Heart rate variability after vigorous physical exercise is positively related to loss aversion.

Background and Objectives of the article: Loss aversion bias, whereby losses loom larger than gains, can be reduced by stress. At the same time, vigorous physical exercise is a powerful neuroendocrine stressor and heart rate variability (HRV) provides an objective measure of the actual exercise impact, relative to each individual physical condition. Our aim was to study whether vigorous exercise can influence loss aversion, considering HRV in this relation. We hypothesized that the lower HRV derived from vigorous exercise (i.e. when stressor produced the most impact) would predict a lower loss aversion.

Methods: Two groups (Experimental, $N = 37$; Control, $N = 39$) completed a loss aversion task, but the experimental group was exposed to an acute physical stressor before.

Results: Results revealed a significant group x HRV interaction. In the control group, the lower HRV was associated with a higher loss aversion. Conversely, as hypothesized, the lower HRV levels derived from exercise were associated with a lesser loss aversion in the experimental group.

Conclusions: Results suggest that physiological changes from physical exercise could affect decision-making by reducing loss aversion.

Key words: Loss aversion; stress; physical exercise; heart rate variability; decision-making

Introduction

Imagine being hungry and suddenly you find an apple tree. In the tree there is also a snake. Maybe you can take the apple and escape unharmed, but you can also get bitten and fall sick without getting the fruit. That is, you can gain or lose something. Would you face the risk? This is a difficult decision that entails the need to consider trade-offs. However, even if the outcome's likelihoods were identical and the pleasure from the apple were proportional to the pain from the bite, our tendency could be to overestimate the latter and choose bite avoidance. Kahneman & Tversky (1979) stated that losses loom larger than gains. In other words, we are more sensitive to losses and our decisions are highly shaped by them: this is known as loss aversion bias.

Loss aversion is one of the most widely accepted ideas in the social sciences, where it is often considered a ubiquitous phenomenon (Gal & Rucker, 2018). This belief is supported by the fact that loss aversion has been documented in more than 50 countries (Wang et al., 2017), with different experimental paradigms (e.g. Margittai et al., 2018; Metz et al., 2020; Viswanathan et al., 2015); outside of the laboratory settings, like in politics (Berejikian & Early, 2013) or playing golf (Loewenstein & Levitt, 2011); and even in other species, such as monkeys (Chen et al., 2006) or rats (Constantinople et al., 2019). In addition, loss aversion has shown a stable neural base, which includes the executive network, the salience network and the reward system (Molins & Serrano, 2019; Sokol-Hessner & Rutledge, 2019), and whose resting state predicts and is correlated with the behavioral loss aversion (Canessa et al., 2017). All this seems to suggest that loss aversion could be a stable trait that affects everyone, all the time.

However, this ubiquity has also been challenged. The review of Gal & Rucker (2018, p.497) point out that "current evidence does not support that losses, on balance, tend to be any more impactful than gains". In fact, in some contexts, the gains could

have more impact than the losses (Gal & Rucker, 2018). Hochman & Yechiam (2011) found that even when the autonomic nervous system is more responsive to losses, this does not necessarily translate into greater behavioral loss aversion. The same authors also hold that the influence that losses exert on decisions would be better explained by attention-based and contrast-based processes rather than by an overestimation of losses (Yechiam & Hochman, 2013). Similarly, Ert & Erev (2013) indicate that loss aversion may only appear when experimental manipulation is conducive to it. For example, when large economic amounts are at stake or when faced with long experiments that do not provide feedback after the successive decisions. In this line, Gigerenzer (2015) pointed out that this bias and many others "cognitive illusions" could be similar to perceptive illusions, which only appear in specific (and often artificial) contexts, but do not usually occur in the real world. So, loss aversion could be caused by the way decisions are evaluated, but not in a real decision-making.

Despite these discrepancies, loss aversion continues gathering studies that try to clarify or nuance the phenomenon. It should be noted that, even if this bias were only an isolated cognitive illusion, it could still have important informational value. Thus, if in a context designed to make loss aversion appear, a person does not show the bias, or has a higher level than expected, this would allow to investigate the underlying processes associated with loss aversion, such as the greater or lesser emotional reactivity (Sokol-Hessner et al., 2013). On the other hand, the current thinking is that loss aversion is not necessarily an illusion nor an ubiquitous phenomenon (complex and variable) subject to contextual and individual influences (Gal & Rucker, 2018).

Some authors have focused on decomposing loss aversion and propose that it could be divided into the valuation bias and the response bias, leading to distinct types of loss-averse decision makers (Sheng et al., 2020). Other studies explore which

individual aspects, such as personality traits, may favor the emergence of bias (Boyce et al., 2016). And others focus on contextual aspects, such as culture (Wang et al., 2017), the presence of unpleasant stimuli (Stancak et al., 2015) or the influence of stress, among others. The latter is the focus of our study.

