Personality and Individual Differences 259 (2026) 113871

Contents lists available at ScienceDirect

Personality and Individual Differences

journal homepage: www.elsevier.com/locate/paid

Sadness-as-information: What is its role in the maintenance of depressive beliefs?

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ARTICLE INFO

Keywords:
Sadness
Affect-as-information
Depression
Depressive realism
Performance evaluation
Cognitive bias

ABSTRACT

A central question in clinical cognitive psychology concerns why dysfunctional beliefs persist despite corrective evidence. The present research examined whether emotional states function as informational cues in evaluating one's own performance, according to the affect-as-information framework, and how this process might contribute to the maintenance of maladaptive depressive beliefs. Two experiments were conducted. Study 1 extended Scott and Cervone's (2002) paradigm to individuals reporting current sadness, categorized by BDI scores into high and low depression groups. Study 2 replicated this paradigm using emotion induction (sadness, anxiety, and neutral conditions) to isolate the specific role of sadness in shaping performance standards and satisfaction. It was hypothesized that both the subclinical and induced-sadness groups would use their emotional state as information, resulting in higher performance standards and lower satisfaction compared to controls. Contrary to expectations, participants in the subclinical and sadness conditions reported lower performance standards and similar levels of satisfaction relative to controls. These findings suggest that sadness may recalibrate, rather than inflate, self-evaluative standards, promoting a closer correspondence between expectations and actual outcomes. Such adjustment may underlie the phenomenon of depressive realism and contribute to the stability of self-evaluative beliefs in depression. Theoretical and clinical implications regarding the affect-as-information mechanism and cognitive maintenance processes in depressive disorders are discussed.

1. Introduction

Understanding the mechanisms that maintain dysfunctional beliefs and psychological disorders, as well as their resistance to change, remains a central question in clinical cognitive science. This paper seeks to address this issue by examining the role of emotions in cognitive processes and how they may reinforce the beliefs underlying emotional experiences in psychopathology.

Our discussion is grounded in appraisal theories of emotion, which posit that both typical and pathological emotional responses stem from an individual's subjective evaluation of the personal significance of a given situation, object, or event along multiple dimensions (e.g. Scherer, 1999). We specifically aim to investigate how emotions influence beliefs—understood here as mental representations, cognitions, or assumptions—and how such influence might contribute to the persistence of some specific psychological disorders, such as mood disorders.

2. The affect-as-information mechanism and emotional reasoning

The terms *Affect-as-Information*, *Emotional Reasoning* (ER), and *ex consequentia reasoning* refer to a psychological process whereby individuals interpret their emotional states as informative cues about external reality. Importantly, this may occur even when the affective response is incidental and unrelated to the situation being evaluated. Accordingly, affect-as-information theory proposes that people often

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https://doi.org/10.1016/j.paid.2026.113871
Received 11 November 2025; Received in revised form 22 April 2026; Accepted 28 April 2026
Available online 1 May 2026

rely on their feelings as a source of evidence when forming judgments and evaluations, sometimes privileging subjective affect over objective information (Arntz et al., 1995; Schwarz & Clore, 1983).

Within this framework, affect is treated as a diagnostic signal that can shape evaluative cognition, particularly when individuals perceive their current emotional state as relevant to the judgment at hand (Clore, 1992; Schwartz & Clore, 1988). From a broader motivational and regulatory perspective, this view is consistent with recent formulations such as the *Affective Gradient Hypothesis* (Shenhav, 2024), which conceptualizes affective fluctuations as informational gradients that guide behavioral regulation and evaluative decisions. Although developed in a different theoretical context, Shenav's account converges with affect-as-information models in emphasizing the adaptive and functional role of affect in directing cognition and action.

Similarly, Forgas' affect infusion model highlights that reliance on affective states is particularly likely under conditions of low motivation, limited cognitive resources, or heuristic processing (Forgas, 2017). In such circumstances, individuals may implicitly use their current mood as a heuristic cue, effectively treating affect as information about the target of judgment, even when this attribution is unwarranted.

Empirical findings provide robust support for these theoretical accounts. For instance, Gasper and Clore (1998) showed that experimentally induced negative affect increases risk appraisal, leading individuals to judge both personal and impersonal negative events as more likely and more severe compared to participants in a positive affect condition. Likewise, Scott and Cervone (2002) demonstrated that incidental negative affect can influence self-regulatory cognition by promoting the adoption of higher performance standards, even when the affective state is unrelated to the performance domain. Together, these studies suggest that affective states can systematically bias evaluative judgments and self-regulatory standards in non-clinical populations.

Overall, research in healthy samples indicates that affective dispositions exert a meaningful influence on core cognitive-evaluative processes. In turn, this affect-driven bias may contribute to self-reinforcing cycles, whereby negative affect strengthens maladaptive interpretations and beliefs, potentially maintaining and amplifying the emotional state itself.

3. Emotional reasoning and the affect-as-information mechanism in anxiety disorders

Despite the growing body of research on how affect influences cognition in non-clinical populations, far less is known about how this mechanism operates among individuals with psychological disorders, such as anxiety or mood disorders. However, several studies showed that anxious individuals tend to easily engage in emotional reasoning, leading them to draw invalid conclusions about a situation based on their subjective emotional assessment or response (Arntz et al., 1995; Gangemi et al., 2007). The mechanism of emotional reasoning has been clearly described by Beck et al. (1985) "*many anxious patients use their feelings to validate their thoughts and thus start a vicious circle: I'll be anxious when I ask for the date, so there must be something to fear*" (p. 198).

More specifically, studies investigating individuals with anxiety disorders (Arntz et al., 1995; Engelhard et al., 2001; Gangemi et al., 2007; Verwoerd et al., 2013, 2016) have consistently highlighted the critical role of emotional reasoning (ER) in this population. Notably, when comparing individuals with clinically significant symptoms to control participants (those with no or mild symptoms), group differences were particularly evident in the absence of objective threat information. In such situations, individuals with anxiety tended to interpret scenarios as more threatening than controls, indicating a greater reliance on ER. For example, Arntz et al. (1995) experiments on emotional reasoning show that adult anxious patients inferred danger based on their anxious response, whereas normal controls inferred danger primarily based on objective information. This suggests that anxious individuals struggle more with accurately distinguishing between safe and dangerous contexts (Arntz et al., 1995). Furthermore, these studies found that the severity of anxiety symptoms correlated positively with the degree of ER, and one study confirmed the temporal stability of these effects (Verwoerd et al., 2016). Finally, Berle and Moulds (2013b) proposed that anxious individuals tend to maintain a heightened sensitivity to both internal and external cues—particularly emotional states—as potential indicators of threat. This hypervigilance is well-documented in disorders such as posttraumatic stress disorder (PTSD), where individuals exhibit persistent anxiety in response to trauma-related memories and internal emotional signals (Engelhard et al., 2001). Similarly, individuals with elevated trait guilt often rely on their guilt-related emotions as diagnostic cues for assessing threat, which may facilitate the onset and persistence of obsessive–compulsive disorder (Gangemi et al., 2007).

