three-way interaction among group, alerting cue, and emotional valence showed a marginal trend, $F(2, 78) = 2.44, p = 0.09, \eta_p^2 = 0.06$. Simple effects analysis revealed that in the No-Cue condition among healthy controls, the ABE magnitude for neutral words was greater than for positive ($p = 0.05$) and negative words ($p = 0.07$), with no difference between positive and negative words ($p > 0.5$). In the depression-prone group, no significant valence differences were observed ($p > 0.5$). In the Cue condition, neither group showed significant valence-related differences in ABE magnitude ($p > 0.5$).

In summary, the magnitude of the Attentional Boost Effect was not fixed but was modulated by the interaction between alerting cues and emotional valence. Specifically, valence-related differences in ABE were more pronounced under the No-Cue condition and attenuated under the Cue condition. The moderating effect of depressive tendency was generally weak, showing only trend-level influences.

4 Discussion

The present study systematically examined the performance of depression-prone individuals in the Attentional Boost Effect (ABE) paradigm by manipulating alerting cues and emotional valence. Several important findings emerged. First, depression-prone individuals exhibited a robust ABE, suggesting that the transient arousal-driven memory enhancement triggered by target detection remains intact in subclinical depression. Second, exogenous alerting cues reshaped emotional processing during encoding, eliminating valence-related differences observed under the No-Cue condition and reflecting an optimization of attentional allocation. Third, the magnitude of the ABE was jointly modulated by alerting state and emotional valence, indicating functional dissociation and dynamic interaction between arousal-driven mechanisms and emotion-bias mechanisms. Finally, depression proneness exerted only marginal moderating effects, implying that negative bias may be context-dependent rather than indicative of structural impairment within fundamental arousal systems.

4.1 Stability of Arousal-Driven Encoding Enhancement in Depression Proneness

Although depression proneness is frequently associated with negative attentional bias, memory dysfunction, and executive impairment [26], the present findings demonstrate that the ABE was not attenuated in the depression-prone group. On the

contrary, a reliable ABE was observed under target-detection conditions, indicating that the transient encoding enhancement triggered by behaviorally relevant events remains preserved in subclinical depressive states.

This finding carries important theoretical implications, as it challenges the generalized assumption that depression necessarily entails a broad impairment of cognitive enhancement mechanisms. According to the Dual-Task Interaction Model 2.0 (DTI 2.0), the ABE critically depends on phasic activation of the locus coeruleus-norepinephrine (LC-NE) system. Depression-prone individuals have often been reported to show reduced LC-NE functioning alongside heightened negative attentional bias [12, 27], which might be expected to weaken the ABE. However, our results suggest that depression proneness does not uniformly disrupt all attention-memory enhancement mechanisms.

A more nuanced interpretation is that depressive cognitive alterations may be selective rather than global. As a subclinical condition, depression proneness may reflect biases in processing priorities or regulatory inefficiencies rather than fundamental deficits in underlying neural mechanisms. From a neurocognitive perspective, emotional attentional bias in depression primarily involves dysregulated interactions within prefrontal-limbic circuits, including bottom-up affective pathways (e.g., amygdala, anterior cingulate cortex) and top-down control systems (e.g., dorsolateral prefrontal cortex) [28–30]. Functional connectivity reductions and impaired regulatory efficiency within these networks contribute to exaggerated emotional bias and difficulty resolving affective conflict [30, 31].

In contrast, the ABE is thought to rely predominantly on phasic LC-NE activation. Target detection elicits brief bursts of LC firing, resulting in widespread cortical norepinephrine release that transiently enhances encoding of temporally coincident information while suppressing irrelevant input [12, 32, 33]. Because this mechanism primarily depends on evolutionarily conserved brainstem-cortical alerting pathways rather than high-level executive control processes, it may remain functionally intact even when prefrontal regulatory systems are compromised.

Thus, the present findings support a functional dissociation between emotion-bias systems and arousal-driven encoding enhancement mechanisms. Even when depression-prone individuals show vulnerabilities in emotional regulation or cognitive control, their foundational arousal-mediated attentional gating system appears preserved, enabling a stable ABE. It should be noted, however, that LC-NE activity was not directly measured in this study; therefore, neural interpretations remain inferential and require future validation using physiological or neuroimaging indices

4.2 Emotional Modulation of Memory Enhancement Depends on Alerting State

A further key finding is that alerting cues substantially altered the processing of emotional information. Under the No-Cue condition, emotional valence differences were evident, reflecting spontaneous allocation biases. However, the introduction of alerting cues attenuated these valence-related differences while simultaneously improving overall processing efficiency and reaction speed. This pattern supports the view that emotional processing depends on limited cognitive resources and that arousal state critically determines how emotion influences memory.