As McEwen (2000, p. 508) defined, stress is "a real or interpreted threat to the physiological or psychological integrity … that results in physiological and/or behavioral responses". Many decisions are made under stress, and accumulating evidence indicates that this factor can significantly influence loss aversion (Margittai et al., 2018); nevertheless, it remains unclear the direction of this effect. Currently, two hypotheses coexist. The "salience-of-losses" hypothesis suggests that acute stress reallocates the resources in the brain, producing a suppression of the executive network and higher activation of the salience network; thus, promoting fear, enhancing salience of losses and amplifying loss aversion (Metz et al., 2020). However, the most accepted is the "alignment" hypothesis (Metz et al., 2020), supported by the STARS model (Stress Triggers Additional Reward Salience) (Mather & Lighthall, 2012). According to this approach, under stress, losses will not loom larger than gains given that the latter become more attractive; this is, "reward and threat susceptibility are aligned" (Metz et al., 2020, p. 2).

Although contrary, both hypotheses would not be mutually exclusive and may reflect different stress phases, depending on their two main physiological pathways: the fast activation of the sympathetic nervous system (SNS), which triggers catecholamines release; and the slower hypothalamus-pituitary-adrenal axis (HPA-axis) activation, resulting in a secretion of cortisol (Hidalgo et al., 2019). In this line, Pabst, Brand, & Wolf (2013) found that during a moderate increase of catecholamines, participants took less risky decisions. Similarly, a positive relationship was found between

norepinephrine brain levels and loss aversion (Sokol-Hessner & Rutledge, 2019; Takahashi et al., 2013). However, Pabst et al (2013) also reported that when the cortisol peak was reached, participants' decisions became riskier, suggesting that a high cortisol level could reverse the catecholamines effect. Complementarily, concurrent glucocorticoid and catecholaminergic activity significantly reduced loss aversion, as described Margittai et al. (2018). So, it seems that depending on the SNS and HPA-axis activity, both hypotheses may be supported. Nevertheless, it is also necessary to consider that different stressors can differ in the expression and the magnitude of their physiological responses (Hidalgo et al., 2019).

Thus, it is important not to study stress in the singular, but how different stressors impact on loss aversion to further develop the current vision. When studying stress and loss aversion, the direct manipulation of catecholamines or cortisol is the most common practice (e.g. Margittai et al., 2018; Metz et al., 2020). Only a few physical or systemic stressors, such as Cold Pressor Test (Sokol-hessner et al., 2016) or an oxygen-depleted environment (hypoxia) (Pighin et al., 2014) have also been tested, reporting mixed results: no-effects and reduced loss aversion, respectively. However, no other stressors have been studied.

Physical exercise (hereafter exercise) is the gold standard recommendation to improve health, obtaining benefits by simply becoming more physically active (Warburton & Bredin, 2017). It is also a common activity in our daily living. According to Eurostat (2018), over 44% of the EU population was engaged in sports and exercise at least once a week in 2014, reaching up to 70% in the Nordic countries and Austria; and the Bureau of Labor Statistics (2018) shows that around 19% of the U.S. population practiced exercise each day in 2018. However, although healthy, exercise, and especially vigorous exercise, disturbs the homeostasis and forces organism to adapt to

these demands. As exercise intensity increases, so does the activation of SNS and HPA-axis, and consequently, the circulating concentrations of catecholamines and cortisol (Hackney, 2006). Thus, exercise is considered a powerful stressor of the neuroendocrine system (Hackney, 2006). In parallel, it has been widely reported that exercise can affect cognition. The regular practice is usually related to improvements, but the acute effect from a single bout is variable. While a moderate exercise showed benefits on cognitive functions, a heavy exercise bout, even briefly, was related to increased perceived stress levels (Hopkins et al., 2012) and impairments on cognitive processing (Shibasaki et al., 2019). Nevertheless, despite the exercise's role as stressor, its effect on cognition and how commonly it is practiced, its influence on decision-making and, more specifically, on loss aversion has never been studied. The aim of the present research is to fill this gap.