4. Emotional reasoning and the affect-as-information mechanism in mood disorders

Although emotional reasoning (ER) has received relatively little empirical attention in the context of anxiety disorders, research into its role in depressive populations is even more limited. So far only 2 studies, both by Berle and Moulds (2013a, b), investigated the relationship between emotional reasoning or affect-as-information and depression using clinical samples (for a systematic review, see Paredes-Mealla et al., 2023). In their experiments, the scholars (Berle & Moulds, 2013a, b) wanted to examine whether people who were experiencing a current major depressive episode engaged in emotional reasoning to a greater extent than those who were not depressed. Furthermore, they tried to verify whether previously depressed individuals engaged in higher levels of emotional reasoning when compared with a group of never-depressed individuals. According to the authors, if important levels of emotional reasoning persist between different episodes, it may indicate that this mechanism can serve as a risk factor or marker for individuals who later develop depression, that it is a more enduring trait-like process, or that the tendency persists as some sort of "cognitive scar" (Lewinsohn et al., 1981). Contrary to their expectations, Berle and Moulds (2013a, b) found that individuals with depression exhibited only marginally higher, non-significant ER scores compared to non-depressed controls. The two scholars offered several potential explanations for these findings. One possibility is depressed individuals vary greatly in the degree to which they engage in emotional reasoning, which may be related to the level of severity of their symptoms. Individuals with depression may display a reduced engagement with present-moment stimuli. Their cognitive focus often remains anchored in past experiences of loss, failure, or personal inadequacy (Berle & Moulds, 2013), potentially diminishing the salience of current emotional states in shaping their interpretations.

According to Paredes-Mealla et al. (2023), while such a framework remains theoretically compelling, further research employing other psychometrically valid measures is essential to clarify whether ER plays a meaningful role in depression and, consequently, whether it should be targeted in psychotherapeutic interventions for depressive disorders.

For this reason, a thorough investigation into the role of the ER or affect as information mechanism in depression, as found by Scott and Cervone (2002) in their study with healthy individuals, would be useful. In Experiment 1, Scott and Cervone (2002) examined the affect-as-information hypothesis by inducing either negative or neutral affect in a sample of undergraduate students. In the negative affect condition, participants listened to a scenario that described their best friend dying of cancer. In the neutral affect condition, participants were asked to visualize their room at home. After the affect induction, participants completed a questionnaire presented as a "survey", which included the main dependent measures: minimal performance standards for academic and social activities (i.e., the lowest level of performance they would find acceptable) and evaluative judgments concerning how satisfied they would feel with their performance outcomes. The results

indicated that participants in the negative affect condition adopted higher minimal performance standards and reported lower satisfaction with their performance, compared to those in the neutral condition. Having demonstrated that negative emotions can lead to higher performance standards and lower satisfaction with one's own performance in non-clinical groups, the authors argued that their results could have important implications for understanding and treating depression. They suggested that depressed individuals may use their negative feelings (e.g. sadness) to validate their thoughts (e.g. "I'm a failure"), creating a vicious cycle that perpetuates their depression. A depressed mood could increase performance standards (e.g. needing to complete every task flawlessly at work), making it less likely that they will be met and increasing dissatisfaction, thereby maintaining the cycle. In other words, this mechanism escalates performance expectations, which become increasingly difficult to fulfil, thereby exacerbating mood disturbance and reinforcing the cycle. While these hypotheses are compelling from a clinical perspective - those with experience of depressed patients will find this very reasonable - they have yet to be empirically validated in clinical or subclinical depressed populations.

To address this issue, we conducted two experiments. Study 1 was inspired by the paradigm developed by Scott and Cervone (2002) and extended to a subclinical sample. Participants were categorized as either High-Depression (HD) or Low-Depression (LD) based on their scores on the Italian version of the Beck Depression Inventory–II (BDI-II; Ghisi et al., 2006). Study 2 was also inspired by Scott and Cervone's (2002) experimental approach, but included three affect-induction conditions (sadness, anxiety, and neutral) in order to determine whether the expected effects were specifically attributable to current sadness rather than to negative affect more broadly. In line with Scott and Cervone's framework, we hypothesized that individuals reporting higher levels of current sadness (i.e., the HD group), as well as those experiencing induced sadness, would be more likely to use their affective state as informational input when evaluating their performance. Consequently, they were expected to adopt higher performance standards and report lower satisfaction with their outcomes.

5. Study 1

The aim of our study was to investigate whether negative emotions, such as current sadness, influence performance standards and satisfaction levels in individuals with subclinical depression.

To this end, participants were categorized into high- and low-depression groups based on a standardized depression measure (i.e. the BDI-II, Ghisi et al., 2006). The experimental task was a shortened version of the Symbol Search subtest from the WAIS-IV battery (Wechsler, 2008; Orsini & Pezzuti, 2013; see below). Once participants had completed the task, they received their performance score and were asked to estimate their expected result and rate their level of dissatisfaction.

We hypothesized that individuals feeling high level of current sadness (i.e. those in the high-depression group) would set higher performance standards for themselves and evaluate their actions as more unsatisfactory than individuals in the low-depression group.

5.1. Method

5.1.1. Participants

An a priori power analysis was conducted using G*Power 3.1 for a two tails independent-samples *t*-test, consistent with the study's directional hypothesis. Following Cohen's (1988) conventions, statistical power was set at 0.80, with $\alpha = 0.05$ and an anticipated medium effect size ($d = 0.50$). The analysis indicated that a minimum of 64 participants per group (total $N = 128$) would be required to detect the expected effect.

Participants were recruited through university courses and social media platforms (e.g., email and WhatsApp). The final sample consisted of 328 individuals (236 females, 92 males) aged between 18 and 70 years ($M = 35.00$, $SD = 11.30$). All participants provided informed consent and took part voluntarily; undergraduate students received one course credit in exchange for participation. The study was approved by the institutional Bioethics Committee.

5.1.2. Materials and procedures

Participants were provided with a direct link to the experimental materials hosted on Google Forms. Upon accessing the link, they were first presented with an informed consent form; only those who provided explicit consent were allowed to continue. The study was conducted remotely, and all materials (including instructions, stimuli, and response options) were implemented within the Google Forms interface to ensure standardized administration.

After providing consent, participants received detailed instructions describing the overall structure of the study, the nature of the tasks, and the required response format.

The experimental task was a reduced computerized version of the *Symbol Search* subtest from the WAIS–IV (Wechsler, 2008; Orsini & Pezzuti, 2013). It consisted of 40 nonverbal visual items presented sequentially. As illustrated in Fig. 1, each item displayed two target symbols (left gray panel) and a set of five search symbols (right panel) on a single row. All symbols were composed of abstract lines and geometric shapes. Participants were required to determine whether either of the two target symbols appeared among the five search symbols. If one target symbol was present, participants selected the corresponding response option; if neither target appeared, they selected the "NO" option. Only one target symbol could be present in each item (see Fig. 1). Before the experimental trials, two practice items were administered to familiarize participants with the procedure. Participants were instructed to complete as many items as possible within a 2.5-min time limit.