According to cognitive resource theories, processing capacity is finite, and emotionally salient stimuli tend to capture disproportionate attention under low-control conditions [19]. In the No-Cue condition, individuals were in a relatively low preparatory state, allowing emotional valence to guide attentional allocation and generate observable bias effects. By contrast, alerting cues function as potent temporal expectation signals that activate the brain's alerting network prior to stimulus onset [34]. This preparatory activation enhances top-down task readiness, concentrating resources on task-relevant dimensions and thereby counteracting interference from emotionally salient but task-irrelevant information.

This shift can also be interpreted through the cue-utilization theory of Easterbrook [35]. Under heightened arousal (Cue condition), attentional focus narrows toward central task-relevant cues, reducing processing of peripheral emotional information. The cognitive mode thus shifts from valence-driven allocation to task-driven prioritization. Importantly, the present results further suggest that alerting cues do not simply suppress emotional processing. Rather, they reorganize attentional gating priorities. When emotional information becomes temporally aligned with task-relevant events, its encoding can be

selectively strengthened under heightened alertness. In this sense, alerting cues recalibrate rather than eliminate emotional influences.

Taken together, the emotional modulation of the ABE appears highly dependent on background alerting state. Alerting cues simultaneously reduce irrelevant emotional interference and deepen processing of target-relevant information. This dual-gain mechanism has important implications for understanding attentional bias in depression proneness. By externally modulating arousal levels, it may be possible to reduce excessive reactivity to irrelevant negative stimuli while enhancing goal-directed attentional focus. This mechanism parallels everyday experiences in which intense task engagement diminishes subjective awareness of negative emotional distractions.

4.3 Depression Proneness as a Context-Triggered Rather Than Persistent Cognitive Bias

A notable finding of the present study is the absence of a significant main effect of group, alongside the clear context-dependent pattern of depression-proneness effects. Specifically, under the Cue condition, depression-prone and healthy control participants did not differ reliably in task performance or ABE magnitude. In contrast, under the No-Cue condition, emotional differences in ABE became more pronounced, and group-related tendencies were more likely to emerge. This pattern suggests that depression proneness may not reflect a chronically active processing bias, but rather a vulnerability that becomes expressed when cognitive control support is reduced.

This interpretation aligns with state-dependent cognitive models of depression. Previous research has shown that depressive cognitive biases are more likely to manifest when executive control resources are limited or when environmental structure is weak [36]. In contexts lacking exogenous guidance, individuals must rely more heavily on endogenous control to maintain task goals. Because depression-prone individuals often exhibit elevated rumination and increased cognitive resource depletion, their limited executive resources may be more easily captured by emotionally salient processing, thereby amplifying bias effects. By contrast, in the Cue condition, explicit temporal structure and exogenous alerting signals likely enhanced attentional coordination efficiency, effectively normalizing performance in the depression-prone group.

Importantly, the moderating effects of depression proneness in this study were generally modest and often limited to trend-level differences. This pattern indicates that subclinical depressive vulnerability does not necessarily produce stable cognitive impairment, but rather increases sensitivity to task-control demands. In other words, depression proneness may primarily affect the resilience of cognitive control systems rather than induce a constant bias toward emotional stimuli.

From a neurocognitive perspective, this context dependence is theoretically plausible. Depression-related abnormalities are frequently observed in sustained regulatory networks, such as the dorsolateral prefrontal cortex (dlPFC) and rostral anterior cingulate cortex (rACC) [30], whereas the ABE is thought to rely primarily on transient arousal-driven mechanisms. According to the locus coeruleus–norepinephrine (LC–NE) model, target detection triggers phasic LC activation that enhances encoding of temporally coincident information [37]. If this phasic arousal system remains relatively intact in depression-prone individuals, exogenous alerting signals may compensate for limitations in sustained control resources. This explanation further supports the functional dissociation between emotion-bias systems and arousal-driven encoding enhancement mechanisms.

Taken together, the findings support a state-dependent framework: depression-prone individuals may maintain relatively normative cognitive performance in structured, task-driven environments, yet become more vulnerable to processing biases under low-structure or low-alert conditions. Such context sensitivity may help explain fluctuations commonly observed in depressive functioning, whereby individuals perform adequately under high task demands but experience greater emotional-cognitive difficulties during periods of reduced vigilance or control.

4.4 Clinical Implications

The present findings carry important implications for both theoretical modeling and clinical translation.

First, at the level of cognitive theory, the results demonstrate that depression does not universally impair all enhancement-related cognitive mechanisms. The remarkable stability of the arousal-driven attention–memory mechanism (i.e., ABE) in

subclinical depression highlights the heterogeneity of depressive cognitive functioning. Future cognitive models of depression should explicitly distinguish between sustained control systems and transient arousal mechanisms. In particular, incorporating the concept of dynamic attentional gating may help explain why negative bias becomes more pronounced under low-control states (e.g., rumination, fatigue), rather than representing a fixed processing trait. Recognizing this state dependency enables more precise identification of cognitive vulnerabilities and their triggering conditions.