Considering the above, we expect that vigorous exercise will reduce loss aversion, as seem to do other stressors that produce the strong activation of both SNS and HPA-axis. However, an issue when studying exercise as stressor is that, even if the intensity exposure is systematized, physical condition can modulate the tolerance to it and, therefore, the real impact of the stressor (Hackney, 2006). Recently, heart rate variability (HRV) has been proposed as an objective non-invasive indicator of the exercise impact (Michael et al., 2017). HRV refers to the variation in the time interval between heartbeats, which occurs through the actions of the cardiac sympathetic (cSNA) and parasympathetic (cPNA) neural activity (Grossmann et al., 2016). It can be studied through many indicators, but concretely, those markers associated with the cPNA (cPNA-HRV) are systematically reduced as exercise intensity increases (Michael et al., 2017). Similarly, a greater preceding intensity is associated with a slower cPNA-HRV recovery (Michael et al., 2017). Therefore, at a constant intensity, measuring

cPNA-HRV provides information on the actual exercise impact relative to each individual physical condition. It is important to include this variable in the exercise–loss aversion relation to not consider solely the exercise practice, but how this exercise is really affecting. In this line, we hypothesize that, when measuring loss aversion with an economical decision-making task, the lower cPNA-HRV derived from vigorous exercise (i.e. when stressor produced the most impact), will predict the lower loss aversion levels.

Materials and methods

Participants.

According to Laborde, Mosley, & Thayer (2017), in a HRV study, a sample of 61 participants is required to detect medium effect size (d = 0.50, power = 80%, α = 0.05). Complementarily, although the current estimate of the usual effect size in psychological research is d = .4 (Brysbaert, 2019), previous studies specifically relating HRV and loss aversion obtained medium effect sizes (d = .5) (Mintoft et al., 2012; Sütterlin et al., 2011). Taking this size as a reference, we carried out an a priori power analysis using G*Power, which indicated a requisite of 74 participants (d = 0.50, power = 80%, α = 0.05) to perform a general lineal model studying main effect (exercise vs control group), covariate (cPNA-HRV) and their interaction (group x cPNA-HRV) on loss aversion.

We oversampled 16 participants to account for possible exclusions. 90 participants were recruited. 14 were eliminated (4 for drug consumption, 4 for accepting or rejecting bets by default when measuring loss aversion, and 6 for not reaching the intensity preset during exercise). A total of 76 participants (women: N = 61; age: M = 22.29, SD = 2.17) were finally included. All of them met the following inclusion

criteria: not having cardiovascular, endocrine, neurological or psychiatric diseases; not having impediments to practice exercise; not consuming more than 5 cigarettes a day; not consuming drugs habitually; not doing more than 10 hours of exercise per week; not having experienced a highly stressful event in the last month; not having practiced extenuating exercise nor consumed drugs 24h before and not having taken stimulant drinks in the 2h before the experiment. Participants were randomly distributed into two groups, experimental ($N = 37$) and control ($N = 39$).

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. All sessions were carried out between 15:30 pm and 19:00 pm and lasted approximately one hour and a half. The procedure was explained, and informed consent was signed. Afterwards, they were connected to the polygraph and had 10 minutes of rest as habituation. Only experimental group was exposed to stress (exercise), but both groups performed an economical decision-making task to measure loss aversion. The control group began directly this task, without waiting for the time that the stressor lasts, given the possible effect that this waiting time could produce, either causing boredom (another type of stressor), or relaxation (different level to the basal). Before and after the stressor, participants were evaluated for positive and negative mood with the Positive and Negative Affect registry, PANAS (Thompson, 2007).

Vigorous exercise stressor.

Participants completed 15 minutes in a cycloergometer. Adapting the protocol of Frith et al. (2017), first 5 minutes were used as warm-up. In the following 5, pedaling intensity was progressively increased. Finally, last 5 minutes had to be performed at 70

- 80% of maximum heart rate (HR_{max}), i.e. vigorous exercise (Ponce et al., 2019). The specific HR was calculated for each participant, using the classic formula of Karvonen et al. (1957), $\left((HR_{max} - HR_{resting}) \times \%intensity\right) + HR_{resting}$, were HR_{max} was estimated with the formula of Tanaka et al. (2001), $HR_{max} = 208 - (0.7 \times age)$; and $HR_{resting}$ was obtained averaging the last 5 minutes of the habituation period (baseline). On average, our sample had to be between 158.11 (*SD* = *3*.42) and 169.46 (*SD* = 2.61) bpm (70 and 80% HR_{max}, respectively).

Mixed Gamble Task (MGT).

Both groups performed a short version of MGT, adapted by Chandrasekhar Pammi et al. (2015). Each trial entailed a bet with one of the combinations randomly extracted from an 8 x 8 losses and gains matrix, until the 64 combinations were completed. Following the gamble ranges used by Chandrasekhar Pammi et al. (2015), as well as by Tom et al. (2007) in the original task, gains could range from €100 to €380 in €40 increments, and losses from €50 to €190 in €20 increments. Tom et al. (2007, p.516) selected these unbalanced ranges because "previous studies indicate that people are, on average, roughly twice as sensitive to losses as to gains; thus (they) expected that, for most participants, this range of gambles would elicit a wide range of attitudes". The authors expected to capture these attitudes with a four-options answer: from strongly accept to strongly reject the bet, although they then compiled the responses only as acceptance or rejection, without further analysis. We decided to retain the original unbalanced approach, presenting larger gains than losses.

