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Logical decisions after a psychosocial stressor: The late phase of acute stress reduces loss aversion

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ABSTRACT

Loss aversion, the principle that losses have a greater impact on decision-making than gains, can be modulated by stress. Most findings reported that stress reduces loss aversion, in line with the alignment hypothesis. Yet, decision-making was always assessed at the early stages of the stress response. Instead, the latter phase of the stress response enhances the salience-network and then, it could amplify the salience of losses, thereby increasing loss aversion. To our knowledge, it has never been studied how the latter stress response influences loss aversion and our aim is to fill this gap. 92 participants were divided into experimental and control group. The first one was exposed to the Trier Social Stress Test, and controls viewed a match-length distractor video. Both groups performed a mixed gamble task to measure loss aversion through a Bayesian-computational model. During and after the stressor, experimental group exhibited signs of both physiological and psychological stress which indicated that stress induction was effective. However, rather than increasing, loss aversion of stressed participants was lower. These results constitute a new evidence of stress influencing loss aversion and are discussed within the alignment hypothesis, according to which stress aligns sensitivity to gains and losses.

1. Introduction

Losses have greater psychological impact and influence decision-making than gains of the same magnitude [1]; this phenomenon is called loss aversion [2] and is one of the most accepted judgmental biases in the social sciences. Loss aversion is considered a fundamental and generalizable principle [3], or even a stable behavioral trait [4], although this position is being called into question [5]. Gal & Rucker [3], for example, stressed that there is no firm evidence to support that losses have always more impact than gains and labeled this phenomenon as a fallacy. Moreover, Ert & Erev [6] stated that loss aversion only would emerge under certain very specific experimental manipulations such as when there are large amounts at stake or when people is submitted to long experiments in which no feedback is provided. Nevertheless, the current position is that loss aversion has moderators and a more contextualized view is advisable [3,5]. Many studies are focused on understanding which factors can shape loss aversion's expression. Since the incidence of stress has risen markedly over the past two decades [7], and many decisions are made under stress, this is one of the most studied factors and the focus of our study.

Recent reports state that stress influences loss aversion [8,9], but the specific direction and its mechanisms are still unknown. Most studies highlight that stress significantly reduces loss aversion (e.g., [8–10]), and these results are usually accommodated within the 'alignment' hypothesis postulates [8]. The biological correlates of the stress response are composed of both the immediate catecholamines rising and the latter cortisol release [11,12]. Both components, but specially cortisol, are known to modulate the brain reward-system by enhancing the dopamine striatal levels, then triggering additional reward salience [13,14]. Therefore, the alignment hypothesis suggests that stress would balance the susceptibility to gains and losses, the former being more attractive, thereby reducing loss aversion [8].

However, a common factor in all these studies is that they were carried out at a very early stage after the stressor onset (e.g., at 5 min) and, except for Margittai et al. [8], none provided hormonal measurements (e.g., [9,10]). So, since it cannot be even assured whether cortisol significantly raised at that point —its peak use to be found between 20 and 40 min [11,15]—, the loss aversion reduction could not be firmly attributed to this hormone nor to its influence over the reward-system. In fact, an alternative explanation could also fit. Concretely, early stages of the acute stress response could favor an optimal arousal level for the prefrontal cortex (PFC) functioning since this region is influenced

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by an inverted U-shaped curve of catecholamines [16]. The PFC constitutes the main hub for the executive control-network and it is responsible for a logical, rule-based and non-biased decision-making [11,16,15]. Previous studies showed, indeed, that using strategies that enhance PFC over the limbic system reduced loss aversion [17,18]. Therefore, the first mild-to-moderate catecholamines increase could be enhancing the PFC arousal and buffering loss aversion. As seen, this could also explain previous results on stress and loss aversion, instead of the proposed alignment hypothesis.