Second, from an intervention perspective, the findings suggest a promising “indirect pathway” strategy for cognitive remediation. Because depression-prone individuals retain sensitivity to exogenous alerting signals, therapeutic protocols may capitalize on this relatively preserved, automatic arousal pathway to enhance treatment efficacy. For example, cognitive training or positive information encoding paradigms could incorporate brief exogenous alerting inductions (e.g., salient auditory tones or visual flashes). By transiently elevating arousal, such manipulations may promote prioritized encoding of adaptive or therapeutic content, effectively bypassing fatigued or inefficient prefrontal regulatory systems.

This arousal-based modulation approach could enhance cognitive restructuring efficiency and provides an empirical basis for personalized digital interventions, such as mobile applications incorporating adaptive alerting cues. Conceptually, this strategy reframes intervention not as direct suppression of negative bias, but as optimization of attentional state to reshape encoding priorities.

4.5 Limitations and Future Directions

Despite revealing the robustness and multi-level modulation of the ABE in depression proneness, several limitations warrant consideration.

First, the issue of sample representativeness should be noted. Participants were university students with subclinical depressive tendencies rather than clinically diagnosed patients. Subclinical and clinical depression may differ qualitatively in cognitive impairment severity and neurobiological alterations (e.g., degree of LC–NE dysfunction). Therefore, generalization to clinically depressed populations should be made cautiously. Future studies should include clinically diagnosed groups to examine whether ABE patterns vary across severity levels and illness stages.

Second, limitations of the experimental paradigm should be acknowledged. The present study employed word-based stimuli, without examining cross-modal materials such as images or sounds. Additionally, the alerting cue manipulation was relatively fixed and did not systematically vary cue intensity, timing, or sensory modality. Future research should incorporate more ecologically valid stimuli (e.g., emotional faces or real-life scenes) and develop dynamic alerting manipulations to better delineate the boundary conditions of the ABE.

Third, emotional dimensions were primarily operationalized through valence, without fully dissociating arousal level, semantic concreteness, or personal relevance. These variables may independently modulate encoding and ABE magnitude. More refined stimulus sets with multidimensional parameter control are needed to disentangle the complex interaction between emotional properties and transient arousal mechanisms.

Finally, the absence of physiological measures limits mechanistic inference. The ABE is theorized to involve phasic LC–NE activation and dynamic interactions with prefrontal control networks. Future research should combine behavioral paradigms with event-related potentials (ERP), functional magnetic resonance imaging (fMRI), pupillometry, or eye-tracking techniques to clarify the temporal dynamics and neural circuitry underlying ABE modulation in depression-prone populations.

5 Conclusion

The present study systematically investigated the Attentional Boost Effect (ABE) in depression-prone individuals under varying alerting states and emotional valence conditions. The results indicate that a robust ABE persists in the absence of alerting cues, suggesting that transient arousal-driven encoding enhancement remains relatively intact in subclinical depression. Furthermore, exogenous alerting cues exert stage-specific modulation effects, altering how emotional valence influences memory processing.

Overall, the ABE in depression-prone individuals demonstrates pronounced contextual sensitivity, jointly shaped by alerting state and emotional characteristics. These findings provide novel behavioral evidence for understanding attention–memory interactions in depressive vulnerability and offer an empirical foundation for future investigations into arousal-based cognitive mechanisms and intervention strategies.

Declarations

Ethics approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. This study was approved by the Biomedical Research Ethics Committee of Fujian Medical University (Approval No. 2025-212). All participants provided written informed consent.

Consent for publication

Not applicable.

Data availability

The data are available from the corresponding author on reasonable request.

Competing interests

The authors declare no competing interests.

Funding

This study was funded by the 2025 Fujian Provincial Science and Technology Program Project [Grant No. 2025J01850].

Authors' contributions

Conceptualization, Methodology, Validation, and Formal Analysis, F.H. M.Z. and X.L.; Software, Investigation, M.Z. Q.X. Y.W. and L.L.; Resources, F.H.; Data Curation, M.Z. X.L. and Q.X.; Writing—Original Draft Preparation, M.Z. X.L. and F.H.; Writing—Review and Editing, F.H. M.Z. and X.L.; Supervision, F.H. M.Z. and X.L; Project Administration, F.H.; Funding Acquisition, F.H. All authors have read and agreed to the published version of the manuscript.

Acknowledgments

We thank all of the participants for their willingness to participate in the research.

References

1. Jiang Y. A theory of the neural mechanisms underlying negative cognitive bias in major depression. *Frontiers in Psychiatry*. 2024;15:1348474.
2. Urban EJ, T CS, J LL, M AD. Depression history and memory bias for specific daily emotions. *PloS one*. 2018;13(9):e0203574.
3. Suslow T, Hußlack A, Kersting A, Bodenschatz CM. Attentional biases to emotional information in clinical depression: A systematic and meta-analytic review of eye tracking findings. *Journal of Affective Disorders*. 2020;274(prepublish):632-42.
4. Everaert J, Podina IR, Koster EHW. A comprehensive meta-analysis of interpretation biases in depression. *Clinical Psychology Review*. 2017;58:33-48.
5. Klawohn J, Alec B, Kreshnik B, Alexandria M, Amit L, Yair B-H, et al. Aberrant attentional bias to sad faces in depression and the role of stressful life events: Evidence from an eye-tracking paradigm. *Behaviour Research and Therapy*. 2020;135:103762-.