This approach could bias and inflate loss aversion, suggesting that it would be better a balanced approach, as other authors have used (e.g. Barkley-Levenson et al., 2013; Canessa et al., 2017, 2013). However, given the extensive and recent literature

that uses the original unbalanced approach (e.g. Chandrasekhar Pammi et al., 2015, 2017; Charpentier et al., 2016; Chib et al., 2012; Duke et al., 2018; Engelmann et al., 2017), which, in addition, shows loss aversion values close to those originally indicated in the classic literature (Kahneman, 2003; Kahneman et al., 1991; Kahneman & Tversky, 1979, 1984), it seems appropriated to use this approach to measure loss aversion. By the other side, given the experimental nature of our study, choosing a balanced or unbalanced approach seems less relevant. Perhaps our task may produce higher values of loss aversion in general, but both groups are submitted to the same task, which still allows us to study if stress affects the differential expression of this phenomenon. This is the main objective. Nevertheless, the results must be interpreted according to the approach used.

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. Logistic regressions were performed with each participant using possible gains and losses as independent variables, and acceptance or rejection as dependent. Thus, β of gains and β of losses were obtained, being able to apply the formula of Tom et al. (2007): $\lambda = \frac{-\beta_{loss}}{\beta_{gain}}$, to calculate λ parameter, which represents loss aversion. $\lambda \geq 0.5$ signals the presence of loss aversion, but this value use to be closely to 2 (Canessa et al., 2017), i.e. the gain must exceed the loss by 2:1 to accept the bet.

Heart Rate Variability (HRV).

Electrocardiogram was recorded using Einthoven's third derivation with 3 reusable electrodes, BIOPAC MP150, an ECG-100C transducer and AcqKnowledge software. Sampling frequency was 1000 Hz. Following Task Force guidelines (1996), five-minute records were standardized. Thus, the 5 baseline minutes and the central 5 of MGT were exported to Kubios software to extract cPNA-HRV. Previously, ECG signals were visually analyzed to detect ectopic beats and other artifacts. 7 participants showed some ectopic beats which were corrected using the Heart Timing Signal method (Mateo & Laguna, 2003), and thus, they were included in the posterior analysis. Since all participants went through an habituation period before taking the baseline, and a training before MGT, the 5 minutes extracted for both periods had only few artifacts due to measurement noise. Nevertheless, in Kubios, each participant was analyzed individually and the threshold-based artefact correction algorithm (Tarvainen et al., 2014), which removes artefacts without distorting normal RR intervals, was applied. According to the recommendations of Tarvainen et al. (2014), since HRV is highly individual, the threshold value, which can range from very low to very strong, was adjusted individually. Finally, as a detrending method, it is, in order to control the possibly slow changes in mean HR during the recording, the smooth prior's filter (Tarvainen et al., 2014) was also applied.

According to Michael et al. (2017), the most reliable cPNA-HRV markers against exercise would be the following: for time domain, the root mean square of successive differences of R-R intervals (RMSSD) in milliseconds. For frequency domain, by means of Fast Fourier Transformation, the high-frequency band (0.15 - 0.40 Hz) in absolute (ms^2/Hz; HF_{powfft}) and normalized (HF_{nu}) units. Low band (LF_{powfft}; 0.04 - 0.15 Hz) and very low band (VLF_{powfft}; 0.0033 - 0.04 Hz) were also extracted, but only to compute HRV total power (TOT_{powfft}), needed to normalize HF_{powfft}: HF_{nu} =

$\frac{HF_{powfft}}{TOT_{powfft}} \times 100$. VLF_{powfft} was included in the denominator as recommend Reyes del Paso et al. (2013). However, LF_{powfft} and VLF_{powfft} are controversial[1], especially when exercise is involved, so their use is discouraged (Michael et al., 2017; Shaffer & Ginsberg, 2017).

In short, the cPNA-HRV markers included in our study were: RMSSD, HF_{powfft} and HF_{nu}, both in a tonic and a phasic level. Phasic level was computed subtracting baseline HRV to MGT HRV (i.e. reactivity). Finally, following Grossmann et al. (2016), to minimize the chance of α-inflation due to multiple testing, and to avoid cherry-picking a marker yielding desirable results from those included, we performed a principal component analysis (PCA) on cPNA-HRV markers and performed primary statistical analyses on the retained factor scores. PCA yielded a one-component solution (HRV-factor): variance explained: 80.58%; loadings: RMSSD = .94, HF_{powfft} = .96, HF_{nu} = .77. PCA was also performed in the phasic level with similar results (HRV_{phasic}-factor): variance explained: 77.96%; loadings: $RMSSD_{phasic}$ = .92, $HF_{powfft\text{-}phasic}$ = .96, $HF_{nu\text{-}phasic}$ = .75.