The total score corresponded to the number of correct responses and ranged from 0 to 40.

After completing the task, participants were shown their total score and were asked to report their expected performance standard by indicating the score they had anticipated (0–40). They were also asked to rate their satisfaction with their actual performance using a 5-point Likert scale.

Participants then completed the Italian version of the Beck Depression Inventory–II (BDI-II; Ghisi et al., 2006), a 21-item self-report

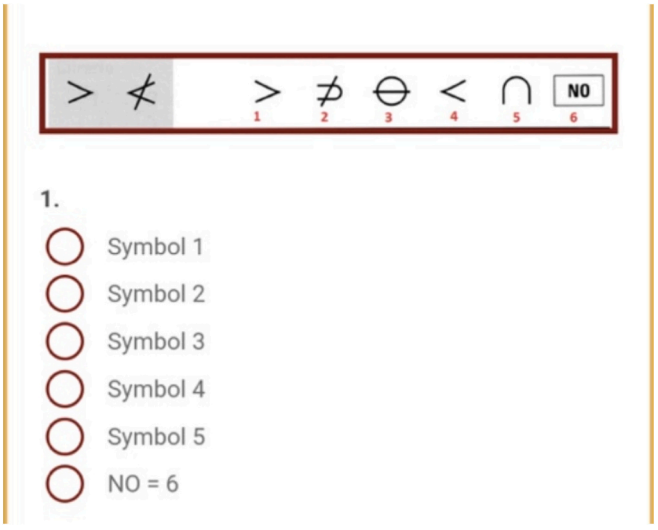

Fig. 1. Structure of a single trial in the symbol search task. *Note.* Each trial consisted of two target stimuli (left panel) and five search stimuli (right panel). Participants performed a forced-choice detection task by indicating the presence of one target symbol or selecting "NO" if neither target was present. The task comprised 40 trials with identical structure.

measure assessing the severity of depressive symptoms according to DSM-IV criteria. Each item is rated on a 4-point scale (0–3), yielding a total score ranging from 0 to 63, with higher scores indicating greater symptom severity. Standard cutoffs classify scores as minimal (0−13), mild (14–19), moderate (20–28), and severe (29–63). The Italian BDI-II shows good internal consistency (Cronbach's α = 0.87) and adequate test–retest reliability (r = 0.76), and supports a two-factor structure (cognitive–affective and somatic–performance). The BDI-II is suitable for individuals aged 13 years and older and requires approximately 5–10 min to complete.

Responses were automatically recorded by Google Forms and exported to Excel files for storage and analysis. Data collection took place in 2025, and no identifying information was collected.

5.2. Results

The first dependent variable was computed as the discrepancy between actual and expected task performance (actual score minus expected score). Positive values indicated better-than-expected performance, whereas negative values indicated worse-than-expected performance. The second dependent variable was participants' satisfaction with their task performance, assessed on a 5-point Likert scale.

It was hypothesized that participants in the high-depression (HD) group would use their current negative affect as informational input when evaluating their performance. Consequently, they were expected to show a larger discrepancy between expected and actual scores and lower satisfaction compared to participants in the low-depression (LD) group.

5.2.1. Results for the high HD and the LD groups

Group differences were examined using independent-samples t-tests. In the full sample, the mean BDI-II score was M = 12.96 (SD = 10.03). Participants scoring below the 25th percentile on the BDI-II were classified as the low-depression (LD) group (M = 2.6, SD = 1.9, range = 0–5), whereas participants scoring above the 75th percentile were classified as the high-depression (HD) group (M = 25.8, SD = 5.8, range = 18–40). This resulted in 87 participants in the LD group and 83 participants in the HD group.

The two groups did not differ significantly in sex distribution, $\chi^2(2, 170) = 5.07$, $p = .08$, Cramér's V = 0.17, age, $t(168) = 1.83$, $p = .069$, $d = 0.28$, 95% CI [−0.02, 0.58], or actual task performance, $t(168) = 0.116$, $p = .91$, $d = 0.02$, 95% CI [−0.28, 0.32].

5.2.2. Difference between the actual and expected task scores

Contrary to our hypothesis, participants in the HD group showed a significantly smaller discrepancy between expected and actual task scores compared to participants in the LD group, $t(168) = 2.41$, $p = .017$, $d = 0.37$, 95% CI [0.07, 0.67]. As shown in Fig. 2, the discrepancy score was lower in the HD group than in the LD group.

5.2.3. Level of satisfaction

Contrary to expectations, no significant differences emerged between groups in satisfaction with task performance, $t(168) = 1.1$, $p = .28$, $d = 0.17$, 95% CI [−0.13, 0.47]. As illustrated in Fig. 2, HD and LD participants reported comparable levels of satisfaction with their performance, despite the group differences observed in performance standards (Table 1).

5.3. Conclusions

Surprisingly, our results suggest that individuals feeling current sadness (i.e. those in the HD group) set themselves lower performance standards than the LD group and evaluate their actions as being as satisfactory as those in the LD group. These results challenge Scott and Cervone's hypotheses that people experiencing chronic sadness (i.e. depressed individuals) use their emotional state as salient information when evaluating their performance, raising their standards and become more dissatisfied with their performance. In line with Berle and Moulds (2013), one potential explanation for these findings is that the degree to which depressed individuals engage in emotional reasoning varies greatly, and this may be related to the amount of the current emotion experienced and the severity of their symptoms. Indeed, our HD group shows a mean BDI score of 26, which corresponds to current depression symptoms in the moderate range (range cut-off: 20–28) (Beck et al., 1985, Ghisi et al., 2006). It is therefore plausible to hypothesize that they may display reduced engagement with present-moment stimuli. Thus, the difference between our results and those of Scott and Cervone (2002) may not lie in the emotion experienced per se, but rather in its