Following this line, a competing hypothesis on stress and loss aversion emerged. The 'salience-of-losses' hypothesis [8] proposes that, while early stages of stress could be beneficial for the PFC functioning, when both catecholamines and —mainly— cortisol have reached a high level, the executive control-network is suppressed and an alertness state is promoted by enhancing the salience-network [11,19]. As the salience-network shares key nodes with the loss aversion aversive-system —e.g., the amygdala—, the salience of losses would be increased and, loss aversion, amplified [8,19]. Then, by assessing decision-making 20 – 40 min after the stressor onset, when the cortisol peak is supposed to be reached and PFC activity should be diminished [11,15], rather than a decrease, a higher level of loss aversion may be found. Nevertheless, to our knowledge, no studies on loss aversion addressed this delayed point of the stress response and our aim is to fill this gap. Based on the salience-of-losses hypothesis, it was expected that, compared to controls, an experimental group would exhibit higher loss aversion after being submitted to a prominent psychosocial stressor such as the Trier Social Stress Test (TSST; [20]), specifically 30 min after the stressor onset.

2. Methods

2.1. Participants

Based on the large effect size found in previous works on stress and loss aversion [9,21], an a priori power analysis using G*Power indicated a requisite between 12 and 40 participants ($\eta_p^2 = [.18, 0.47]$, power = 80%, $\alpha = 0.05$) to perform an ANOVA and compare loss aversion between groups (experimental vs control group). To ensure an adequate statistical power, we recruited 94 participants and randomly distributed them into two groups, experimental ($N = 47$) and control ($N = 47$). Yet, two participants of the experimental group did not complete the experimental season, so our sample was finally composed by a total of 92 participants (age: $M = 19.11$, $SD = 1.87$; women: $N = 77$, 83.7%). They met the following inclusion criteria: not having cardiovascular, endocrine, neurological, or psychiatric diseases; not consuming more than 5 cigarettes a day; not consuming drugs habitually; not doing more than 10 h of exercise per week and not having experienced a highly stressful event in the last month. In addition, participants were asked to not perform extenuating exercise or take drugs or alcohol in the last 24 h, and not smoke or take stimulant drinks in the 2 h before the experimental session.

2.2. Procedure

This study was approved by the Ethics Research Committee of the University of Valencia in accordance with the ethical standards of the 1969 Declaration of Helsinki. Experimental session was carried out between 15:00 pm and 20:00 pm and lasted approximately one and a half hours. Participants were connected to the electrodermal activity (EDA) sensor and had 10 min of habituation. The last 5 min were taken as baseline. Then, experimental group was exposed to the virtual version of the Trier Social Stress Test (TSST-VR), while the control group was submitted to a distractor (watch a length-matched documentary). Before and after the stressor/distractor, participants were evaluated for positive and negative mood with the Positive and Negative Affect Scale (PANAS) and were asked about the subjective stress they felt. 30 min after the stressor/distractor onset, both groups performed an economical decision-making task to measure loss aversion.

2.3. Virtual reality version of the trier social stress test (TSST-VR)

To induce stress we utilized the software of Montero-López et al. [22] which constitutes a virtual-reality adaptation of the traditional TSST [20]. This software designs a 3D audience, which was projected onto a Screen of 27″ at 1 m from the participant. As in the original, the stressor consisted of four phases. First, participants faced the screen showing a 3D image of a stage curtain and were told that they had to give a speech to convince the audience that they were suitable for a position in their dream job. To lend credibility, a microphone and a camera were added, telling them that both the content and formal aspects of their speech would be analyzed in real time and that the virtual audience would react accordingly. The second phase, the anticipatory stress period, lasted 5 min and participants had to prepare the speech. The third phase was the speech itself. During this period, the virtual audience appeared, and participants had to deliver their speeches. They were instructed to speak for the entire 5 min without interruption. The virtual audience remained neutral for 2.5 min but was manipulated to show signs of impatience for the remaining time. Finally, the last phase of the stressful task was an arithmetic task and participants had to repeatedly subtract a fixed amount from a given number —e.g., subtracting 7 from 123 as quickly as possible—. If they completed a series, they were given a new number and started over. Similarly, if they made a mistake they also had to start over.

Comparable to the original TSST, this protocol demonstrated good reliability in inducing stress responses, which were manifested by the increase in EDA, blood pressure, catecholamines and cortisol levels, as well as in the negative mood [22]. To ensure that our stress induction was also effective, we assessed EDA, negative and positive mood, and subjective stress perception.