[1] The use of HF_{nu} could also be controversial since it is complementary to LF and VLF (adding up to 100), being affected by them and, as we said, the interpretation of these bands is not clear. However, we still decided to include this measure in the study for several reasons. First, because we have other main measures such as RMSSD and HF_{powfft} and we do not base our results exclusively on HF_{nu}. Laborde et al. (2027), in fact, recommend using RMSSD and HFpowfft as main indicators, but complement this with other measures related to vagal tone to see if the results can be generalized. This is good practice to avoid publication bias (Grossmann et al., 2016; Laborde et al., 2017). On the other hand, the use of HF_{nu} for other purposes, such as obtaining the LF/HF ratio, would present a greater risk of interpretative errors (Reyes del Paso et al., 2013; von Rosenberg et al., 2017), but in this case, it will only be used as a normalized measure that facilitates cross-subject comparison (von Rosenberg et al., 2017). This measure is useful to compare the relative weight of HF to the total, regardless of absolute levels, which may be subject to more individual variability (e.g. by sex). This measure also facilitates intra-subject comparison under different conditions (e.g. rest vs. exercise) (Shaffer & Ginsberg, 2017), which can be useful in our experimental design. Finally, previous studies on the relation between HRV and loss aversion, such as that of Mintoft et al. (2012) or that of Sütterlin et al. (2011) also include HF_{nu} as indicator, which may allow us to directly compare our results with the existing ones.

Respiratory Frequency (RF).

Laborde et al. (2017) recommend not to engage in routine correction of HRV for respiration, while still monitoring RF, especially after exercise, to check if it remains between 9 and 24 cycles per minute, needed for the proper interpretation of HF power band. Post-exercise RF (i.e. during MGT) was measured with a sampling frequency of 1000 KHz, using a breathing band under the chest muscles and the RSP-100C transducer. Signal was re-sampled at 50 Hz and filtered by digital Band Pass FIR filter (low cut-off frequency: 0.05 Hz; high cut-off frequency: 1 Hz). Visual inspection was performed to detect artefacts and correct them if needed. To extract RF, positive peaks were detected without removing the baseline and threshold was set at 0 ppm.

Statistical analyses.

Outliers were detected with the 2.5 standard deviations method and Mahalanobis distance. Kolmogorov-Smirnoff with Lilliefors correction was used to check normality. RMSSD and HF_{powfft} in baseline, and RMSSD, HF_{nu} and HF_{powfft} during MGT, were log10 normalized (Field, 2009). In addition, although HF_{nu} baseline followed normal distribution, it was also log10 transformed to ease the comparison. Analyses included Pearson's correlations and multivariate general linear models for cPNA-HRV and loss aversion. The α significance level was set at .05. Partial eta square (η^2_p) symbolizes the effect size and β-1 (power) represents power. All analyses were performed with IBM SPSS Statistics 23, except for the extraction of λ which was done with R i386.

Results

Preliminary Analysis.

As can be seen in Table 1, experimental and control groups were homogeneously distributed, with no significant differences in age, BMI, exercise hours

per week nor in positive/negative mood. There were more women than men, but both groups maintained the same proportion. It can also be checked that the experimental group's heart rate during last 5 minutes of exercise (HR 15') was within the expected range (between M = 158.11, SD = *3*.42 y M = 169.46, SD = 2.61). In addition, extra analysis for the experimental group showed that positive and negative mood pre- and post-exercise did not differ significantly (p = .32 and p = .52, respectively).

[Table 1 near here]

Impact of exercise on HRV

First, multivariate general lineal model on HRV showed a significant main effect of the moment (baseline vs. MGT), $F(3, 72) = 39.42$, $p < .001$, $\eta^2_p = .65$, power = 1; and a significant condition (experimental vs. control) x moment interaction, $F(3, 72) = 14.73$, $p < .001$, $\eta^2_p = .41$, power = 1. We explored this effect further. When contrasting by groups for differences in cPNA-HRV markers between the baseline and the MGT, all the experimental group variables were significantly lower in the MGT than in the baseline: RMSSD, $F(1, 36) = 168.1$, $p < .001$, $\eta^2_p = .82$, power = 1; HF_{powfft}, $F(1, 36) = 96.48$, $p < .001$, $\eta^2_p = .73$, power = 1; HF_{nu}, $F(1, 36) = 8.57$, $p = .007$, $\eta^2_p = .19$, power = .8. However, the control group only revealed differences in HF_{powfft}, $F(1, 38) = 7.54$, $p = .009$, $\eta^2_p = .17$, power = .76; and RMSSD, $F(1, 38) = 14.95$, $p < .001$, $\eta^2_p = .31$, power = .96; showing higher levels in the baseline than in the MGT. Means and SD can be consulted in Table 2. On the other hand, intergroup analysis revealed that both groups do not differ in any cPNA-HRV marker in the baseline (see Table 2). However, when these variables were contrasted after exercise (i.e. during the MGT), they all showed statistically significant differences. As we see in Table 2, the experimental group had lower RMSSD, HF_{powfft} and HF_{nu} values with respect to the control group. Figure 1 summarizes the results for an easy interpretation.