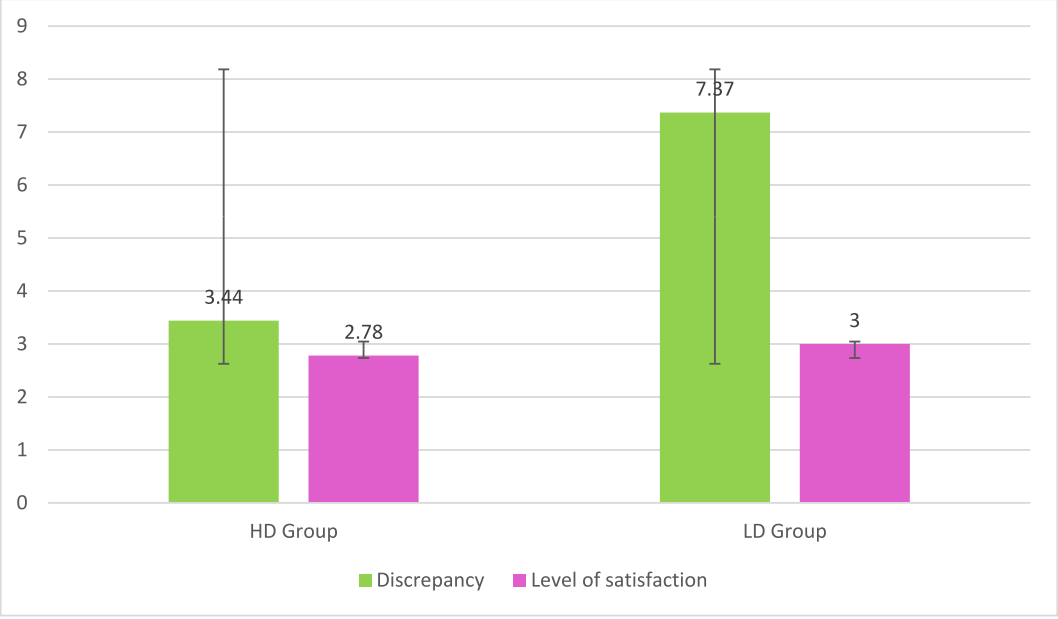

Fig. 2. Group differences in performance-standard discrepancy and satisfaction (Study 1). *Note.* Bars represent mean scores for the high-depression (HD) and low-depression (LD) groups. Discrepancy was calculated as actual minus expected task performance. Error bars denote ±1 SD.

abnormal intensity, as typically observed in subjects with current depressive symptoms. According to the Hyper Emotion Theory (Johnson-Laird et al., 2006), the difference between a healthy individual and someone affected by an anxiety or mood disorder is not the quality of the emotion experienced, but rather its intensity. In patients, this intensity is typically exaggerated and abnormal.

Another possible explanation is that the mechanism they proposed was not specifically driven by sadness, the core emotion of depressive disorders, but by any negative emotion, such as anxiety. To elicit negative emotions, Scott and Cervone (2002) asked participants in the negative emotion induction group to listen to an audio recording informing them that their best friend had died of cancer. They assumed that such a story would evoke a sense of loss and, consequently, sadness. However, they did not use any instruments to verify whether the induction was effective or to determine which negative emotion was elicited. Listening to the story could have induced anxiety, fear of potential loss, guilt or anger, perhaps because the loss was perceived as unfair.

6. Study 2

In our earlier study, we found that individuals with current depression symptoms (HD group) exhibited non-significant ER scores compared to LD group. Contrary to our expectations, they showed lower performance standards than the LD group and evaluate their performance as being as satisfactory as those in the LD group.

To clarify the findings of the previous experiment and determine whether they were driven by the severity of current depressive symptoms (see Berle & Moulds, 2013) or by a current negative emotion other than sadness, we conducted a second experiment. In this study, three groups of participants from the general population were randomly assigned to one of three emotion-induction conditions: sadness, anxiety, or a neutral state. Anxiety was included as a contrasting negative emotion because it represents a qualitatively distinct form of negative affect compared to sadness.

As in the previous experiment, after completing the cognitive task (i.e., the *Symbol Search* subtest from the WAIS–IV), participants were provided with their performance score and were asked to report their expected performance (i.e., their performance standard) and to rate their satisfaction with their actual outcome.

To verify the effectiveness of the emotion induction procedures, participants completed the Positive and Negative Affect Schedule (PANAS; Terracciano et al., 2003) along with a manipulation check questionnaire (see below). These measures were included to ensure that the sadness and anxiety conditions successfully elicited the intended negative emotional states.

Consistent with Scott and Cervone (2002), we hypothesized that participants in the sadness condition would use their emotional state as informational input when evaluating their performance. Specifically, they were expected to adopt higher performance standards, show a greater discrepancy between expected and actual scores, and report lower satisfaction compared to participants in the anxiety and neutral conditions.

6.1. Method

6.1.1. Participants

An a priori power analysis was conducted using G*Power 3.1 to determine the required sample size for a one-way fixed-effects ANOVA with three groups. A medium effect size was assumed ($f = 0.25$), with $\alpha = 0.05$ and desired statistical power of 0.80, under the assumption of equal group sizes. The analysis indicated that a minimum total sample of 159 participants (approximately 53 per group) would be required.

Participants were recruited through university courses and social media platforms (e.g., e-mail and WhatsApp). The final sample consisted of 407 individuals aged between 18 and 65 years ($M = 32.00$, $SD = 8.5$), including 294 females and 113 males. All participants provided informed consent and took part voluntarily; students received one course credit in exchange for participation.

Participants were randomly assigned to one of three affect-induction conditions: sadness ($n = 139$), anxiety ($n = 125$), or neutral ($n = 143$).

Preliminary analyses indicated that the three groups did not differ significantly in sex distribution, $\chi^2(2, N = 407) = 4.19$, $p = .12$, Cramér's $V = 0.10$, 95% CI [0.00, 0.20], age, $F(2, 404) = 2.18$, $p = .12$, $\eta^2 = 0.01$, 95% CI [0.00, 0.03], or actual task performance, $F(2, 404) = 2.80$, $p = .06$, $\eta^2 = 0.01$, 95% CI [0.00, 0.04].

The study was approved by the institutional Bioethics Committee.

6.1.2. Materials and procedures

The study was administered remotely using Google Forms. Participants received a direct link to the experimental materials and were first presented with an informed consent form. Only those who provided explicit consent were allowed to proceed. All instructions, stimuli, and response options were implemented within the Google Forms interface to ensure standardized administration.

The procedure is illustrated in Fig. 3. At the beginning of the session, participants completed the Positive and Negative Affect Schedule (PANAS; see below) and Part 1 of the manipulation check questionnaire to assess baseline affect. They then received written instructions for the affect induction corresponding to their assigned condition (sadness, anxiety, or neutral). Immediately after the induction, participants completed the PANAS again along with Part 2 of the manipulation check questionnaire to verify the effectiveness of the manipulation.

6.1.2.1. Baseline affect. Before the writing task, participants completed Part 1 of the manipulation check questionnaire, which included six visual analogue scales (VASs): three assessing the target emotions (sadness, anxiety, and neutrality) and three filler emotions (disgust, anger, and guilt). Participants rated the intensity of each emotion on a 0–10 scale (0 = not at all; 10 = completely).

Baseline affect was also assessed using the Italian version of the PANAS (Terracciano et al., 2003), which consists of 20 emotion adjectives rated on a 5-point scale (1 = very slightly or not at all; 5 = extremely). Items were grouped into positive and negative affect scales, which demonstrated acceptable internal consistency ($\alpha = 0.73$ and $\alpha = 0.88$, respectively).

This instrument was used in order to quantify the sadness and the anxiety induction effect through the "sadness factor" "anxiety factor" scores.