2.4. Electrodermal activity (EDA)

EDA is one of the most important physiological signals for detecting stress [23]. It has been consistently demonstrated that when the stress induction is effective, it is accompanied by the pronounced increase in EDA [24]. It was recorded and analyzed following recommendations for electrodermal measurements [25]. Two electrodes were placed on non-dominant hand —index and middle finger distal phalanges—, using isotonic gel to amplify the signal. BIOPAC, with EDA-100C transducer, 1000 Hz sampling frequency and AcqKnowledge software were also used. The electrodermal registry was re-sampled with the linear interpolation method at 250 Hz and filtered by smoothing factor with a median value of 5. The average of skin conductance level (SCL) in microsiemens (μS) was extracted from three different periods of 5 min: (1) baseline —last five min of the habituation period—, (2) stressor/-distractor —middle part— and (3) economical task.

2.5. Positive and negative affect registry (PANAS)

PANAS [26] is a 20 Likert-type items scale —from 1, more than usual, to 4, much less than usual— that evaluates positive and negative mood. Each dimension is composed of the sum of 10 items and ranges from 10 to 40 points. The higher the score, the more positive or negative the mood, respectively. PANAS was evaluated before and after the stressor/distractor.

2.6. Perceived stress

Before and after the stressor/distractor, participants were also asked about the subjective stress they felt. This question, designed *ad hoc* for the study, asked: "How much stress do you feel right now?". The answer was given on a Likert scale where 0 is "no stress", and 10 is "a lot of

stress".

2.7. Mixed gamble task (MGT)

To measure loss aversion, both groups performed a short version of MGT [27]. Each trial entailed a bet with one of the combinations randomly extracted from an 8 × 8 losses and gains matrix, until the 64 combinations were completed (see Fig. 1). Following gamble ranges used by Chandrasekhar Pammi et al. [27], as well as by Tom et al. [28] in the original task, gains could range from €100 to €380 in €40 increments, and losses from €50 to €190 in €20 increments. In each trial there was a 50% chance of gaining and 50% chance of losing. Participants had to decide whether to accept or reject the bet. They were instructed that €200 was their initial amount and each bet had to be done with that reference. Betting results were not presented immediately, however, they had to choose carefully in each trial since, at the end, four bets would be randomly picked and played heads or tails, affecting the initial amount. Loss aversion was obtained through the Prospect-Theory computational model [18].

2.8. Prospect-Theory computational model

The Prospect-Theory model [18] follows the classical approach of the Prospect Theory [1] were a bet would be accepted or rejected as a function of the expected utility that it brings to the individual. Following the original paper of Sokol-Hessner et al. [18], the utility of accepting a bet (U_{Accept}) depends on both the utility of the potential gain, estimated through the equation $u(x^{gain}) = x^{\rho}$; and the utility of the potential loss, estimated through the equation $u(x^{loss}) = - \lambda \times (-x)^{\rho}$. Finally, the probability of accepting a gamble is estimated through the SoftMax function, $P_{(Accept)} = 1/(1 + e^{-\mu(U(Accept) - U(Reject))})$; see Sokol-Hessner et al. [18] for a detailed math description. As can be seen, three parameters are derived from this model: λ —loss aversion coefficient—, ρ —the curvature of the utility function or risk attitude—, and μ —the logit or consistency parameter—. However, as we were interested in specifically addressing loss aversion, following Ahn et al. [29] and Molins et al. [9], we set the risk aversion parameter to 1 and using the Maximum likelihood estimation method, λ and μ were obtained. $\lambda = 1$ indicates that gains and losses were valued equally, however, when $\lambda > 1$, losses were overvalued relative to gains —loss aversion—. The logit parameter (μ) represents the amount of "randomness" in the subject's choices or, in other words, consistency over choices. Higher levels of the parameter would represent that participants rely more on rule-based decision-making [18].

These parameters were estimated for each participant through Hierarchical Bayesian Analyses (HBA; see Anh, 2008 for more details), performed with the hBayesDM package [29] for the R software. The hBayesDM uses Stan 2.1.1 [30] with the Hamiltonian Monte Carlo (HMC) algorithm as MCMC for sampling the posterior distributions. Following Alacreu-Crespo et al. [31], we drawn 40.000 samples, after burn-in of 23.333 samples, in three different chains —in sum, a total of 120.000 samples and 70.000 burn-in—. The Gelman-Rubin test [32] was used to study if the chains converged ($\hat{R}$) to the target distribution. $\hat{R}$ values were 1, which means that convergence was achieved. In addition, to confirm this convergence, the MCMC chains were visually inspected.