[Table 2 near here]

[Figure 1 near here]

Checking for normal Respiratory Frequency (RF).

The experimental group RF during MGT (M = 18.12, SD = 2.47) does not significantly differ from control group RF (M = 17.15, SD = 2.64), $F(1, 74) = 2.68$, p = .11. In addition, both groups show a normal breathing (between 9 and 24 bpm), making our results interpretable.

Loss aversion and HRV.

First, we checked if our sample was loss averse (i.e. $\lambda \geq 0.5$). Both, experimental (M_{λ} = 3.11, SD = 1.5), and control group (M_{λ} = 2.98, SD = 1.59), showed behavioral loss aversion. Moreover, we examined how condition (experimental vs. control), tonic HRV-factor, and their interaction predict λ parameter. BMI, exercise hours per week and respiration rate were included as covariates; results without covariates were very similar. We observed no significant main effects, p's > .05, but, as expected, a significant condition x HRV-factor interaction, B = 1.46, SE = 0.44, t = 3.27, p = .002, η^2_p = .17, power = .9. The same interaction was also found at the phasic level, B = 1.22, SE = 0.53, t = 2.27, p = .026, η^2_p = .08, power = .61. As Figure 2 and Table 3 indicate, in the experimental group, each HRV marker was positively associated with loss aversion. In contrast, only HF_{nu} and HRV-factor (at the tonic level) were associated with loss aversion in the control group; in addition, these relations were negative.

[Figure 2 near here]

[Table 3 near here]

Discussion

The present work provides the first direct test of the physical exercise as a stressor influencing loss aversion. As was expected, after a vigorous exercise bout, the lower cPNA-HRV, which would indicate the greater exercise impact, is associated with lower loss aversion levels. This relationship was robust across both time-method- and frequency-method-based markers of tonic and phasic cPNA-HRV. Therefore, we could think that exercise constitutes a stressor capable of influencing decision-making. These results will be discussed in depth below. Nevertheless, since this is the first evidence linking physical stress and loss aversion, caution is recommended in their interpretation until further research replicates these findings.

First, it is important to address whether the stress manipulation was effective. As we see, excepting for HF_{nu} in the control group, both groups showed lower cPNA-HRV during the betting task (MGT) respecting to their baseline. HRV is susceptible to many kinds of stressors and can be reduced against them (Ciabattoni et al., 2017). MGT itself could constitute an acute cognitive stressor since it is cognitively demanding (Ciabattoni et al., 2017) and, therefore, the decrease found may be attributable to the task itself. However, intergroup analysis during the MGT revealed that, in average, this decrement was significantly higher in the experimental group. Thus, reflecting the expected exercise influence.

Attending now to loss aversion bias, the values in our study (near to 3) are higher than 2-2.5, which is usually what is stated in the literature (Duke et al., 2018; Kahneman & Tversky, 1979; Tom et al., 2007). These values could be due to the use of a task with unbalanced gain/loss ranges, with gains larger than losses, which may artificially inflate loss aversion. However, as example, Barkley-Levenson et al. (2013) used a balanced approach and found loss aversion values even larger for both teenagers (up to 5.7) and adults (up to 3.3), which would indicate that there may be other factors,

and not the task itself, that explain the high value. Nevertheless, although the average loss aversion is usually 2-2.5, the literature also accepts as valid, both with balanced and unbalanced approaches, values close to 3 and higher (e.g. Barkley-Levenson et al., 2013; Chandrasekhar Pammi et al., 2017; Gelskov et al., 2015); therefore, it seems that our results would be generalizable to the rest of the field of study. Even so, they should be interpreted under the unbalanced approach we have used, and more research is needed to see if this could affect the expression of loss aversion.