6.1.2.2. Emotion induction. Affective states were induced using an autobiographical recall writing task (cf. Schwarz & Clore, 1983). Participants were instructed to write about a personally relevant event associated with sadness, anxiety, or a neutral experience, depending on condition. They were asked to describe the event as vividly as possible, including details of their thoughts and feelings at the time. Participants were given 15 min to complete the task. Importantly, the induced affect was incidental and unrelated to the subsequent cognitive task.

6.1.2.3. Manipulation check. Following the writing task, participants completed Part 2 of the manipulation check questionnaire. They rated the intensity of sadness, anxiety, neutrality, disgust, anger, and guilt on a 0–10 scale (0 = not at all; 10 = completely).

Participants also completed the PANAS again to assess affective changes. A principal component analysis was conducted on selected PANAS items. Two anxiety-related items ("afraid" and "scared") were combined into an anxiety factor (eigenvalue = 4.30, 54% of variance explained), and two sadness-related items ("upset" and "distressed") were combined into a sadness factor (eigenvalue = 1.26, 16% of variance explained).

6.1.2.4. Cognitive task and dependent measures. Participants then

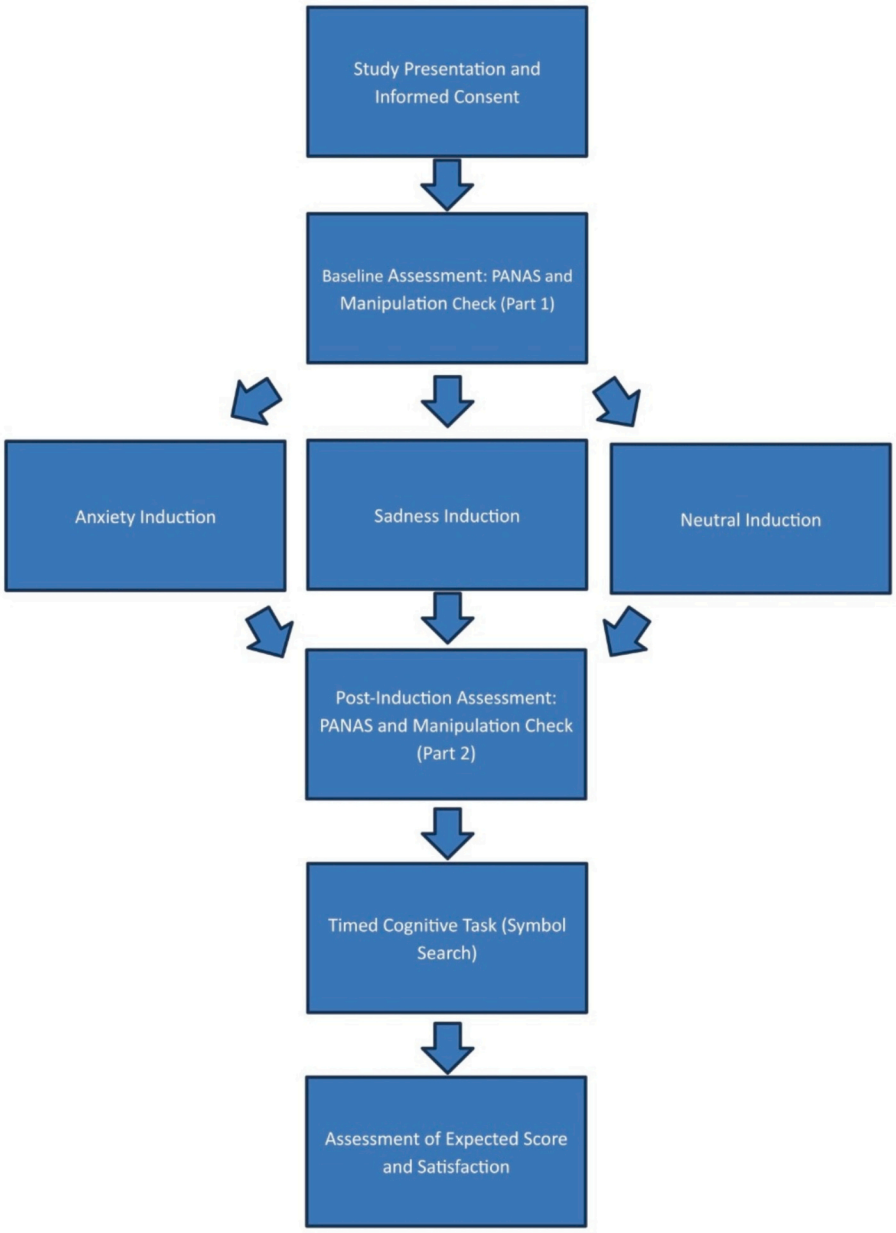

Fig. 3. Procedure of Study 2. *Note.* Flow diagram illustrating the sequence of experimental phases, including baseline affect assessment, emotion induction (sadness, anxiety, or neutral), post-induction manipulation checks, cognitive task administration, and assessment of performance standards and satisfaction.

completed the same shortened version of the Symbol Search subtest from the WAIS–IV used in Study 1. Performance was scored as the number of correct responses (range: 0–40).

After completing the task, participants were shown their total score and asked to report their performance standard (i.e., the minimum score they would consider satisfactory; 0–40). They also rated their satisfaction with their actual performance on a 5-point Likert scale, with lower scores indicating greater dissatisfaction.

Responses were automatically recorded via Google Forms and exported to Excel for analysis. Data collection took place in 2025, and no identifying information was collected.

6.2. Results

6.2.1. Manipulation check: measures of affect induction

Fig. 4 and Fig. 5 show the mean affect ratings on scales of sadness and anxiety for participants in all three affect induction conditions both before and after the affect induction procedure. Each measure was subjected to a 2 × 3 ANOVA, comparing Time (before vs after) as a within group factor, and Affect Induction group (sadness, anxiety, or neutral) as a between group factor (Table 2).

For sadness, a significant Time X Emotion Induction group interaction was found, $F\,(2, 404) = 81.77$, $p < .001$, $\eta^2 = 0.28$, CI [0.21–0.36] CI [0.21–0.36]. The nature of the interaction was analysed by studying what groups displayed a significant pre-to-post increase in sadness. As suggested by Fig. 4, the increase in the sadness induction group was significant ($t\,(138) = 13.77$, $p < .001$, $d = 1.5$, CI [1.26, 1.74]), but no significant effect was found in the anxiety induction group ($t\,(124) = 1.13$, $p = .26$, $d = 0.10$, CI [−0.08, 0.28]), while a significant pre-to-post decrease in sadness was found in the neutral group ($t\,(142) = 3.48$, $p < .001$, $d = 0.29$, CI [0.12, 0.46]).