2.9. Statistical analyses

Outliers were analyzed with the 2.5 standard deviations method and Mahalanobis distance for repeatedly measured variables —e.g., EDA—. Kolmogorov-Smirnoff with Lilliefors correction was used to check normality. Analyses included repeated-measures ANOVAs, with the group —experimental vs control— as a between-participants factor, to test the stress induction effectiveness, both at the physiological (EDA) and at the subjective level —perceived stress, positive affect, and negative affect—. Moreover, the loss aversion level was compared between groups through one-way ANOVA. The α significance level was set at 0.05 and partial eta square (η_p^2) symbolizes the effect size. All analyses were performed with IBM SPSS Statistics 25.

3. Results

3.1. Preliminary analyses

Experimental and control groups were homogeneously distributed with no significant differences in age (experimental: $M = 19.36$, $SD = 2.14$; control: $M = 18.87$, $SD = 1.56$), $p = 0.21$; in BMI (experimental: $M = 22.01$, $SD = 3.05$; control: $M = 21.75$, $SD = 3.48$), $p = 0.69$; nor in socioeconomic status (experimental: $M = 6.42$, $SD = 1.17$; control: $M = 6.43$, $SD = 0.90$), $p = 0.98$. Moreover, there were more women than men, but the chi-square test revealed that both women (experimental: 84.4%; control: 83%) and men (experimental: 15.6%; control: 17%), $p = 0.84$, maintained similar proportion in both groups.

3.2. Stress induction

3.2.1. Physiological stress

A repeated-measures ANOVA including group —experimental vs. control— as a between-factor was performed to test whether the stress

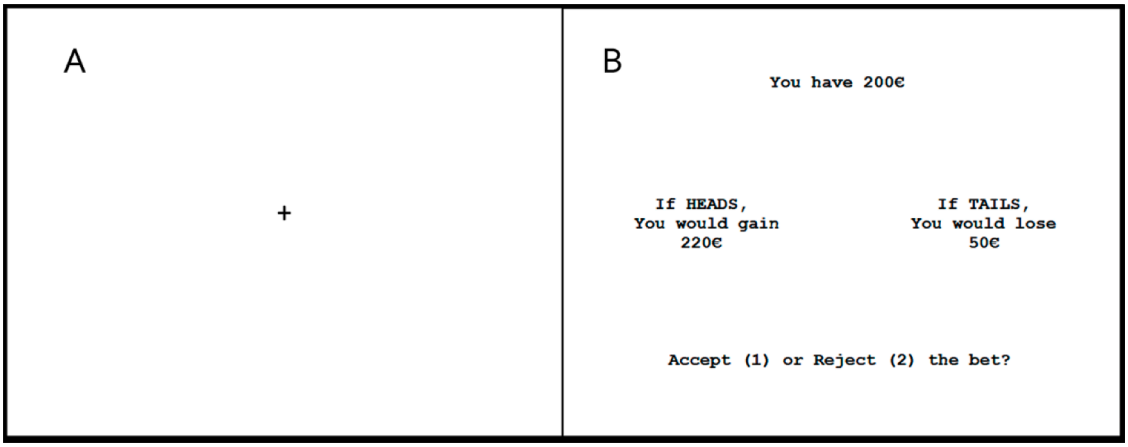

Fig. 1. A representative mixed gamble task trial.
Each of the 64 trials in the task consists of (A) 5 s of fixation point and (B) a bet. The bet offers a possible gain and a possible loss, both with a probability of 50% (heads or tails). The participant must decide whether to play or reject that bet.

induction was effective at the physiological level. Analyses revealed a significant moment (baseline vs. stressor/distractor vs. MGT) × group interaction, $F(2, 180) = 22.19$, $p < 0.001$, $\eta_p^2 = 0.20$, which indicated that the EDA evolution was different for both groups (see Fig. 2). We explored this effect further.

