



Article

Spanish Validation of the Maastricht Acute Stress Test (MAST): A Cost-effective Stress Induction Protocol

Daniel Santos-Carrasco  and Luis Gonzalo De La Casa 

Departamento de Psicología Experimental, Universidad de Sevilla (Spain)

Abstract

The rise of stress studies has led to the existence of multiple stress induction protocols. However, cultural differences in stress reactivity are often overlooked. Therefore, this study aims to validate the Maastricht Acute Stress Test (MAST) in the Spanish population. A sample of 96 young adult participants was divided into an experimental group and a control group based on whether they were exposed to the MAST or a non-stressful control task. State anxiety and positive and negative affects were measured before and immediately after the protocol, while physiological stress (blood pressure and heart rate) was monitored throughout the experiment. The experimental group exhibited an increase in state anxiety, negative affect, and systolic and diastolic blood pressures after the MAST protocol. Participants with higher psychopathological risk presented higher scores of psychological stress than those with lower risk. Finally, it was found that participants with higher mathematical performance exhibited lower anxious reactivity following stressor exposure. Our results confirm the efficacy of the MAST and its validation for use in the Spanish population.

Keywords: anxiety; acute stress; MAST; stress reactivity; sympatho-adrenal-medullary (SAM) axis

(Received: 21 May 2024; revised: 23 December 2024; accepted: 21 January 2025)

Stress is one of the most extensively studied adaptive responses over the last century. Operationally, stress is understood as the organism's response to perceived demands or threats (Levine & Ursin, 1991). Exposure to stressful events leads to the activation of autonomic stress systems with the aim of restoring homeostatic levels (Ulrich-Lai & Herman, 2009). Physiologically, this entails an increase in the activity of the sympatho-adrenal-medullary (SAM) and hypothalamus-pituitary-adrenal (HPA) axes. Activation of the SAM axis results in the release of circulating catecholamines, such as adrenaline and noradrenaline, ultimately increasing heart rate or blood pressure (McEwen, 2008; Ulrich-Lai & Herman, 2009). On the other hand, the parallel activation of the better-known HPA axis leads to the release of glucocorticoids into the bloodstream, such as cortisol (Ulrich-Lai & Herman, 2009).

In the field of experimental stress research, two main types of stressors have traditionally been utilized: physical stressors (e.g., physical pain) and psychological stressors (e.g., difficult tasks) (Shields, 2020). A variety of stress tools of both natures have emerged over time, including high-difficulty tasks, such as progressive matrices or the Stroop test (Ferreira, 2019; Gianaros et al., 2005). However, due to the need for standardized tools to yield comparable results, protocols specifically designed for stress induction have become prevalent. Among them, two have stood out in the literature: the cold pressor test (CPT) (Lovallo, 1975), in which

the participant must immerse their hand in ice water (physical stressor); and the trier social stress test (TSST) (Kirschbaum et al., 1993), where the participant must undergo a job interview (psychological stressor).

These two stress tools have been the gold standard in acute stress induction. However, it has been observed that the type of stressor utilized activates different systems involved in the stress response. Physical stressors entail activation of the autonomic nervous system (HPA axis) through an immediate bodily reaction that requires activation of the hypothalamus and brainstem (Ulrich-Lai & Herman, 2009). In contrast, psychological stressors activate areas such as the thalamus or frontal lobes, with prefrontal-limbic connections that consequently activate the HPA axis (Dickerson & Kemeny, 2004). These differences lead to observations that the TSST appears to be more effective in maintaining elevated glucocorticoid levels over a longer period (Schwabe et al., 2008). However, physical stressors seem to have greater ease in activating the SAM axis (typically controlled via blood pressure, e.g., Chrousos, 2009; Chu et al., 2024; Lundberg & Frankenhaeuser, 1980). Hence, the higher concentration of glucocorticoids when participants are exposed to the TSST versus the CPT could be related to the nature of the stressor, making both types of stressors relevant for eliciting experimental stress (e.g., Skoluda et al., 2015).

Therefore, subsequent protocols have aimed to integrate both the physical and psychological components of the stressors. A new version of the CPT, the socially evaluated cold pressor test (SECPT) (Schwabe et al., 2008), was created. Although the SECPT has generally yielded better results than the CPT, it does not elicit as high stress responses as the TSST (Schwabe & Wolf, 2009; Smeets,

Corresponding author: Luis Gonzalo De la Casa; Email: delacasa@us.es

Cite this article: Santos-Carrasco, D., & De La Casa, L. G. (2025). Spanish Validation of the Maastricht Acute Stress Test (MAST): A Cost-effective Stress Induction Protocol. *The Spanish Journal of Psychology* 28, e1, 1–10. <https://doi.org/10.1017/SJP.2025.4>

© The Author(s), 2025. Published by Cambridge University Press on behalf of Universidad Complutense de Madrid and Colegio Oficial de la Psicología de Madrid. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

2011). This led Smeets et al. (2012) to analyze which components were the most effective in these protocols and merge them into the Maastricht Acute Stress Test (MAST). The MAST combines the immersion of the hand into ice water as the physical stressor and a complex arithmetic task as the psychological stressor. This tool has been validated to elicit robust physiological and psychological stress responses (e.g., Capello & Markus, 2014; Meyer et al., 2013; Shilton et al., 2017; Smeets et al., 2012), being more effective in eliciting these responses than both the CPT and the SECPT and having similar benchmark values to the TSST (Smeets et al., 2012).