6. Winer ES, Salem T. Reward devaluation: Dot-probe meta-analytic evidence of avoidance of positive information in depressed persons. *Psychological bulletin*. 2016;142(1):18-78.
7. Gotlib IH, John J, Martin B, Jutta J. Memory for affectively valenced and neutral stimuli in depression: evidence from a novel matching task. *Cognition & emotion*. 2011;25(7):1246-54.
8. Duque A, Vázquez C. Double attention bias for positive and negative emotional faces in clinical depression: Evidence from an eye-tracking study. *Journal of Behavior Therapy and Experimental Psychiatry*. 2015;46:107-14.
9. Swallow KM, Jiang YV. The Attentional Boost Effect: Transient increases in attention to one task enhance performance in a second task. *Cognition*. 2009;115(1):118-32.
10. Swallow KM, Jiang YV. Attentional load and attentional boost: a review of data and theory. *Frontiers in psychology*. 2013;4:274.
11. Swallow KM, Jiang YV. Goal-relevant events need not be rare to boost memory for concurrent images. *Attention, perception & psychophysics*. 2012;74(1):70-82.
12. Swallow KM, W. BA, Elizabeth R, B. TH. Grounding the Attentional Boost Effect in Events and the Efficient Brain. *Frontiers in Psychology*. 2022;13:892416-.
13. Villalobos D, Pacios J, Vázquez C. Cognitive Control, Cognitive Biases and Emotion Regulation in Depression: A New Proposal for an Integrative Interplay Model. *Frontiers in Psychology*. 2021;12:628416-.
14. Rouhani N, Yael N, J FM, Lars S. Multiple routes to enhanced memory for emotionally relevant events. *Trends in cognitive sciences*. 2023;27(9):867-82.
15. Meng Y, Zheng S, Wang D, Nie A. Limits to the Attentional Boost Effect: the Moderating Influence of Negative Emotion. *Journal of Psychological Science*. 2018;41(02):298-304.
16. Zhou X, Meng Y. The Influence of Positive Emotion on the Attentional Boost Effect. *Journal of Weifang Engineering Vocational College*. 2020;33(04):74-80.
17. Clelia R-A, Pietro S, Marco C, Daniele S, Vincenzo C. Divided attention enhances the recognition of emotional stimuli: evidence from the attentional boost effect. *Memory (Hove, England)*. 2018;26(1):42-52.
18. Duan Y, Xin T, Yin Y, Sun Y, Liu W, Chen N. Attentional boost effect: research based on source memory and emotional materials. *Psychological research*. 2024;88(8):1-14.
19. Nobre A, Correa A, Coull J. The hazards of time. *Current Opinion in Neurobiology*. 2007;17(4):465-70.
20. Petersen SE, Posner MI. The Attention System of the Human Brain: 20 Years After. *Annual Review of Neuroscience*. 2012;35(1):73-89.
21. Huang F, Guyang L, Meng Y, Lin Y, Zheng S. The role of alerting in the attentional boost effect. *Frontiers in psychology*. 2023;14:1075979-.
22. Faul F, Edgar E, Albert-Georg L, Axel B. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods*. 2007;39(2):175-91.
23. Li H, Yang X, Zheng W, Wang C. Emotional regulation goals of young adults with depression inclination: An event-related potential study. *Acta Psychologica Sinica*. 2019;51(6):637-.
24. Wang Y, Zhou L, Luo Y. The Pilot Establishment and Evaluation of Chinese Affective Words System. *Chinese Mental Health Journal*. 2008(08):608-12.
25. Daniells E. Simultaneous inference in clinical trials: Simultaneous confidence intervals for treatment effects in subgroups 2020.
26. Takagaki K, Okamoto Y, Jinnin R, Mori A, Nishiyama Y, Yamamura T, et al. Behavioral characteristics of subthreshold depression. *Journal of Affective Disorders*. 2014;168:472-5.
27. Li H, Lu J, Zhang Q, Qiu J. Difficulty in Disengaging Attention from Emotionally Negative Stimuli among Individuals with Sub-threshold Depression. *Psychological Development and Education*. 2016;32(05):513-20.
28. Šimić G, Mladenka T, Vana V, Damir M, Ena Š, Marina Š, et al. Understanding Emotions: Origins and Roles of the Amygdala. *Biomolecules*. 2021;11(6):823-.