Regarding the influence of stress on loss aversion, our data suggest that each group needs to be discussed separately. Focusing on the control group, most cPNA-HRV markers were not associated with loss aversion, but tonic HF_{nu} and HRV-factor did it in a negatively way. In fact, HRV-factor was explaining 20% of loss aversion variance, which is remarkable. This result is very similar to that found by Sütterlin et al. (2011), who reported that tonic HF_{nu} was negatively correlated and explained 23.2% of the variance of the framing effect, a phenomenon closely linked to loss aversion. The same can be observed with the result of Mintoft et al., (2012), who found an inverse correlation between HF_{nu} and the disposition effect, another phenomenon explained by loss aversion. As Sütterlin et al. (2011) argued, this findings could be explained by the reduced inhibitory capacity that people with lower HRV levels could have. According to dual-process approaches (Evans, 2008), biases are originated in the automatic, impulsive and emotional System 1; while the controlled and reflective System 2 is responsible for curbing these biases. In parallel, a lower HRV has been linked with self-control and emotion-regulation difficulties (Grossmann et al., 2016; Park & Thayer, 2014). Taken together, these evidences would give theoretical meaning to our results and would support the association between lower HRV and the more biased decisions.

However, this relation was reversed in the experimental group, where all cPNA-HRV markers, both at tonic and phasic level, were positively associated with loss aversion. As we mentioned, experimental group was affected by the exercise. Given the constant exercise intensity (70 – 80% HR_{max}), a steeper reduction in cPNA-HRV markers within the experimental group, would indicate a greater exercise impact (Michael et al., 2017). These variations in HRV reflect mechanical changes in the heart and do not necessarily wider changes in the organism, but as Michael et al. (2017) suggest, since these variations are produced by exposure to vigorous exercise stressor, it would be expected that they also reflect the known autonomous heart regulation during exercise, i.e. a PNS withdrawal and a SNS increase (White & Raven, 2014), in line with other stressors (Hidalgo et al., 2019). Thus, as was hypothesized, a higher exercise stressor impact would be linked to lesser loss aversion levels.

Our result seems to be consistent with the alignment hypothesis (Margittai et al., 2018), whereby stress may lead to a loss aversion decrease due to the reward system activity increasing. However, the present findings do not necessarily support such a mechanism. Exercise could be increasing the attractiveness of gains, but also reducing the aversion to losses, or doing both. In fact, there may be an alternative explanation that has not been considered to date. Stress in general, and also physical exercise in particular, can induce endogenous analgesia, reducing the perceived pain (Butler & Finn, 2009; St-Aubin et al., 2019). SNS and HPA-axis are also involved in this mechanism, but other substances such as endogenous opioids play a key role (Butler & Finn, 2009). In addition, pain and aversion have overlapping characteristics, both at their neural pathways and substrate level (Butler & Finn, 2009); indeed, some authors even talk about “pain of losses” when defining loss aversion (e.g. Hintze et al., 2015, p. 2). Therefore, a lower loss aversion associated to a higher stress impact could be also

explained by another mechanism, such as an endogenous analgesia induction. Nevertheless, this is only speculation and more research are needed, addressing complementary physiological and neural data, to further develop this novel hypothesis or shed light on the previous one (i.e. the alignment hypothesis).

In fact, the absence of such measures is the main limitation of our work. Given the exercise intensity, and how HRV is behaving, we can infer that participants are being physiologically stressed. However, we cannot explore the role of other factors such as catecholamines, cortisol or opioids since their exact levels are unknown. It is also important to stress that the control group did not engage in a placebo-like task. We decided that this group would not engage in any less vigorous exercise to avoid completely any physiological stress. We also did not make them wait for the time the stressor lasted in the experimental group (e.g. reading magazines), to avoid boredom (another type of stressor) or relaxation (physiological level different from the basal one). However, these decisions threaten the internal validity of our findings. How do we know that changes in loss aversion are not caused by the mere fact of being involved in a task and not by physical stress? Future designs must therefore take this important limitation into account.

Moreover, our results were found studying stress effects 5 minutes after the exercise (i.e. the recovery phase). As was explained, different results could be found depending on the stress phase and, therefore, further research is necessary to explore if our findings are replicated, for example, performing MGT still on the cycloergometer (i.e. the acute phase). It has to be also mentioned that, as PANAS questionnaire revealed, the exercise was not producing unpleasant emotions on our sample; that is, it was stressful but not distressful (Hackney, 2006). It is known that subjective perception also plays a key role in the stress response and maybe facing an unpleasant stimulus

could modify our results. However, subjective perception is not usually addressed when studying stress and loss aversion, being necessary its consideration in the future. Finally, previous data have reported age and sex differences when taking risk and facing stress (Hidalgo et al., 2019; van den Bos et al., 2009), but our study was made only with young people and the sample was predominantly female, preventing a thorough exploration of these issues. Future research should also address the influence of these variables.