However, it should be noted that there are many factors influencing stress reactivity, such as psychopathological risk. Thus, higher basal levels of depression and anxiety are associated with increased physiological (Kibler & Ma, 2004; Light et al., 1998) and psychological stress reactivity (de Rooij et al., 2010). However, other studies revealed that individuals with higher depressive symptoms exhibit a reduced cardiovascular response following stressor exposure (e.g., Chida & Hamer, 2008; Phillips et al., 2011). On the other hand, individuals who perceive the task as controllable appear to have lower stress reactivity (Glass et al., 1971). Another factor influencing stress reactivity is sex, as some studies have found that men tend to have higher reactivity to acute stress than women (Kirschbaum et al., 1992; Liu et al., 2017). Furthermore, beyond the need to address all these variables, an element that appears to be consistently overlooked when conducting stress research is the cultural factor. Thus, it has been found that the glucocorticoid response differs among cultures (e.g., Miller & Kirschbaum, 2019; Souza-Talarico et al., 2014). Consequently, cultural differences are found in stress appraisal, coping, reactivity, and maintenance (Bernardi et al., 2019; Popa et al., 2014; Urizar et al., 2019), as well as pain beliefs and tolerance (Hsieh et al., 2010; Nayak et al., 2000). A recent meta-analysis by Miller and Kirschbaum (2019) evaluated reactivity to the TSST in different cultures, reporting that Spanish people exhibited different levels of stress reactivity than other cultures, such as the Dutch and Australian population, where the MAST has already been validated (Smeets et al., 2012 and Shilton et al., 2017, respectively).

Therefore, it is important for standardized stress tools to be validated across cultures, ensuring their efficacy beyond the population in which they were initially tested. However, to our knowledge, no validation of the MAST has been conducted in the Spanish population to assess its efficacy in eliciting SAM axis stress responses. The present study aims to validate and evaluate the efficacy of the MAST in eliciting both psychological and physiological stress responses in the Spanish population.

From this main objective, several specific objectives and hypotheses emerge: (i) Analyze sex differences in reactivity to acute stress. It is expected to find a greater stress response in men than in women. (ii) Evaluate differences in stress reactivity depending on baseline levels of depression, anxiety, and stress. While we expect to find greater psychological reactivity among participants with higher baseline psychopathological risk, the hypothesis is uncertain regarding cardiovascular reactivity. (iii) Examine whether the performance index in mental arithmetic of the MAST is related to stress reactivity. We expect to find that higher arithmetic performance is associated with lower reactivity.

Method

Participants

To determine the required sample size, an a-priori power analysis was conducted using G*Power software (version 3.1, Faul et al.,

2007), with α set at .05 and $1 - \beta$ at .9. A review on stress induction studies indicated that the typically observed effect size is medium to high. Thus, being conservative, for an effect size estimated as medium ($\eta^2 = .06$), is needed a minimum of 88 participants (selecting F tests, repeated-measures ANOVA in G*Power, with four groups [Group: Experimental versus Control \times Sex: Men versus Women]). Anticipating potential withdrawals from the experiment, a sample size approximately 30% larger than necessary was recruited.

Therefore, the main sample consisted of a total of 120 participants, who were undergraduate psychology students at the University of Seville that participated in the study for course credits. These data were collected as part of a larger study intended to evaluate the effects of stress on prepulse inhibition of the startle response and cognitive function (Santos-Carrasco and De la Casa, 2024). Regarding inclusion criteria, the study included young adult Spanish participants who did not have a current or recent history (last 6 months) of any psychological, neurological, endocrine, or cardiovascular disorder, nor drug consumption (alcohol, psychoactive drugs, and other substances). In addition, it was verified that they did not present a baseline risk score for depression, anxiety, or stress through the DASS-21 scale. Thus, out of the total of 120 participants, one was excluded for presenting hypertension values and 23 were excluded for having high scores in the DASS-21 classified as "Severe" or "Extremely Severe" (following the guidelines of Shields et al., 2020).

This left a total sample of 96 participants, who were randomly assigned to two groups: Experimental Group ($n = 47$; M/W: 18/29) and Control Group ($n = 49$; M/W = 16/33). The mean age for the groups was similar, with 20.55 years ($SD = 3.16$) for the Experimental Group and 21.29 years ($SD = 4.19$) for the Control Group. However, in secondary analyses, where the relationship between depression, anxiety, and stress levels with stress reactivity was examined, the complete sample was considered except for the participant with hypertension ($n = 119$; Control Group = 60 participants [M/W = 21/39]; Experimental Group = 59 participants [M/W = 21/38]).

Instruments

Depression, anxiety and stress scale (DASS-21)

The Depression, Anxiety, and Stress Scale (DASS-21) developed by Lovibond & Lovibond (1995) was administered. This self-administered scale consists of a total of 21 Likert-type items with four options, making it a cost-effective screening tool for detecting depressive, anxious, or stress symptoms (e.g., Ruiz et al., 2017). The Spanish validated version by Daza et al. (2002) was utilized. This version has demonstrated high levels of internal consistency in both Spanish university and general populations, with Cronbach's alpha coefficients ranging from 0.90 to 0.95 (Fonseca-Pedrero et al., 2010; Ruiz et al., 2017). The subscales show alpha values between 0.80 and 0.92 for the depression subscale, 0.73–0.87 for the anxiety subscale, and 0.81 to 0.86 for the stress subscale. In addition, it presents good levels of convergent, discriminant, and internal validity (Daza et al., 2002), with a hierarchical factor structure of three first-order factors (depression, anxiety, and stress) and a second-order factor (emotional symptoms) (e.g., Fonseca-Pedrero et al., 2010; Ruiz et al., 2017). In the current sample, Cronbach's alpha coefficients were 0.85, 0.81, 0.62, and 0.68 for the total score of the DASS-21 and the subscales of depression, anxiety, and stress, respectively.

Maastricht acute stress test (MAST)

To induce acute stress, the standardized MAST protocol (Smeets et al., 2012) was used, which has been validated for inducing both

physiological and psychological stress (e.g., Moses et al., 2023; Shilton et al., 2017). To adapt the MAST protocol for the Spanish sample, two independent translations were performed by two translators, following the back-translation procedure according to international guidelines and recommendations (Muñiz et al., 2013; Muñiz & Bartram, 2007). Specifically, a bilingual native Spanish–English translator translated the MAST protocol from English to Spanish. Then, a bilingual native English–Spanish translator translated the protocol from Spanish back into English. Following this, the scale was retranslated into Spanish by an independent reviewer using the back-translation procedure. Finally, discrepancies between the original protocol and the back-translated version were resolved by members of the research team. Subsequently, a pilot test was conducted with five Spanish participants (two males, three females; aged 18–27 years) to ensure comprehension of all instructions. After making necessary corrections, the final version of the MAST for the Spanish population was obtained.