Also, for the anxiety measure there was a significant Time X Emotion Induction group interaction, $F\,(2, 404) = 21.84$, $p = .001$, $\eta^2 = 0.10$, CI [0.06, 0.15]. The nature of the interaction was analysed by studying

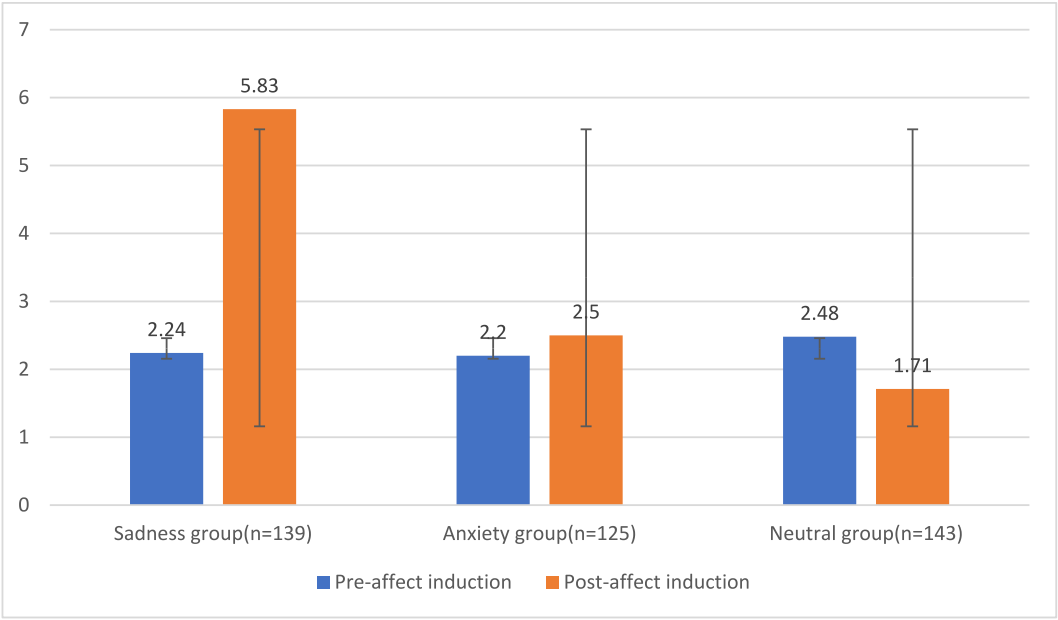

Fig. 4. Pre- and post-induction sadness scores across emotion conditions in Study 2. *Note.* Mean sadness ratings (0–10 scale) are shown for the sadness, anxiety, and neutral groups before and after the induction procedure. Error bars represent ±1 SD.

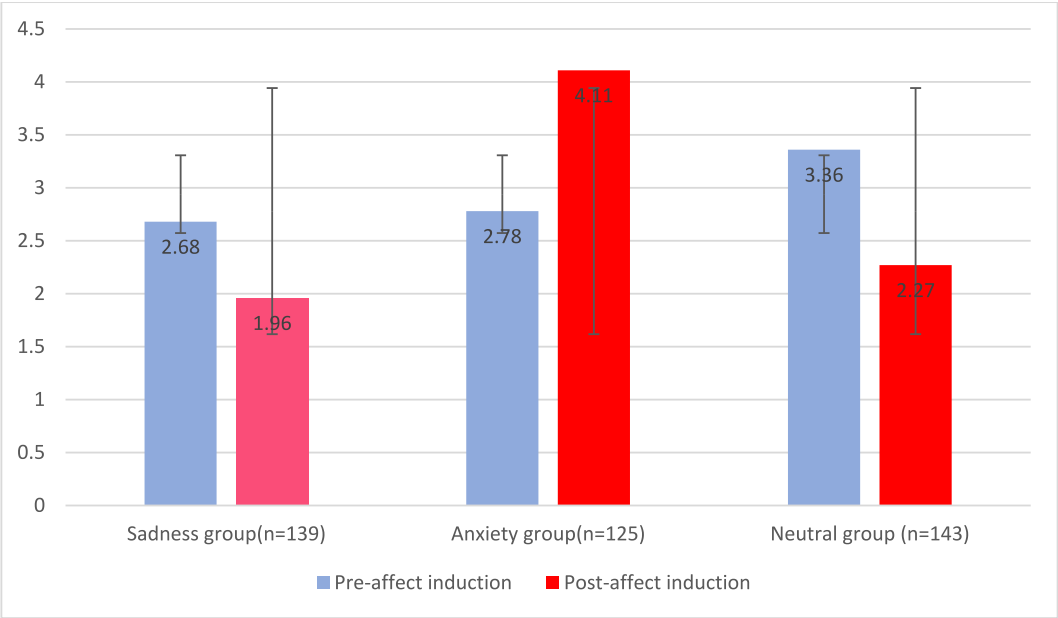

Fig. 5. Pre- and post-induction anxiety scores across emotion conditions in Study 2. *Note.* Mean anxiety scores are shown for the sadness, anxiety, and neutral groups before and after the induction procedure. Error bars represent ±1 SD.

what groups displayed a significant pre-to-post increase in anxiety. A significant pre-to-post increase in anxiety was found in the anxiety induction group (t (124) = 4.53, $p < .001$, $d = 0.41$, CI [0.22, 0.59]). A significant pre-to-post decrease was found both in the sadness induction group (t (138) = 3.15, $p = .002$, $d = 0.27$, IC 95% [0.10, 0.44]) and in the neutral group (t (142) = 3.67, $p < .001$, $d = 0.31$, CI [0.14, 0.48]) (see Fig. 5).

Moreover, in the case of the sadness-factor measure from PANAS, a significant Time X Emotion Induction group interaction was found, F (2, 404) = 37.11, $p < .001$, $\eta^2 = 0.15$, CI [0.10–0.21]. A significant pre-to-post increase in sadness measure was displayed by the sadness induction group (pre: M = 1.95; SD = 0.89; post: M = 2.58; SD = 99 (t (138) = 8.62, $p < .001$, $d = 0.73$, CI [0.55, 0.91]), but no significant effects were found neither in the anxiety induction group (pre: M = 1.76; SD = 0.69; post: M = 1.7; SD = 85 (t (124) = 1.04, $p = .30$, $d = 0.09$, CI [−0.08, 0.26]), nor in the neutral group (pre M = 1.96; SD = 88; post: M = 1.94; SD = 92 (t (142) = 0.41, $p = .68$, $d = 0.03$,CI [−0.13, 0.19]).

Finally, as regards the anxiety-factor measure from PANAS, a significant Time X Emotion Induction group interaction was found, F (2, 404) = 32.65, $p < .001$, $\eta^2 = 0.13$, CI [0.07–0.20]). A significant pre-to-post increase in anxiety measure was displayed by the anxiety induction group (pre: M = 1.79; SD = 0.98; post: M = 2.4; SD = 1.22) (t (124) = 6.25, $p < .001$, $d = 0.56$, CI [0.37, 0.75]), but no significant effects were found in the sadness induction group (pre: M = 1.94; SD = 0.93; post: M = 1.8; SD = 1.07) (t (138) = 1.96, $p = .52$, $d = 0.17$, CI[−0.00, 0.34]), while in the neutral group a significant pre-to-post decrease was found

(pre M = 2; SD = 0.95; post: M = 1.8; SD = 0.99) (t (142) = 3.5, $p < .001$, $d = 0.29$, CI[0.12, 0.46]).