29. Caterina G, Haoxin S, Petersen SE. Control networks and hubs. *Psychophysiology*. 2018;55(3):e13032.
30. Fales CL, M BD, M RM, A MM, Z SA, D CJ, et al. Altered emotional interference processing in affective and cognitive-control brain circuitry in major depression. *Biological psychiatry*. 2008;63(4):377-84.
31. Etkin A, F SA. Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *The American journal of psychiatry*. 2011;168(9):968-78.
32. Hirsch P, Nolden S, Declerck M, Koch I. Common Cognitive Control Processes Underlying Performance in Task-Switching and Dual-Task Contexts. *Advances in cognitive psychology*. 2018;14(3):62-74.
33. Wu Y, Sui G, Cao X. ERP Studies of Endogenous and Exogenous Attention. *Advances in Psychological Science*. 2007(01):71-7.
34. Kim AJ, Nguyen K, Mather M. Eye movements reveal age differences in how arousal modulates saliency priority but not attention processing speed. *bioRxiv : the preprint server for biology*. 2024.
35. Easterbrook JA. The effect of emotion on cue utilization and the organization of behavior. *Psychological review*. 1959;66 3:183-201.
36. Joormann J, D C, #39, Avanzato. Emotion regulation in depression: Examining the role of cognitive processes. *Cognition and Emotion*. 2010;24(6):913-39.
37. Healey MK, Ngo KWJ, Hasher L. Below-Baseline Suppression of Competitors During Interference Resolution by Younger but Not Older Adults. *Psychological Science*. 2014;25(1):145-51.

Figures

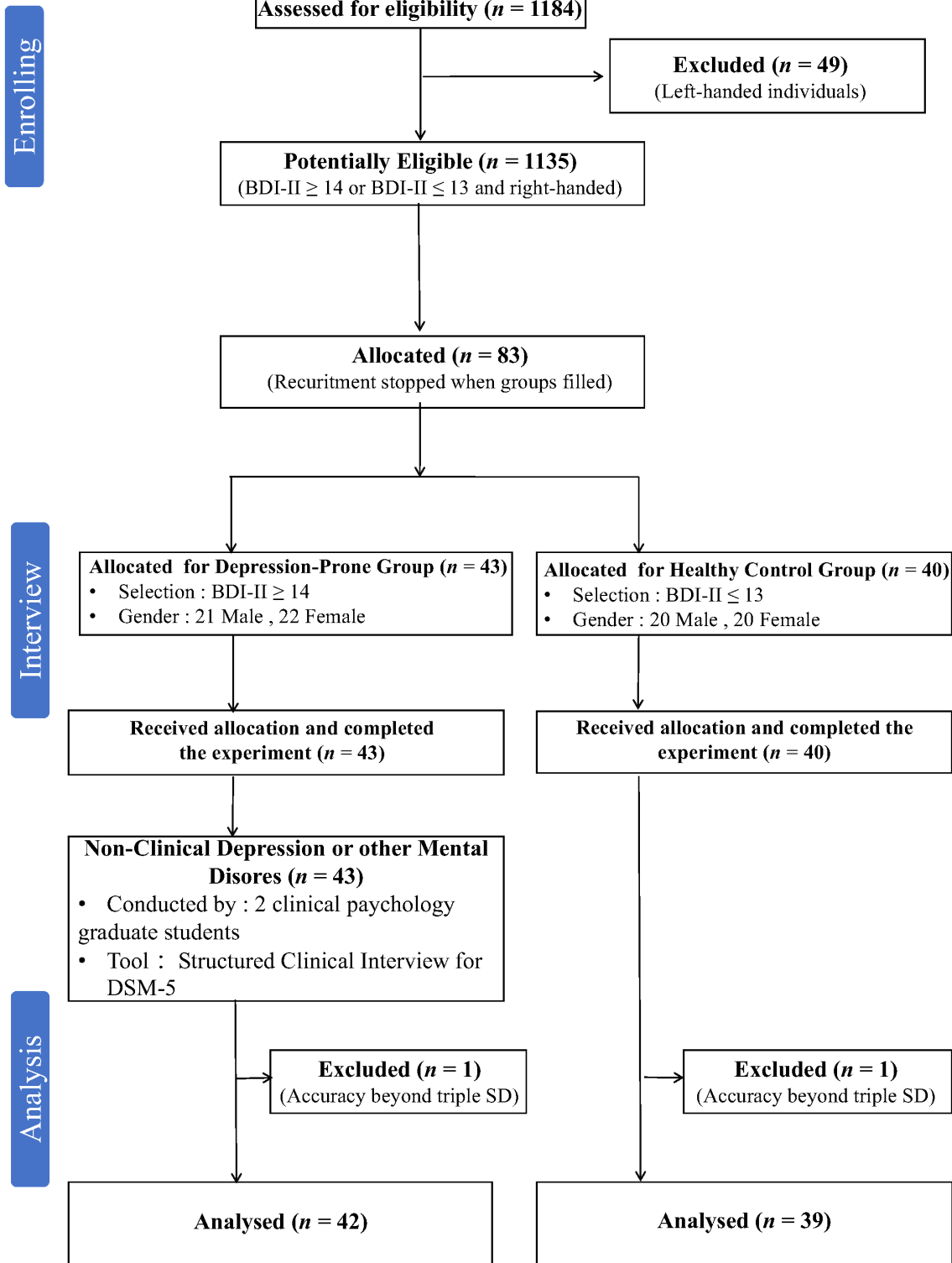
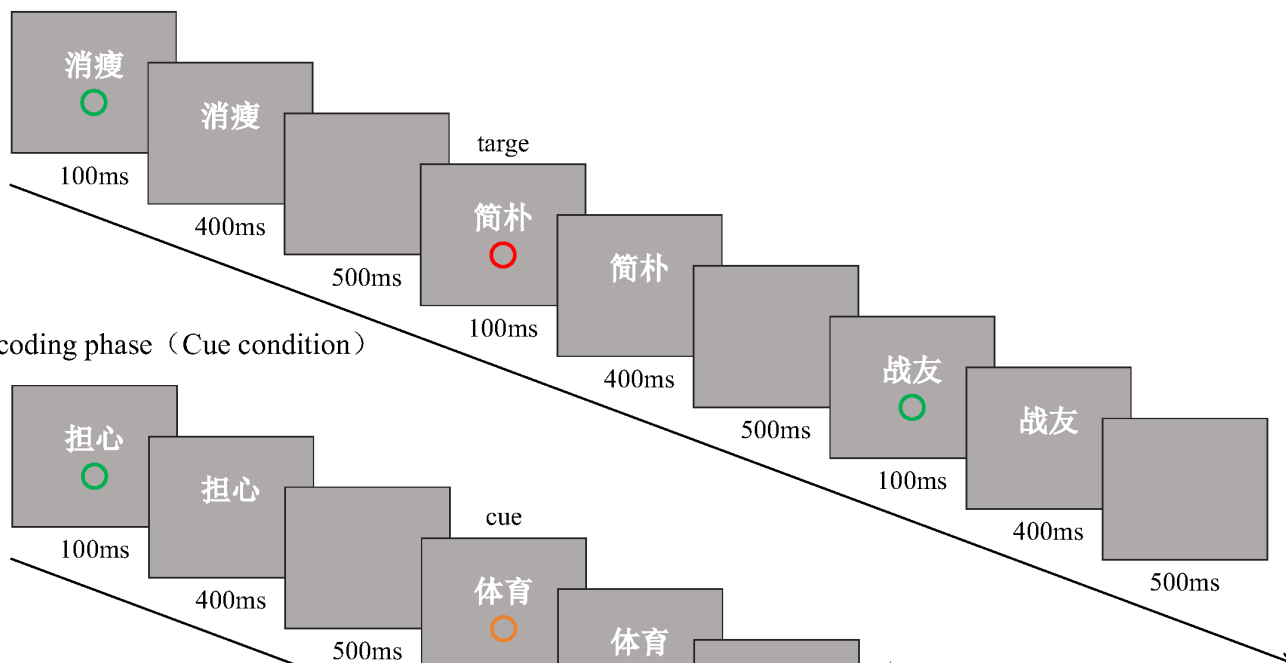