Participants in the experimental group underwent the MAST protocol, which starts with a 5 min preparation period where they read instructions on PowerPoint slides. This was followed by a 10 min stress induction phase, combining psychological and physical stress trials. During the psychological trials, participants count backwards from 2043 in steps of 17, and during the physical trials, they immerse their hand in cold water (4–6°C). The experimenter continuously monitors them, providing only negative feedback, and participants believe they are being videotaped. The control group performed a non-stress-related task, alternating between submerging their hand in warm water (35–37°C) and counting aloud from 1 to 25. There was no performance evaluation or videotaping in the control group.

As a novel element compared to previous studies using the MAST, the correct arithmetical responses of participants in the experimental group were registered during the psychological stress trials.

State-trait anxiety inventory (STAI)

To assess trait anxiety at baseline, the STAI was administered in its trait version to screen for participants with elevated anxiety levels. This self-report scale, developed by Spielberger et al. (1982), includes the trait anxiety subscale (STAI-T), consisting of 20 Likert-type items with four response options. In addition, to evaluate changes in state anxiety from baseline (prior to MAST) to post-stress, the state anxiety subscale of the STAI (STAI-S) was administered just before and immediately after completing the protocol. This subscale also comprises 20 Likert-type items but focuses on assessing current levels of anxiety.

The Spanish version of the inventory was used (Buela-Casal et al., 2011). This version has a rescaled scoring (ranging from 0 to 3 points), resulting in a total score ranging from 0 to 60. This version of the STAI demonstrates high internal consistency, with Cronbach's alpha values ranging from 0.80 to 0.97 for the STAI-S, and between 0.88 and 0.95 for the STAI-T (Fonseca-Pedrero et al., 2012; Guillén Riquelme & Buela Casal, 2011; Ortuño Sierra et al., 2016). The scale demonstrates good test-retest reliability and evidence supporting its internal structure for both the three-factor (Guillén Riquelme & Buela Casal, 2011) and four-factor models (Fonseca-Pedrero et al., 2012; Ortuño Sierra et al., 2016). It also shows concurrent validity when compared with other anxiety scales such as the IPAT, TMAS, and Burns-A (Ortuño Sierra et al., 2016).

In the current sample, Cronbach's alpha coefficients were 0.89, 0.89, and 0.92 for the STAI-T, the STAI-S before the MAST and the STAI-S after the MAST, respectively. Temporal stability was calculated using the test-retest reliability coefficient known as

intraclass correlation coefficient (ICC, selecting two-way random for absolute agreement of the average measure). To do this, the data from the control group and the experimental group were analyzed separately, as each underwent a different treatment (stress induction via MAST or a stress-free task). In the current sample, the ICC for the STAI-S in the control group was 0.88, and for the experimental group, it was 0.73 (all p 's < .001).

Positive affect and negative affect scales (PANAS)

The PANAS scale (Watson et al., 1988) was also administered before the MAST and immediately after stress induction. This scale assesses both positive and negative affect through two subscales of 10 items each, where participants rate how they currently identify with various positive (positive affect) and negative (negative affect) items on a scale from 1 to 5. The Spanish version of the PANAS (Sandin et al., 1999) was utilized. This version of the PANAS demonstrates high internal consistency, with Cronbach's alpha values ranging from 0.86 to 0.90 for the positive affect subscale and from 0.84 to 0.91 for the negative affect subscale, both in university students and the general population (e.g., Ortuño-Sierra et al., 2015; Sandin et al., 1999). In addition, the scale shows evidence supporting its internal structure for both a two-dimension affect model (Sandin et al., 1999) and a three-dimension model (Ortuño-Sierra et al., 2015).

In the current sample, Cronbach's alpha coefficients were 0.75, 0.82, and 0.89 for the total score of the PANAS, and the subscales of positive and negative affect before the MAST, respectively. After the MAST, the alpha values for these indices were 0.74, 0.85, and 0.90. In addition, temporal stability was calculated through the test-retest reliability coefficient known as ICC (selecting two-way random for absolute agreement of the average measure). To do this, the data from the control group and the experimental group were analyzed separately, as each underwent a different treatment (stress induction via MAST or a stress-free task). In the current sample, the ICC value for positive affect was 0.91, and for negative affect, it was 0.77 for the control group. For the experimental group, the ICC values were 0.79 and 0.66 for the positive and negative affect subscales, respectively (all p 's < .001).

Physiological stress measures

Parameters directly related to the sympathetic-adrenal-medullary (SAM) axis were monitored, as this system provides reliable markers of stress reactivity (e.g., Chrousos, 2009; Chu et al., 2024; Skoluda et al., 2015). Thus, systolic blood pressure (SBP), diastolic blood pressure (DBP), as well as heart rate (HR) were evaluated. These measurements were registered with an automatic oscillometer wrist cuff positioned on the upper participant's arm (Medisana MTP PLUS) a device already validated in previous studies (Erdem et al., 2011).

The physiological stress measurements were registered at four consecutive time points for each participant to obtain a comprehensive monitoring of the duration of acute stress at the physiological level. Thus, measurements were extended up to 30 min following exposure to the protocol, following the procedure outlined by Shilton et al. (2017). Accordingly, measurements were obtained at baseline before the stress induction protocol [TBase], immediately after completing the MAST [T0], and at 15 [T15] and 35 min [T35] after protocol completion.