Therefore, it seems that the emotion experimental manipulation was successful. The sadness induction did induce an increase in this emotion, but the other manipulations did not. Likewise, the anxiety induction group was characterized by increases in anxiety, while no anxiety increase was observed in the other conditions.

6.2.2. Cognitive task

As in the earlier study, the first dependent variable was computed as the discrepancy between actual and expected task performance (actual score minus expected score). Positive values indicated better-than-expected performance, and negative values indicated worse-than-expected performance.

The second dependent variable was participants' satisfaction with their task performance, assessed on a 5-point Likert scale.

It was hypothesized that participants in the sadness group would show a greater discrepancy between actual and expected scores, as well as a lower level of satisfaction, than participants in the anxiety and neutral groups.

6.2.3. Difference between the actual and expected task scores

A significant effect of Affect Induction emerged with regard to *discrepancy* between *expected and actual score* ($F_{(2,404)} = 3.4$, $p = .03$, $\eta^2 = 0.01$, CI [0.0–0.04]) (see Fig. 6 and Table 3), but the result did not go in the expected direction. Individuals in the sadness induction group showed a smaller *discrepancy* than both participants in the anxiety condition (t (262) = 2.3, $p = .22$, $d = 0.47$, CI [0.23, 0.71]) and in the neutral induction group (t (280) = 2.5, $p = .01$, $d = 0.53$,CI [0.29–0.77]).

6.2.4. Level of satisfaction

Contrary again to our expectations, no differences were found in the satisfaction level with performance scores for the task between the sadness group and the other two groups ($F_{(2,404)} = 2.16$, $p = .12$, $\eta^2 = 0.01$, CI [0.00–0.04]). As shown in Fig. 6, the sadness induction group reported the same level of satisfaction with their performance on the task, as the anxiety group (t (262) = 0.93, $p = .35$, $d = 0.12$,CI [−0.13, 0.36]), and the neutral group (t (280) = 1.14, $p = .25$, $d = 0.14$, CI [−0.10, 0.37]) (Table 3).

6.3. Conclusions

As in the earlier study, and contrary to expectations, our findings suggest that individuals experiencing sadness (i.e. those in the sadness induction group) set lower performance standards than participants in the two control groups (anxiety and neutral induction). Nevertheless, they rated their performance as being as equally satisfactory to that of the control participants. These results are consistent with those of our previous experiment, suggesting that the observed pattern was not likely to be due to the severity of current depressive symptoms. This leaves open the possibility that the effects reported in Scott and Cervone's study were due to the induction of a negative emotion other than sadness, such as anxiety or general distress. These findings challenge thus the original formulation of the sadness-as-information hypothesis, which suggests that individuals experiencing sadness use their emotional state as salient input when evaluating their performance, thereby increasing their standards and becoming more dissatisfied. However, our data suggest the opposite pattern, raising the possibility that Scott and Cervone's mechanism may not apply specifically to sadness, but rather to other negative emotions.

7. General discussion

Scott and Cervone (2002) proposed a clinically relevant Emotional Reasoning (ER) or affect-as-information mechanism, whereby negative emotions increase personal performance standards while decreasing satisfaction. This fosters a self-perpetuating cycle of depressive mood. Although this model was derived from studies of healthy individuals, it provides a compelling framework for understanding how negative emotions may contribute to depression. However, its applicability to clinical or subclinical populations has yet to be empirically established, partly because the researchers did not specifically investigate sadness.

Our research aimed to empirically extend these findings to individuals experiencing current sadness, as populations with current depressed symptoms. To this aim, Study 1 examined participants with varying levels of current depression, high and low depression groups based on BDI-II scores, while Study 2 involved the experimental induction of specific emotional states (sadness, anxiety or neutrality), to isolate the effects of sadness. In both studies, we expected participants experiencing either current or induced sadness to use their emotional state as information when evaluating their performance, thereby raising their standards and decreasing their satisfaction. Nevertheless, our findings challenge Scott and Cervone's hypotheses, showing that participants in the high depression group (Study 1) and sad group (Study 2) reported lower performance standards than their respective control groups (low depression group in Study 1 and anxious and neutral groups in Study 2) and evaluate their performance as being as satisfactory as

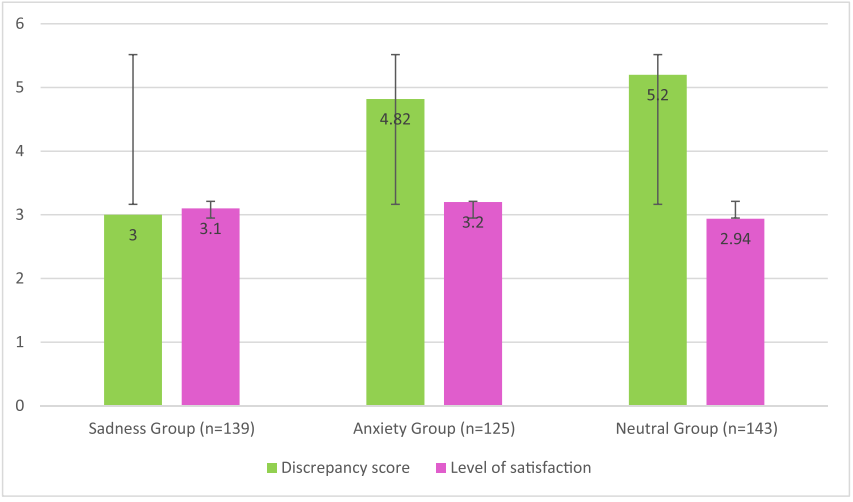

Fig. 6. Mean performance-standard discrepancy and satisfaction across emotion conditions (Study 2). *Note.* Bars represent mean discrepancy scores (actual minus expected performance) and satisfaction ratings for the sadness, anxiety, and neutral groups. Error bars denote ±1 SD.

those in the control groups. Although this result is in line with several studies on affect as information in depression which have so far failed to provide consistent support for its role in this symptomatology, it raises a fundamental question: how can we explain the pattern of results observed? We believe there are at least three plausible interpretations, each of which sheds light on a different aspect of the underlying mechanisms.

Firstly, the absence of ER effects may reflect a fundamental characteristic of sadness itself—namely, that it does not trigger ER in the same way as other negative emotions, such as anxiety. This may be especially true in cases of current or clinical sadness, as observed in depressive states, where emotional reactivity tends to be blunted and cognitive disengagement from external stimuli often prevails. In this sense, the findings from Study 1, which involved individuals with moderate depressive symptoms, may not indicate a failure of ER per se, but rather a more general reduction in the salience or accessibility of emotional cues. For example, Berle and Moulds (2013) found that individuals with depression exhibited only slightly higher, and statistically non-significant, ER scores compared to non-depressed individuals.