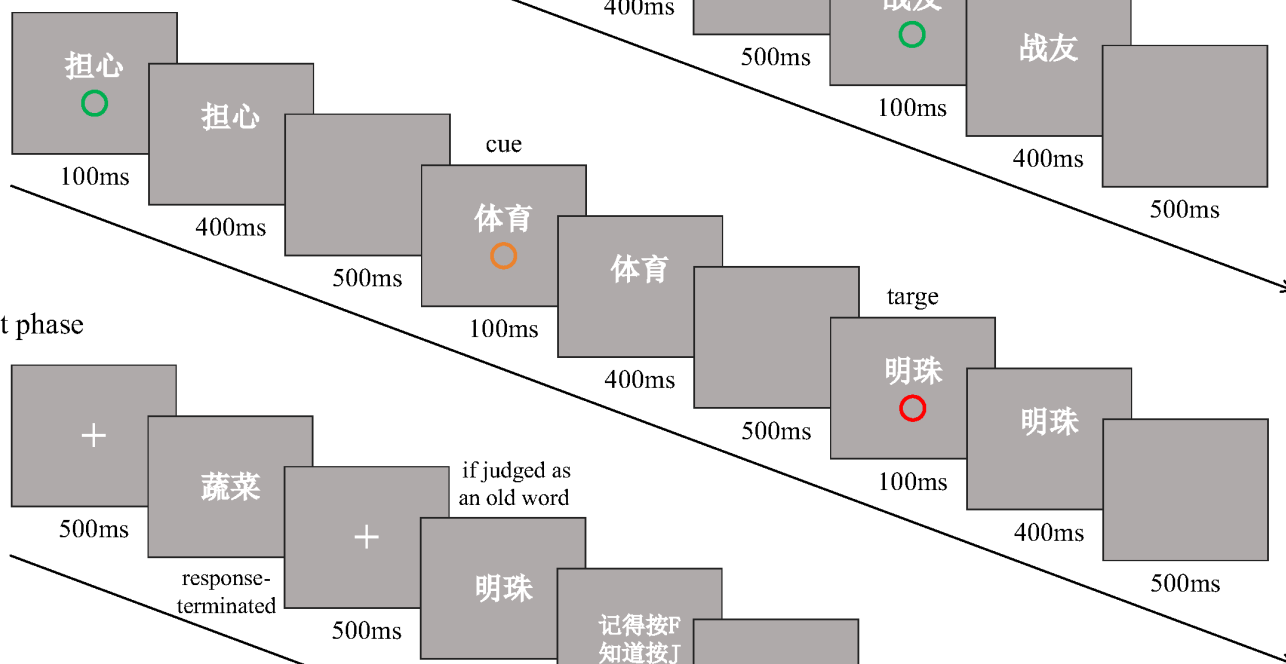
Figure 1

Flowchart of participant recruitment and inclusion.

encoding phase (No-Cue condition)



encoding phase (Cue condition)



test phase

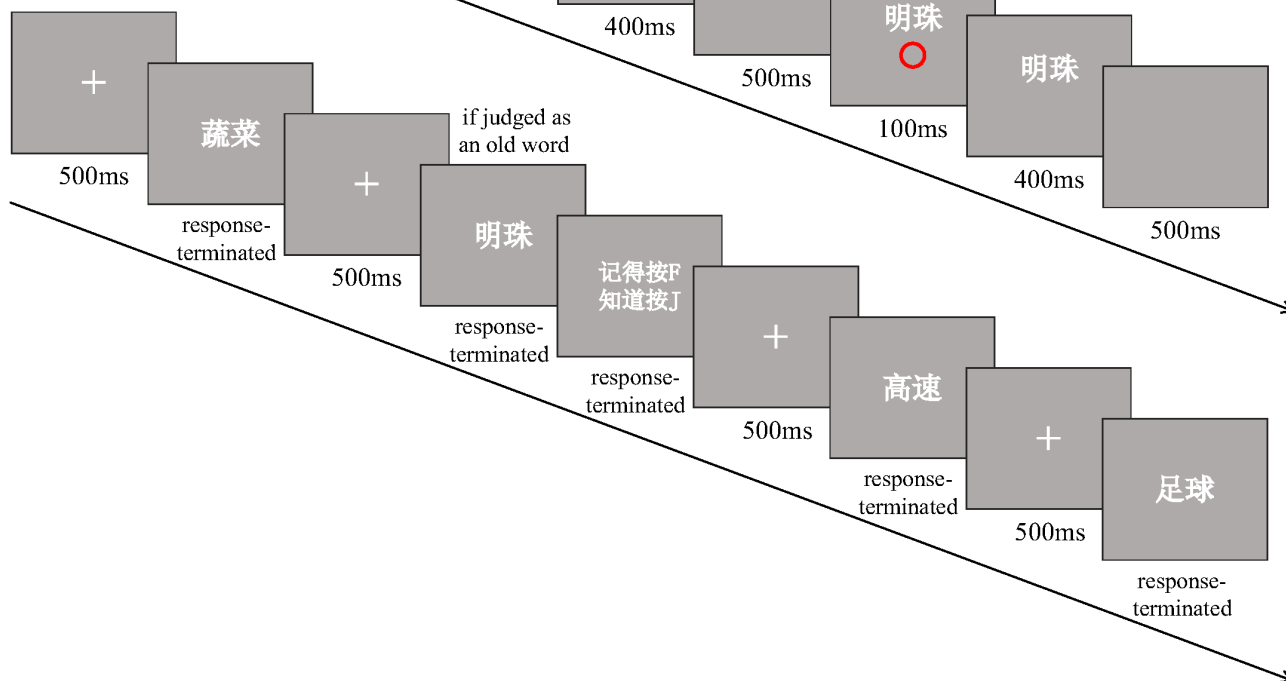


Figure 2

Schematic diagram of the encoding and test phases.