Procedure

This study was approved by the Research Ethics Committee of the University of Seville (0559-N23). The experimental sessions were

conducted between 9:00 AM and 7:00 PM in an isolated room. Participants were instructed not to consume caffeinated, energy, or sugary drinks, as well as to avoid heavy meals, nicotine, alcohol, or other drugs, and intense physical exercise in the 2 h preceding the study. Upon arrival, participants read the study information sheet, and they voluntarily decided to participate by signing the informed consent form. Subsequently, participants were interviewed to check inclusion criteria, after which they completed the DASS-21, STAI-T, STAI-S, and PANAS scales, followed by the initial measurement of blood pressure and heart rate [TBase]. Afterward, participants were exposed to either the MAST protocol or the non-stress-related control task. Upon completion, physiological and psychological stress measures were reassessed [T0]. Blood pressure and heart rate were reevaluated at 15 [T15] and 35 min [T35] following MAST exposure.

Data analysis

All statistical analyses were conducted using version 29 of SPSS. To determine the significance, alpha was set at 0.05 for all tests. Omega squared (ω^2) was used as the effect size measure in all ANOVA analyses. In addition, the assumption of normality was tested using the Shapiro-Wilk normality test. The Greenhouse–Geisser correction was applied when the sphericity assumption was violated, as verified using Mauchly's sphericity test. Post-hoc analyses using Bonferroni correction for multiple comparisons were performed. The Bonferroni method was chosen as it is sufficiently conservative and less susceptible to Type I errors due to its limits on alpha inflation (e.g., McHugh, 2011; Midway et al., 2020). When the assumption of normality was not met, the corresponding non-parametric test was utilized. There was no missing data.

Results

Demographic and baseline differences

To assess age and sex differences between groups, Mann–Whitney's tests were used. There were no differences between groups in terms of age ($u = 1210.5$, $p = .65$, $d = .05$) nor sex ($u = 1086.5$, $p = .57$, $d = .06$).

Mean scores on the DASS-21 subscales (depression, anxiety, and stress) and STAI-T are shown in Table 1. A mixed 2×2 ANOVA (Group: Experimental versus Control \times Sex: Men versus Women) was conducted on DASS-21 subscales and STAI-T scores. The main effect of Group was not significant in any case (all F 's < 1). The main effect of Sex on stress and trait anxiety scores was significant, $F(1,92) = 3.97$, $p = .049$, $\omega^2 = .03$, and $F(1,92) = 7.77$, $p = .006$, $\omega^2 = .065$, respectively, with women reporting higher levels of both stress and trait anxiety than men. The main effect of sex on depression score was non-significant ($p = .215$). Only the Group \times Sex interaction for the anxiety score was significant, $F(1,92) = 11.23$, $p = .001$, $\omega^2 = .096$ (all p 's $> .15$ for the remaining interactions). Post-hoc comparisons (Bonferroni correction for multiple comparisons, $p_{\text{bonf}} < .05$) conducted to identify the source of the Group \times Sex interaction revealed that women in the control group reported more anxiety than men but the opposite occurred in the experimental group.

Physiological and psychological stress responses

Regarding physiological stress responses, preliminary one-way ANOVAs were conducted on baseline SBP, DBP, and HR with Group and Sex as main factors (Groups: Control versus Experimental \times Sex:

Table 1. Mean scores for STAI-T and DASS-21 (depression, anxiety, and stress subscales) before experimental treatment and STAI-S, PANAS+, and PANAS- scores before and after response to the MAST in both groups, stratified by gender. Standard deviations appear between brackets

	Control group		Experimental group	
	Men	Women	Men	Women
STAI-T baseline	14.56 (6.66)	22.88 (10.39)	18.89 (9.27)	21.48 (8.71)
DASS-D baseline	2.37 (2.60)	3.82 (3.14)	3.00 (3.03)	3.10 (2.68)
DASS-A baseline	1.75 (1.44)	3.73 (2.01)	3.67 (2.27)	2.65 (2.33)
DASS-S baseline	5.62 (3.61)	7.54 (2.83)	6.11 (2.49)	6.62 (2.61)
STAI-S pre	12.75 (8.21)	15.15 (8.34)	14.00 (7.32)	14.10 (5.49)
STAI-S post	10.50 (7.62)	13.27 (8.19)	18.78 (9.35)	17.83 (8.28)
PANAS + pre	37.69 (5.09)	36.36 (5.99)	36.83 (4.57)	34.97 (5.62)
PANAS + Post	37.00 (6.41)	35.76 (5.93)	35.39 (4.67)	33.52 (6.17)
PANAS- pre	14.06 (4.57)	15.61 (6.60)	14.78 (5.15)	13.69 (3.29)
PANAS- post	12.94 (3.89)	14.27 (4.96)	16.00 (5.04)	16.59 (6.61)

Men versus Women). The main effect of groups was non-significant for all cardiovascular measures (all F 's ≤ 1). However, significant main effects of sex were found for SBP ($F(1,92) = 10.57$, $p = .002$, $\omega^2 = .092$) and HR values ($F(1,92) = 4.05$, $p = .047$, $\omega^2 = .031$), with women presenting lower SBP and higher HR than men. Neither the main effect of sex for DBP nor any Group \times Sex interaction was significant (all p 's $> .29$). The means, standard deviations, and statistics for all the one-way ANOVA comparisons conducted are given in Table S1 (Supplementary Material).

The mean scores of SBP are displayed in Figure 1. The analyses of SBP were conducted with a $2 \times 2 \times 4$ repeated measures mixed ANOVA (Group: Control versus Experimental \times Sex: Men versus Women \times Period: TBaseline versus T0 versus T15 versus T35). The main effect of Period was significant, $F(3,276) = 4.13$, $p = .007$, $\omega^2 = .007$, revealing a progressive reduction of SBP in both groups. The Period \times Group interaction was also significant, $F(3,276) = 8.07$, $p < .001$, $\omega^2 = .017$. Post-hoc analyses (Bonferroni correction for multiple comparisons, $p_{\text{bonf}} < .05$) revealed that the interaction was due to a gradual reduction in SBP for the Control Group, and an increase for the Experimental Group from TBaseline to T0 (immediately after the MAST protocol). The SBP peak in the experimental group at T0 was significantly higher than all other time points (all p 's $\text{bonf} < .042$). The main effect of Group and Sex was also significant, $F(1,92) = 10.46$, $p = .002$, $\omega^2 = .048$, and $F(1,92) = 21.88$, $p < .001$, $\omega^2 = .101$, respectively. The main effect of Group was due to participants in the Experimental Group showing an overall higher SBP