Many debates remain about loss aversion, but it seems to be a phenomenon that could affect many of our decisions. At the same time, physical exercise is a common activity in our daily living which can be stressful for our organism. However, their relation had not been addressed to date. Although further research is necessary to investigate whether the reported results can be replicated and if they are behaviorally meaningful when facing a real-world decision, the present research provides a first step. Our work suggests that physiological changes derived from a high-intensity exercise could affect decision-making by reducing loss aversion. If future research supports this, these results could be relevant and should be considered when predicting risky decisions. Physical exercise may push individuals to take less biased decisions, but also to be less cautious when deciding. As Gigerenzer & Gaissmaier (2011) proposed, it is now necessary to consider the context in which decisions are made in order to understand whether their effect is beneficial or harmful. After all, it seems that if people were physically stressed, they might take the apple and ignore the snake.

Disclosure statement

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this manuscript.

Funding

This study was supported by a grant from Ministerio de Economía y Competitividad (grant number: PSI2016-78763-P).

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TABLE 1 | Homogeneity between groups and heart rate during stressor.

	Experimental (N = 37)	Control (N = 39)	*F*	gl between	gl intra	*p*-value
Age	*M* = 22.62 ± 2.67	*M* = 21.97 ± 1.54	1.69	1	74	.19
Sex						
Men	16.2%	23.1%	0.6†	1	15†	.43
Women	83.8%	76.9%	0.02†	1	61†	.89
BMI	*M* = 22.32 ± 2.97	*M* = 22.45 ± 2.79	0.03	1	74	.87
Exercise hours per week	*M* = 3.28 ± 2.61	*M* = 3.01 ± 2.39	1.1	1	74	.65
PANAS						
Positive mood	*M* = 27.54 ± 4.69	*M* = 28.1 ± 3.89	0.32	1	74	.57
Negative mood	*M* = 21.54 ± 4.53	*M* = 22.64 ± 4.54	1.11	1	74	.29
Vigorous exercise (bpm)						
HR 5’	*M* = 130.47 ± 12.94	-	-	-	-	-
HR 10’	*M* = 155.73 ± 9.21	-	-	-	-	-
HR 15’	*M* = 164.14 ± 4.8	-	-	-	-	-

M, mean; ±, *SD*; BMI, weight(kg) / height(m)^2; Positive mood, 10 items of positive mood added; Negative mood, 10 items of negative mood added; Vigorous exercise (bpm), beats per minute in 5-minute intervals. † these values correspond to χ^2 and not to *F,* as well as to *N* and not to gl intra.

TABLE 2 | HRV intergroup differences during baseline and Mixed Gamble Task (MGT)

		Experimental (N = 37)	Control (N = 39)	*F*	gl between	gl intra	*p*-value	η^2_p	*β*-1
Baseline	RMSSD	*M* = 45.53 ± 19.70	*M* = 44.25 ± 21.25	0.12	1	74	.72	.02	.064
	HFpowfft	*M* = 1030.22 ± 936.51	*M* = 831.72 ± 728.05	0.49	1	74	.48	.07	.107
	HFnu	*M* = 31.02 ± 14.01	*M* = 31.31 ± 12.34	0.009	1	74	.92	.00	.051
MGT	RMSSD	*M* = 19.9 ± 12.35	*M* = 32.92 ± 12.4	25.65**	1	74	< .001	.26	.99
	HFpowfft	*M* = 299.05 ± 472.54	*M* = 580.78 ± 497.74	22.19**	1	74	< .001	.23	.99
	HFnu	*M* = 26.14 ± 14.76	*M* = 34.17 ± 13.58	8.57**	1	74	.005	.10	.82

M, mean; ±, *SD*; Although some contrasts were made with variables transformed with log10, original means are shown to facilitate interpretation; ** significant contrast at the .01 level.

TABLE 3 | Correlations between λ and heart rate variability markers (tonic and phasic) during MGT (by group)

		Loss Aversion parameter (λ)		
		r	*N*	*p*-value
Experimental	RMSSD	.362*	36	.042
	HF_{powfft}	.424*	36	.016
	HF_{nu}	.435*	36	.013
	HRV-factor	.418*	36	.017
	Phasic RMSSD	.391*	36	.029
	Phasic HF_{powfft}	.363*	36	.045
	Phasic HF_{nu}	.422*	36	.018
	Phasic HRV-factor	.384*	36	.04
Control	RMSSD	-.105	38	.54
	HF_{powfft}	-.122	38	.47
	HF_{nu}	-.522**	38	.001
	HRV-factor	-.447**	38	.007
	Phasic RMSSD	-.06	38	.73
	Phasic HF_{powfft}	.008	38	.96
	Phasic HF_{nu}	-.202	38	.23
	Phasic HRV-factor	-.23	38	.19

p-values were calculated with Pearson correlations. ** significant correlation at the .01 level; *significant at the .05 level.