But why does the ER—or sadness-as-information—mechanism appear to have little or no impact on depressive symptomatology? One possibility, consistent with Berle and Moulds (2013), is that there is considerable variability among depressed individuals in the extent to which they engage in ER. Such variability may depend on both the intensity of the emotional experience and the severity of depressive symptoms. Accordingly, the discrepancy between our findings and those of Scott and Cervone may not be due to the specific emotion involved, but rather to its exaggerated intensity—a feature commonly observed in individuals with psychological disorders. According to Hyper-Emotion Theory (Johnson-Laird et al., 2006), the key difference between psychologically healthy individuals and those with mood or anxiety disorders lies not in the quality of the emotion experienced, but in its abnormal intensity.

It is also plausible that individuals with depression tend to be disengaged from present-moment experiences, as their attention and cognition often remain focused on past losses, failures, and enduring inadequacies (Berle & Moulds, 2013). If these individuals have reduced emotional awareness, they may be less likely to use affective states as information when interpreting ambiguous situations. Consequently, they may be less susceptible to emotional reasoning biases. Indeed, participants in our high-depression group had a mean BDI score of 26, corresponding to the clinical threshold for moderate depression (Beck et al., 1985, Ghisi et al., 2006), suggesting a potential reduction in present-focused engagement.

Secondly, perhaps we failed to find evidence of ER or sadness as information driven by sadness because Scott and Cervone's study never actually examined sadness. While their paradigm aimed to induce a negative emotional state, there is reason to question whether the emotion elicited was actually sadness. It was this ambiguity that led us to design Study 2, in which sadness was induced directly and specifically in a controlled manner. Therefore, the mechanism proposed by Scott and Cervone may not have been driven by sadness per se—the core affective component of depression—but rather by general negative affect, such as anxiety or anger. In their study, negative affect was induced by having participants listen to an audio recording describing the death of their best friend from cancer. The authors assumed this would evoke sadness through a sense of loss. However, no instruments were employed to assess whether the induction was successful, or to identify which specific negative emotion was elicited. It is conceivable that the story may have instead induced anxiety, anticipatory fear of potential loss or anger, perhaps because the loss was perceived as unfair.

The crucial point is that, even under more controlled conditions such as those in our studies, we did not observe the predicted ER effects. This prompted us to re-evaluate the role of sadness in this process, resulting in a third and perhaps most intriguing interpretation. The results of both our studies point in the same direction: sadness may influence ER in the opposite way to that originally proposed by Scott and Cervone. Rather than increasing performance standards and reducing satisfaction, sadness — particularly when sustained or pathological — might cause individuals to lower their standards, bringing them closer to their actual level. Indeed, in both studies, the experimental groups (HD/sad) showed a smaller discrepancy between their actual and expected performance than the control groups (LD/anxious-neutral), indicating a more realistic evaluation. However, this shift in expectations does not appear to decrease satisfaction. In fact, they evaluated their actions as being as satisfactory as those in the control groups, probably because they were in line with their realistic expectations.

In other words, based on our findings, we propose that the sadness-as-information mechanism functions by lowering evaluative standards and expectations, thus making them more realistic. It is precisely this alignment between expectations and actual performance that may account for the moderate level of satisfaction reported by participants. Unlike anxiety, which often leads to inflated standards and dissatisfaction, sadness may ground evaluations in a framework that is more congruent with reality.

This mechanism appears to align with the well-established theory of depressive realism. (Alloy & Abramson, 1979), whereby individuals with depression are said to have a more accurate and realistic perception of themselves and the world, particularly in situations involving personal control or competence. According to this theory, non-depressed individuals often display a positivity bias, whereas depressed individuals perceive their environment — and their own abilities — more in line with objective outcomes. Importantly, while depressive realism is usually explained in terms of self-related cognitive beliefs, our data suggest that sadness may cause and reinforce this process. Through the lens of the affect-as-information framework (Schwarz & Clore, 1983), sadness may serve as a cue that confirms and stabilizes pre-existing negative self-beliefs. Rather than merely influencing momentary evaluations, sadness could thus act as an internal signal that validates dysfunctional self-schemas, thereby contributing to the persistence and maintenance of depressive symptoms. Sadness as information may therefore help to explain why depressive realism is observed only among individuals with depression, and what accounts for the correspondence between their expectations and objective outcomes.

However, we are aware that our study has several limitations.

For example, it only provides preliminary findings regarding *our* sadness as information mechanism in healthy individuals and those experiencing subclinical levels of current depression. Yet, we think it also offers interesting points for reflection, and we hope it will prompt further studies. For instance, future research should further replicate our findings using different types of tasks that are more realistic, ecological, or relevant to individuals' lives. The fact that people experiencing temporary sadness tend to use this emotion to evaluate their performance, even when the task is not important, suggests that this may be a robust phenomenon. One might expect the relationship to be stronger with tasks related to family, work or social life, for example. Furthermore, having demonstrated that the emotion of sadness can lead to lower performance standards and adequate satisfaction with one's performance in non-clinical and subclinical groups, we argue that our results could have significant implications for the understanding and treatment of depression.

However, future studies with clinical populations of people with depression would be required to investigate whether the sadness-as-information process, as hypothesized here, specifically contributes to the vulnerability to and maintenance of depressive disorder and whether this process influences only this disorder or other psychological conditions such as anxiety disorders. The observation that sadness influences performance evaluations in a non-clinical sample indicates that this may be a relatively strong effect. It is therefore reasonable to hypothesize that the association could be even more pronounced in clinical populations, who may exhibit heightened sensitivity to sadness.

Moreover, the sample showed a marked gender imbalance, with a

predominance of female participants. This asymmetry has to be considered when interpreting the generalizability of our results.

Finally, future research should examine the sadness-as-information mechanism further, particularly in relation to its potential causal link with depressive realism, as hypothesized in the present study.

Due to the current lack of empirical research in this area (see Paredes-Mealla et al., 2023) and the methodological limitations of our study, further investigation is necessary. Studies that more directly explore the relationship between the sadness-as-information process, performance standard evaluation, satisfaction with one's own actions and the depressive realism hypothesis would be particularly valuable. Such research could contribute significantly to theoretical models of affect and cognition, as well as to clinical understanding and intervention.

CRediT authorship contribution statement

Amelia Gangemi: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Formal analysis, Conceptualization. **Giulia Bongiovanni:** Writing – review & editing, Writing – original draft, Resources, Data curation, Conceptualization. **Paolo Spina:** Writing – review & editing, Writing – original draft, Resources, Data curation, Conceptualization. **Febronia Riggio:** Writing – review & editing, Formal analysis. **Marco Saettoni:** Writing – review & editing, Writing – original draft, Resources, Data curation, Conceptualization. **Chiara Rizzotto:** Writing – review & editing, Writing – original draft, Data curation. **Francesco Mancini:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Conceptualization. **Andrea Gragnani:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Conceptualization.

Declaration of competing interest

All authors have approved the manuscript and confirm that it has not been published elsewhere nor is under consideration by any other journal. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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