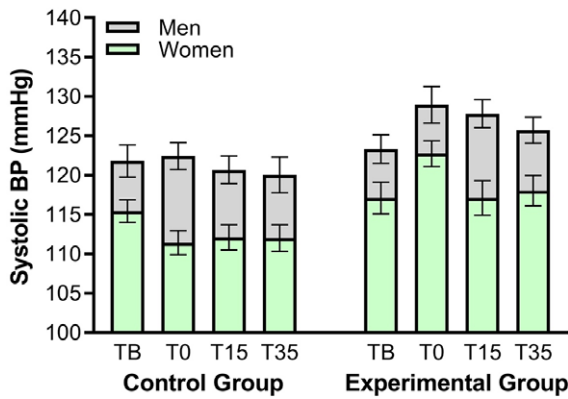


Figure 1. Mean systolic blood pressure values before and after the MAST for Control and Experimental groups, stratified by gender. Error bars represent SEMs.

than the Control Group. The main effect of Sex reflects lower SBP values for women than for men at all time points in both groups. Neither the Period \times Sex, Group \times Sex, nor the three-way interactions were significant (all p 's $>$.13).

All DBP results obtained by the participants can be observed in Figure 2. A $2 \times 2 \times 4$ repeated-measures mixed ANOVA was conducted on DBP scores (Group: Control versus Experimental \times Sex: Men versus Women \times Period: TBaseline versus T0 versus T15 versus T35). Mauchly's test of sphericity indicated a violation of the sphericity assumption for DBP measures, so the Greenhouse-Geisser correction was used. The main effect of Period was significant, $F(2.224) = 4.39, p = .009, \omega^2 = .009$, showing a progressive decrease of DBP in both groups. The Period \times Group interaction was also significant, $F(2.224) = 6.13, p = .001, \omega^2 = .013$. Post-hoc analyses (Bonferroni correction for multiple comparisons, $p_{\text{bonf}} < .05$) revealed that while the Control Group exhibited a gradual reduction in DBP from baseline, participants in the Experimental Group experienced a continuous increase in DBP, peaking after the MAST (T0) with levels significantly higher than those at baseline and T35 (both p 's $\text{bonf} < .001$), but comparable to T15 ($p_{\text{bonf}} = 1$). The main effect of Group was significant, $F(1.92) = 9.09, p = .003, \omega^2 = .042$. This effect was due to higher DBP in the Experimental compared to the Control Group. Neither the main effect of sex nor the remaining interactions were significant (all p 's $>$.06).

The HR values throughout the experiment can be observed in Figure 3. A $2 \times 2 \times 4$ repeated-measures mixed ANOVA (Group: Control versus Experimental \times Sex: Men versus Women \times Period:

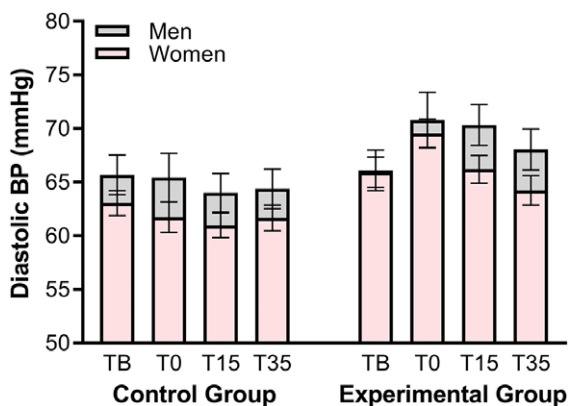


Figure 2. Mean diastolic blood pressure values before and after the MAST for Control and Experimental groups, stratified by gender. Error bars represent SEMs.

TBaseline versus T0 versus T15 versus T35), with Greenhouse-Geisser correction, was applied to the HR results. The analyses revealed a significant main effect of Period, $F(3.252) = 5.15, p = .002, \omega^2 = .009$, due to a progressive decrease in HR across time points in both groups. No more significant differences appeared (all p 's $>$.07).

Regarding the psychological stress measures, a one-way ANOVA with Group and Sex as main factors (Group: Control versus Experimental \times Sex: Men versus Women) was conducted on pre-induction period scores (baseline). Neither the main effects nor any interaction resulted significant (all p s $>$.31).

All psychological stress measures values are depicted in Table 1 and were submitted to a $2 \times 2 \times 2$ repeated measures mixed ANOVA (Group: Control versus Experimental \times Sex: Men versus Women \times Period: Pre- versus Post-manipulation). Regarding changes in state anxiety, a main effect of Group was observed, $F(1.92) = 4.31, p = .041, \omega^2 = .017$, with participants in the Experimental Group showing higher anxiety levels than those in the Control Group. The Period \times Group interaction was also significant, $F(1.92) = 26.71, p < .001, \omega^2 = .035$. Post-hoc analyses (Bonferroni correction for multiple comparisons, $p_{\text{bonf}} < .05$) revealed a significant increase in anxiety from pre- to post-MAST period in the Experimental Group, but a reduction in the Control Group. Neither the main effects of Period and Sex, nor any of the interactions were significant (all p 's $>$.07).

For positive affect scores, the main effect of Period was significant, $F(1.92) = 6.09, p = .01, \omega^2 = .007$, indicating a reduction in positive affect in both groups from the pre- to post-manipulation. All other comparisons were non-significant (all p 's $>$.17). Regarding negative affect, the Period \times Group interaction resulted significant, $F(1.92) = 10.92, p = .001, \omega^2 = .021$. Post-hoc comparisons (Bonferroni correction for multiple comparisons, $p_{\text{bonf}} < .05$) revealed a significant increase in negative affect from the pre- to post-MAST period for the Experimental but a reduction for the Control Group. The statistics for all the mixed repeated measures ANOVA comparisons conducted are given in Table S2 (Supplementary Material).