When contrasting by groups, the experimental group's EDA revealed a significant main effect for the moment, $F(1, 88) = 56.05$, $p < 0.001$, $\eta_p^2 = 0.56$, and *posthoc* comparisons indicated that every point of the protocol differed from each other (p's < 0.001); specifically: the highest EDA's level was found during the stressor, followed by the level exhibited during the MGT. The lowest level was found during the baseline. Similar results were found in the control group, where EDA also revealed a significant main effect for the moment, $F(1, 92) = 11.69$, $p < 0.001$, $\eta_p^2 = 0.21$. However, *posthoc* comparisons only revealed significant differences between the baseline EDA's level and both the level during the distractor, $p = 0.009$; and during the MGT, $p < 0.001$; while the latter two points did not differ between them, $p = 0.24$. All means can be consulted in Table 1.

Lastly, the intergroup analysis controlling for basal levels —see Table 1— revealed that, although both groups did not differ in their EDA at the baseline, the experimental group showed significantly higher EDA than the control group during both the stressor /distractor and the MGT.

3.2.2. Psychological stress

Regarding the psychological impact of the stress, repeated measures ANOVAs —including group as between-factor— were carried out to study differences pre- and post-stressor/distractor in the subjective-perceived stress and both the positive and the negative affect measured with PANAS. Focusing on the perceived stress, it was found a significant group × moment interaction, $F(1, 90) = 94.86$, $p < 0.001$, $\eta_p^2 = 0.51$. So, while the control group did not show significant differences between pre- and post-distractor levels, $F(1, 46) = 2.06$, $p = 0.15$, $\eta_p^2 = 0.044$; the experimental group experienced an increase in perceived stress after being submitted to the stressor, compared to their pre-stressor level, $F(1, 44) = 95.48$, $p < 0.001$, $\eta_p^2 = 0.68$ —means can be consulted in Table 1—. Complementarily, both groups did not differ at their basal level, but the perceived stress of the experimental group was significantly higher than the level reported by the control group after the stressor/distractor —see Table 1—.

By the other side, regarding positive affect assessed with PANAS, no pre-post changes or differences between groups were found (p's > 0.05). However, regarding negative affect, analyses also revealed a significant group × moment interaction, $F(1, 90) = 43.54$, $p < 0.001$, $\eta_p^2 = 0.32$. Experimental and control groups did not present differences pre-stressor/distractor —see Table 1—. Yet, the stress group suffered a significant increase in negative affect after the stressor, $F(1, 44) = 11.85$, $p = 0.001$, $\eta_p^2 = 0.21$; while the control group kept a similar level after the distractor, $F(1, 46) = 1.12$, $p = 0.11$, $\eta_p^2 = 0.031$. So, levels post stressor/distractor significantly differed between groups —again, means and statistics can be consulted in Table 1—.

3.3. Loss aversion

First, it was checked if our sample was loss averse. Both control ($M = 2.55$, $SD = 0.61$) and experimental ($M = 2.23$, $SD = 0.56$) groups showed an average λ —loss aversion— value higher than 1, indicating that both groups expressed loss aversion during MGT. However, the ANOVA revealed that the group submitted to stress (experimental group) manifested a significantly lower level of loss aversion than the control group, $F(1, 90) = 6.79$, $p = 0.011$, $\eta_p^2 = 0.07$. Moreover, regarding the second parameter yielded by the Prospect-Theory model, stressed participants showed a higher consistency (μ) in their decisions ($M = 0.064$, $SD = 0.01$) than the control group ($M = 0.038$, $SD = 0.008$), $F(1, 90) = 166.22$, $p < 0.001$, $\eta_p^2 = 0.64$.

4. Discussion

The present study examined how the late phase of the acute stress influences loss aversion. Intergroup analysis revealed that the experimental group's EDA was significantly higher compared to controls, not only during exposure to TSST-VR, but also during MGT. Specifically, with respect to their baseline, the experimental group suffered an increase in EDA of 42.85% during the stressor, comparable to the average increase (45.56%) observed in previous literature also using the TSST-VR [22,33–35]. In addition, the experimental group also showed a higher subjective stress perception, as well as a worse mood after exposure to TSST-VR, while the control group did not show any differences after watching the documentary. Thus, both physiological and psychological measures suggest that our stress manipulation worked. Nevertheless, and despite we addressed the latter stress response, our results showed a loss aversion reduction in the experimental group, contrary to the hypothesized based on the salience-of-losses hypothesis. Once again, stress seems capable of altering decision-making, but on the absence of complementary measures such as cortisol or neural activity, it is difficult to determine whether these results are in line with the alignment hypothesis —as is often proposed in previous literature— or whether other mechanisms are involved in this late phase of stress. The following arguments attempt to shed light on this debate, but caution is advised as this is only speculation that needs to be verified by future research.