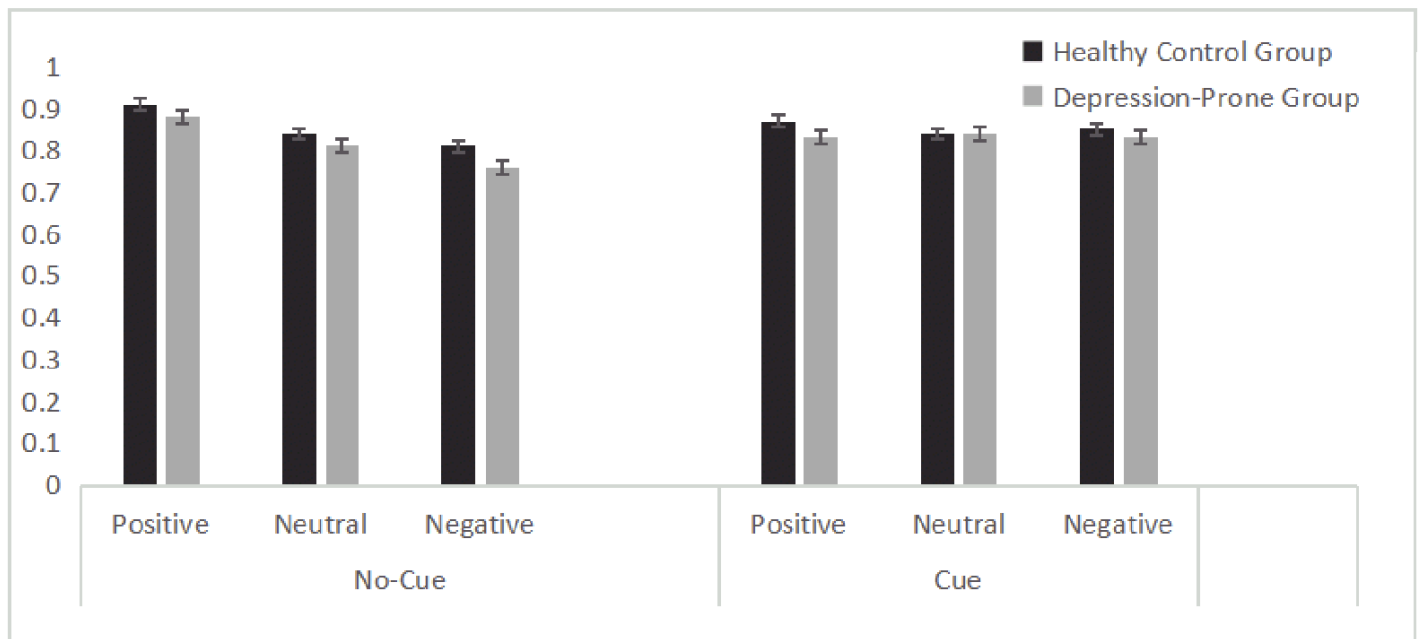


Figure 3

Accuracy of the target detection task.

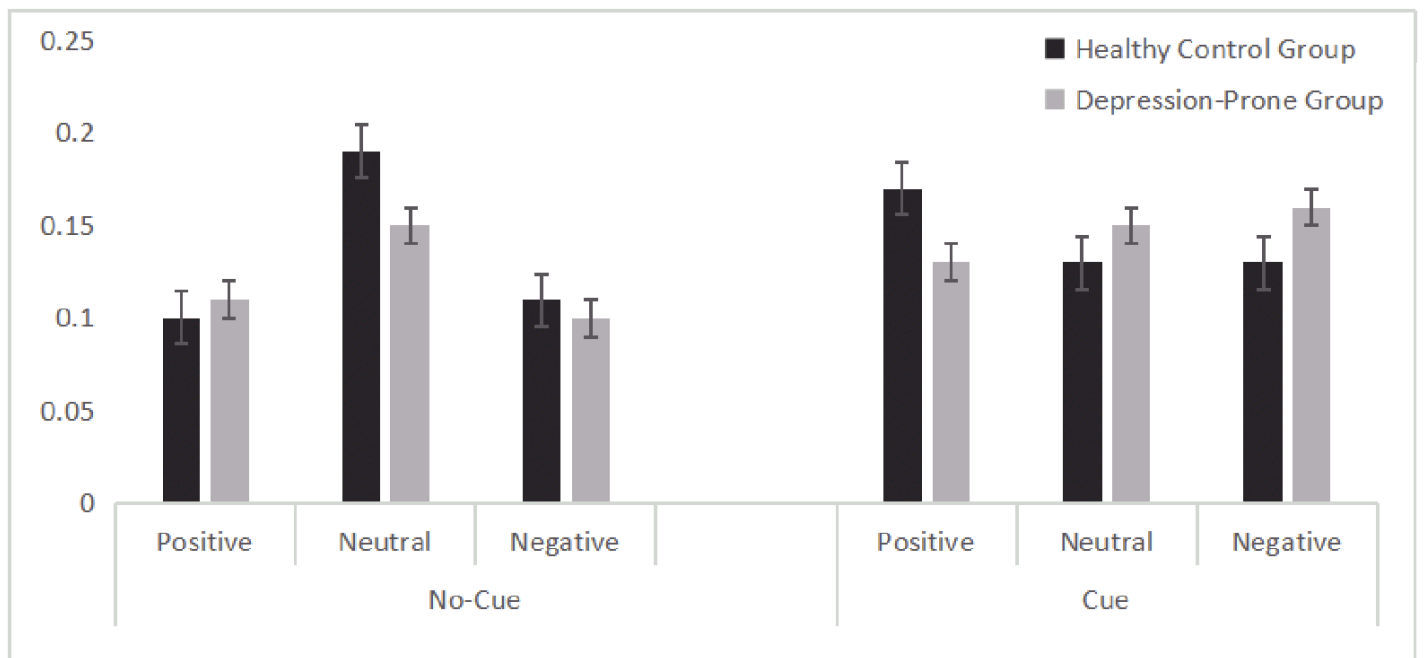


Figure 4

Magnitude of the Attentional Boost Effect.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [data.7z](#)