Correlations between stress reactivity, arithmetical performance, and psychological measures

The performance in the mental arithmetic task of the MAST was correlated with stress reactivity measures. These analyses only considered data from participants in the Experimental Group, since the Control Group did not undergo this task. The scores on the four

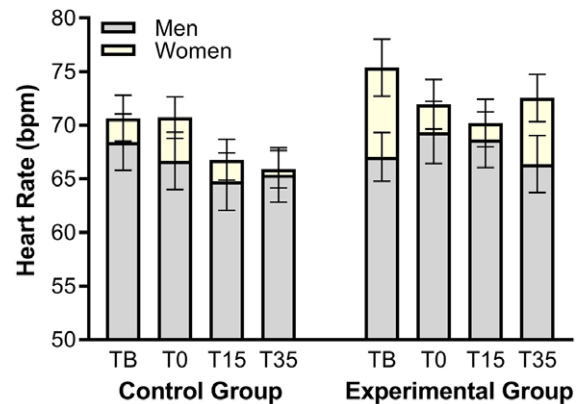


Figure 3. Mean heart rate values and after the MAST for Control and Experimental groups, stratified by gender. Error bars represent SEMs.

mental arithmetic trials as well as the total performance were correlated with the stress reactivity coefficients. Negative significant correlations were found between the total performance in mental arithmetic and STAI-S reactivity ($\rho = -.29, p = .043$), and between the STAI-S reactivity and performance in mental arithmetic in the first trial ($\rho = -.39, p = .007$). All other correlations were non-significant (all p 's $> .05$). The graphical representation of all correlations in a heatmap and the scatterplot depicting the relationship between anxious reactivity and total arithmetic performance can be observed in left and right sections of Figure 4, respectively.

For the following analyses, the total sample ($n = 119$) was considered to see if there were differences in stress response based on whether participants had psychopathological risk ("Severe" or "Extremely Severe" scores) or not ("Normal" or "Moderate" scores) based on their scores on the DASS-21. First, scores on the DASS-21 and STAI-T scales were correlated with stress reactivity coefficients and arithmetic performance. None of the correlations were significant (all p 's $> .05$). Next, repeated-measures ANOVAs (Risk: Psychopathological risk versus Low risk \times Period: Pre- versus Post-manipulation) were conducted. No differences were found between subjects in physiological reactivity (all p 's $> .53$), but differences were found in two of the psychological stress measures. Specifically, participants with higher psychopathological risk exhibited higher levels of state anxiety ($F(1,115) = 16.34, p < .001, \omega^2 = .062$) and negative affect ($F(1,115) = 13.54, p < .001, \omega^2 = .051$) both pre- and post-MAST as compared to the non-risk group. Figure 5 depicts the values obtained by participants with higher and lower psychopathological risk in state anxiety (left section) and negative affect (right section).

Discussion

The present study was aimed to validate the MAST in the Spanish population. The results indicated that the experimental group showed an increase in SBP and DBP from baseline to immediately after the MAST. Moreover, an increased psychological stress reactivity was found, as evidenced by the augmented state anxiety and negative affect from baseline to post-MAST in the experimental

group. In addition, participants with higher psychopathological risk (depression, anxiety, and stress subscales from the DASS-21) exhibited higher state anxiety and negative affect scores both before and after exposure to the stressor. Finally, a significant correlation was found between math performance and anxiety reactivity, indicating that participants with higher arithmetic performance exhibited lower anxiety reactivity following MAST.

The increases from baseline to post-MAST period observed in the Experimental Group for SBP and DBP are consistent with previous studies using this protocol (e.g., Smeets et al., 2012; Shilton et al., 2017), supporting its ability to elicit robust autonomic responses. However, there was a reduced HR variability, which in fact may indicate the effective functioning of the homeostatic system whereby HR recovery post-stressor exposure is relatively fast (e.g., Goswami et al., 2010; Shilton et al., 2017; Steptoe et al., 2003). As for the sexual differences, it was found that women exhibited lower SBP and higher HR than men at baseline, a sexual discrepancy in cardiovascular indexes that is consistent with previous studies (e.g., Ben-Dov et al., 2008). However, there were no sexual differences in DBP, possibly due to the greater sensitivity of SBP in detecting such differences (e.g., Kajantie & Phillips, 2006). On the other hand, no sexual differences were found in stress reactivity, diverging from findings in studies where higher reactivity is generally observed among men (e.g., Kirschbaum et al., 1992; Kudielka & Kirschbaum, 2005; Liu et al., 2017). This discrepancy may be due to the lack of control for menstrual cycle hormonal fluctuations or sexual orientation, factors known to influence stress response (Juster et al., 2015; Kirschbaum et al., 1999). In addition, it is worth noting that previous studies using the MAST and cardiovascular measures to assess physiological stress reactivity did not evaluate sexual differences, which may suggest the insensitivity of this stressor in eliciting sex-dependent stress responses.

Participants in the Experimental Group showed a significant increase in state anxiety and negative affect from baseline to post-exposure, unlike the Control Group, which showed a decrease. This aligns with studies reporting increased negative states in MAST participants (e.g., Bos et al., 2014; Meyer et al., 2013; Smeets et al., 2012). The reduction in the Control Group likely reflects habituation to the experimental room, previously