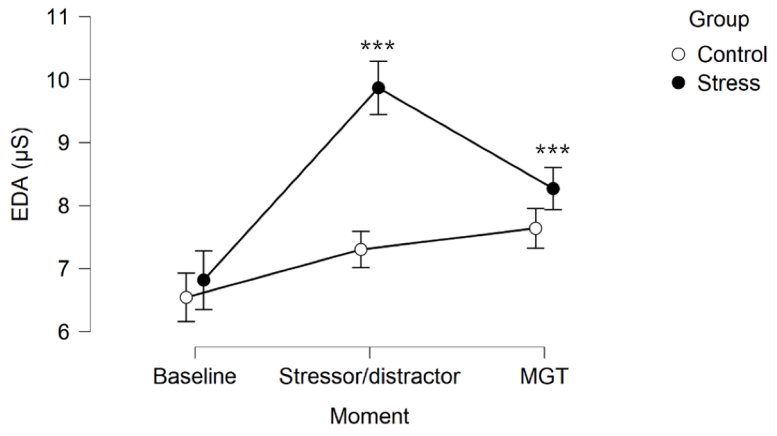

Fig. 2. Electrodermal activity during baseline, stressor/distractor, and MGT by group.
Experimental (stress) and Control groups significantly differed in their EDA level during the stressor/distractor and during the MGT. *** Significant contrast at the 0.001 level; $M \pm$ 95% confidence interval.

Table 1
Intergroup differences in electrodermal activity (EDA) during baseline, stressor/distractor, and MGT; and differences in perceived stress and negative mood with PANAS pre- and post-stressor/distractor.

		Experimental ($N = 45$)	Control ($N = 47$)	F	df between	df intra	p-value	η_p^2
EDA (μS)	Baseline	$M = 6.81 \pm 0.49$	$M = 6.54 \pm 0.42$	0.17	1	90	0.67	0.002
	Stressor / distractor	$M = 9.86 \pm 0.60$	$M = 7.30 \pm 0.42$	31.03	1	90	<0.001***	0.26
	MGT	$M = 8.27 \pm 0.48$	$M = 7.64 \pm 0.40$	23.88	1	90	<0.001***	0.21
Perceived Stress	Pre-Stress	$M = 3.76 \pm 1.92$	$M = 3.96 \pm 1.97$	0.24	1	90	0.62	0.003
	Post-Stress	$M = 6.38 \pm 1.81$	$M = 3.49 \pm 1.79$	116.16	1	90	<0.001***	0.56
PANAS -	Pre-Stress	$M = 20.76 \pm 4.95$	$M = 20.91 \pm 4.20$	0.028	1	90	0.86	0.000
	Post-Stress	$M = 23.47 \pm 6.07$	$M = 20.04 \pm 4.03$	30.56	1	90	<0.001***	0.25

M, mean; ± *SD*; df, degrees of freedom.
*** significant contrast at the 0.001 level.

As mentioned, and in accordance with the main premise of this work, stress influenced loss aversion. Both groups exhibited loss aversion values compatible with previous evidence, which stablished average values between 2 and 2.5 —i.e., that participants accepted gambles if gains were at least twice as large as losses— [1,36,28], yet the experimental group exhibited a significantly lower level of loss aversion. These results contradict our hypothesis and the salience-of-losses hypothesis by which stress would amplify loss aversion [8]. Instead, they may fit better with most previous evidence showing reductions in loss aversion under stress (e.g., [8–10]). Nevertheless, an important difference exists between all this evidence and our work, which may imply different mechanisms as responsible of the loss aversion reduction. As introduced, previous studies addressed decision-making at the very early stage of the stress response. Cortisol is one of the main implicated in the reward-system modulation, enhancing striatal dopamine and being able to balance sensitivity to gains and losses, thereafter, reducing loss aversion —alignment hypothesis— [8,19]; but cortisol peak concentrations in the brain are not reached within 20 min after stressor onset [11,16,37], which implies that 'the role of corticosteroids in the immediate stress response must be limited' ([11], p. 306). Therefore, as suggested Pabst et al. [16], the lower loss aversion found in works addressing early stress stages may be rather attributed to the beneficial role that the initial catecholaminergic release can exert on the PFC activity, which can buffer the manifestation of biases and enhance logical and rule-based decisions [17,38].