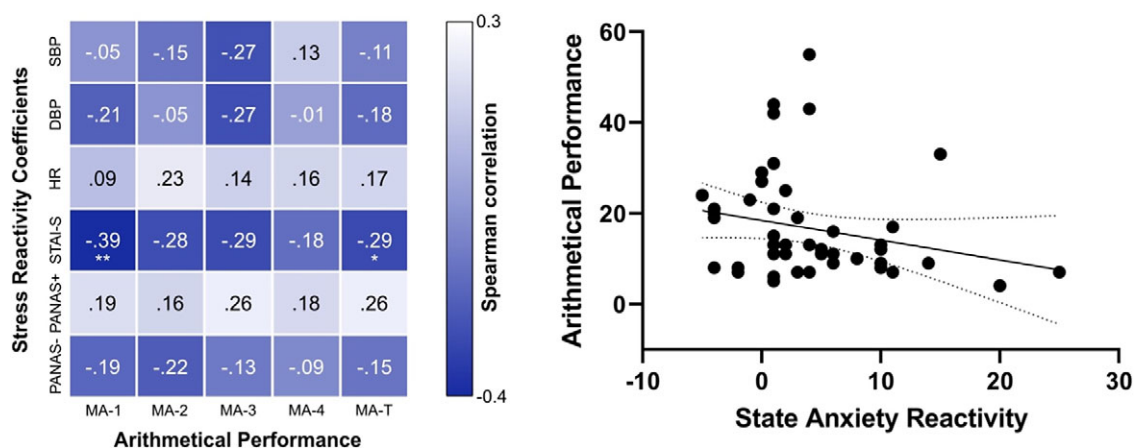


Figure 4. Heatmap of correlations between arithmetical performance and stress reactivity coefficients. Statistical differences are represented by * ($p \leq .05$) and ** ($p \leq .01$) (left section), and Scatterplot of the correlation between total arithmetical performance and state anxiety reactivity (right section). Dotted lines represent the 95% mean confident interval.

Note. Abbreviations: DBP, diastolic blood pressure; HR, heart rate; MA-1, arithmetical performance in the first trial; MA-2, arithmetical performance in the second trial; MA-3, arithmetical performance in the third trial; MA-4, arithmetical performance in the fourth trial; MA-T, total arithmetical performance; PANAS-, negative affect; PANAS+, positive affect; SBP, systolic blood pressure; STAI-S, state anxiety

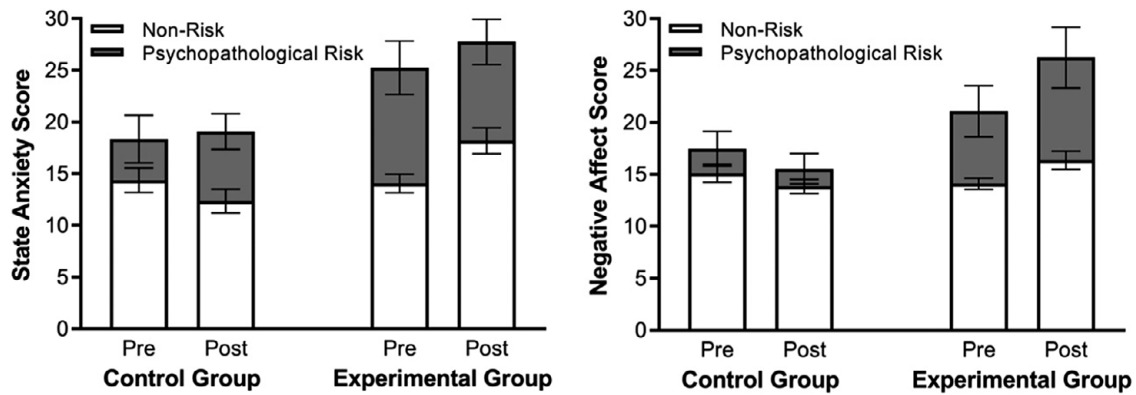


Figure 5. Mean values of state anxiety (left section) and negative affect (right section) before and after the MAST for Control and Experimental groups, stratified by the level of psychopathological risk. Error bars represent SEMs.

documented as a potentially threatening situation (e.g., Nash & Heiss, 1967; Soto et al., 2012). Both groups experienced a decline in positive affect, possibly due to the monotonous nature of the tasks (e.g., Chin et al., 2017; Rubenking, 2017). Contrary to our initial hypotheses, no sex differences appeared in psychological stress reactivity, possibly due to the insensitivity of the psychological scales (e.g., Lim et al., 2020; Trofimova, 2013) or the MAST itself.

To this point, having established the effectiveness of the MAST, we can state that this protocol has been validated in a young-adult Spanish sample. However, given that the nature of this validation inherently considers cultural differences in stress reactivity (e.g., Bernardi et al., 2019; Souza-Talarico et al., 2014; Urizar et al., 2019), it is relevant to discuss whether the results found in the Spanish sample are similar to those found in other cultures. Thus, our results on psychological stress reactivity were similar to that of previous studies (e.g., Smeets et al., 2012; Shilton et al., 2017; Bos et al., 2014), with the impossibility of verifying similarity in terms of the duration of psychological stress since we only registered them at two time points (pre- and post-MAST). As for cardiovascular reactivity, Smeets et al. (2012) found a significant BP increase from baseline after MAST in Dutch people, and a return to baseline values after 5 min (Smeets et al., 2012). Similarly, a subsequent study, also with Dutch population, found that BP peak persisted until 10 min after MAST exposure (Bos et al., 2014). In a sample of Australian population, it appeared an increase in BP from baseline to 20 min after MAST exposure (Shilton et al., 2017).

A meta-analysis by Miller and Kirschbaum (2019) found that Spanish and Dutch populations show lower stress responses after TSST compared to Australians, who exhibit higher reactivity. Our findings align with this, as the cardiovascular stress response in our Experimental Group was short-lived, similar to the Dutch sample, whereas other studies found more sustained responses, like in Australians. Notably, acute stress measured with glucocorticoids (e.g., salivary cortisol -HPA axis-) typically lasts longer than with cardiovascular responses (e.g., SAM axis) (e.g., Hidalgo et al., 2020; Mirete et al., 2021; Smeets et al., 2012). Therefore, the short duration of cardiovascular stress response reflects the autonomic system's quick action to restore homeostasis (e.g., Goswami et al., 2010; Steptoe et al., 2003). The significant differences observed confirm the MAST protocol's success in generating acute stress. Thus, MAST is effective in eliciting robust autonomic stress responses in the Spanish population, using both physical and psychological stressors to analyze effects on behavior and cognition.