In contrast, our work addressed loss aversion 30 min after the stressor onset, focusing on the latter stress response. At this point, acute stress should have promoted an 'off-line' state in the PFC [39,40] and boosted the salience-network —which also includes striatum— by action of both catecholamines and cortisol [11,12]. Therefore, a loss aversion reduction at this point of the stress response may fit better with the alignment hypothesis. In fact, our data could be in line with previous evidence, where people made more conservative decisions until 18 min after the stressor onset, but the tendency was reversed, by making more risky choices, when the cortisol peak was reached 28 min after the stressor onset [16]. Complementarily, concurrent glucocorticoids and catecholamines prompted an alignment of reward- with loss-sensitivity, and thus diminished loss aversion [8]. Accordingly, and rather than into the salience-of-losses hypothesis [8], the effect of the latter, acute stress response on loss aversion may be better suited within the alignment hypothesis postulates [8]. Nevertheless, as stated, since we did not bring complementary neural or hormonal measures, this cannot be firmly assured, and further research is needed to shed light into the specific mechanisms whereby stress is reducing loss aversion. In fact, an alternative explanation to the alignment hypothesis could also fit.

Loss aversion parameter (λ) was computed considering how sensible is someone to losses relative to gains [18]. Then, a reduction in loss aversion could represent a higher sensitivity to gains, but also a lower sensitivity to losses, or even both. Therefore, the 'off-line' state that stress promotes in the PFC [11,40] may also account for our data. So, PFC plays a key role in valuation, stablishing preferences, as well as in reward and threats sensitivity [16,41,42]. In this line, Genauck et al. [43] found that alterations in the connectivity between PFC and limbic regions were associated with lower loss aversion. Thus, our results may also reflect that stressed individuals have difficulties when processing gains and losses and then, emotional influence in decision-making would be reduced. This may also explain why, according to the logit parameter (μ), stressed participants showed a greater reliance on a rule-based decision-making [18]. As seen, these arguments may compete with the alignment hypothesis and further research is needed to determine where they fit best.

Future studies must overcome limitations presented by our work, especially the absence of complementary hormonal or neural measures. Addressing catecholamines, noradrenaline, or opioids, will help to disentangle the specific mechanisms by which stress affects decision-making. It is also necessary to not consider stress in the singular, but rather to address whether its different phases —rapid vs. slow response— differentially influence decision-making, as some studies —including this one— already suggested [9,16]. For this reason, the inclusion in future studies of different groups directly testing the differences between early and late phase stress would be desirable. Additionally, our control situation —watching a documentary— had differences in form from the TSST-VR (e.g., one condition involves talking and the other does not). This could lead to the differences found in the EDA being attributed to movement rather than stress. Although our study also reflects through subjective measures that participants are actually perceiving stress, it would be desirable for future studies to further homogenize the stress and control conditions. On the other hand, previous studies showed sex differences in the psychophysiological response to stress, as well as in the effects of stress on decision-making [37,44]. All analyses were replicated controlling for sex and did not yield significant results. However, our sample was disproportionate and included significantly more women than men, so these analyses could be underpowered, and caution is advisable.

Despite limitations, our work is the first one addressing whether the latter phase of the acute stress response also modulates loss aversion. Specific mechanisms of such modulation remain unclear, and it is necessary further research to confirm whether they can be accounted within the reward alignment hypothesis. Nevertheless, we provide further evidence that stress can influence risky decision-making by reducing such a prominent bias as loss aversion and making decisions more logical. Our data reflect that stress can modulate how people perceives rewards and threats. Therefore, it should be considered when assessing and describing how people make decisions, opening the door to explanations that fit into a biological rationality, instead of the classic economic rationality.

Disclosure statement

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Data availability

Data will be made available on request.

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