In addition, the results revealed that participants with the highest psychopathological risk exhibited the greatest levels of anxiety and negative affect both before and immediately after the MAST. This supports our hypothesis, as well as replicates previous findings indicating that participants with higher anxiety and depression scores display greater psychological stress reactivity (e.g., de Rooij et al., 2010). However, we did not find differences between groups in physiological measures, unlike other studies that have reported either an increase (Kibler & Ma, 2004; Light et al., 1998) or a reduction (Chida & Hamer, 2008; Phillips et al., 2011) in cardiovascular stress reactivity. Regarding arithmetic performance, we found that participants with higher performance showed a reduced change in anxiety levels from baseline to the post-MAST period. This is consistent with a key element in stress induction, namely the sense of uncontrollability (e.g., Dickerson & Kemeny, 2004; Shields, 2020). Thus, when participants perceive tasks as controllable, they report lower levels of stress reactivity (e.g., Glass et al., 1971), suggesting that having control over the arithmetical task would lead to perceiving it as controllable, thereby increasing self-efficacy, which, in turn, is associated with lower stress levels (e.g., Nierop et al., 2008). However, these results should be interpreted with caution since, to our knowledge, this is the first time that the arithmetic index from the MAST has been independently analyzed. Moreover, as no baseline assessment of the participants' arithmetic performance was conducted, it is not possible to determine whether higher arithmetical performance influences stress reactivity, or vice versa.

Some possible limitations of our study must be considered. Thus, it could have been relevant to record more time points from MAST protocol presentation for the psychological stress assessment to evaluate changes in subjective stress. Moreover, participants in our study performed some cognitive tasks after the post-MAST measurements. While these tasks were not stressful in nature, they could have induced changes in BP or HR measurements. However, this seems unlikely given that all participants (both in Control and Experimental groups) performed the same tasks, the tasks were fully counterbalanced, and the results of cardiovascular reactivity were similar to previous reports using the MAST. In future studies, it would be interesting to compare the extent to which acute stress reactivity to the stressor is similar to baseline values in individuals affected by pathological acute stress, aiming to evaluate the ecological validity of the MAST. In addition, it is important to note that the relationship between arithmetic performance and stress reactivity is difficult to discern in this study because we do not have a baseline measure of arithmetic performance. Future studies using the MAST

should include a baseline arithmetic measure to explore this relationship in depth, considering the possible role of self-efficacy in subjective stress induction. Finally, continuing the research initiated by Miller and Kirschbaum (2019) to further validate these experimental stressors across different cultures will enable comparisons of stress reactivity between cultures.

In summary, our study tested the efficacy of the MAST to induce robust acute stress responses in a sample of young Spanish adults. Specifically, participants exposed to the MAST, as opposed to controls, exhibited a significant increase in state anxiety, negative affect, as well as a rise in SBP and DBP. Therefore, we have validated MAST as an effective stress induction protocol in the Spanish population, with some values differing from those reported in other cultures. However, since university students may not be fully representative of the general population in Spain, follow-up studies are recommended to test the generalizability of the results with more diverse samples. Thus, considering the impact of culture on stress reactivity, it is imperative to continue validating this and other stress protocols to analyze how similar or different cultures are under stress.

Supplementary material. To view supplementary material for this article, please visit <http://doi.org/10.1017/SJP.2025.4>.

Data availability statement. The data and materials that support the findings of this study are available from the corresponding author upon request to comply with the protection of personal data of the participants.

Acknowledgment. We would like to thank Sergio Villa-Consuegra, María de los Ángeles Cintado, Gabriel González and Lucía Cárcel for technical advice and Dr. Tom Smeets for sharing with us the materials of the MAST.

Author contribution. D.S.-C.: Conceptualization, data curation, formal analysis, investigation, methodology, writing (original draft, reviewing and editing); L.G.D.I.C.: Conceptualization, formal analysis, funding acquisition, methodology, project administration, supervision, writing (original draft, reviewing and editing).

Funding statement. This research was funded by the Agencia Estatal de Investigación (AEI) of Spain (grant n°: PID2019-107530GB-I00/AEI/10.13039/501100011033) and by the Spanish Ministerio de Ciencia, Innovación y Universidades (PhD grant ref. FPU21/00344).

Competing interests. The authors declare none.

References

- Ben-Dov, I. Z., Mekler, J., & Bursztyn, M. (2008). Sex Differences in Ambulatory Blood Pressure Monitoring. *The American Journal of Medicine*, *121*(6), 509–514. <https://doi.org/10.1016/j.amjmed.2008.02.019>
- Bernardi, J., Engelbrecht, A., & Jobson, L. (2019). The impact of culture on cognitive appraisals: Implications for the development, maintenance, and treatment of posttraumatic stress disorder. *Clinical Psychologist*, *23*(2), 91–102. <https://doi.org/10.1111/cp.12161>
- Bos, M. G. N., Jacobs van Goethem, T. H., Beckers, T., & Kindt, M. (2014). Cortisol response mediates the effect of post-activation stress exposure on contextualization of emotional memories. *Psychoneuroendocrinology*, *50*, 72–84. <https://doi.org/10.1016/j.psyneuen.2014.07.030>
- Buela-Casal, G., Guillén-Riquelme, A., & Seisdedos Cubero, N. (2011). *Cuestionario de ansiedad estado-rasgo: Adaptación española*. TEA Ediciones.
- Capello, A. E. M., & Markus, C. R. (2014). Effect of sub chronic tryptophan supplementation on stress-induced cortisol and appetite in subjects differing in 5-HTTLPR genotype and trait neuroticism. *Psychoneuroendocrinology*, *45*, 96–107. <https://doi.org/10.1016/j.psyneuen.2014.03.005>
- Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin*, *134*(6), 829–885. <https://doi.org/10.1037/a0013342>
- Chin, A., Markey, A., Bhargava, S., Kassam, K. S., & Loewenstein, G. (2017). Bored in the USA: Experience sampling and boredom in everyday life. *Emotion*, *17*(2), 359–368. <https://doi.org/10.1037/emo0000232>
- Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology*, *5*(7), 374–381. <https://doi.org/10.1038/nrendo.2009.106>
- Chu, B., Marwaha, K., Sanvictores, T., & Ayers, D. (2024). Physiology, Stress Reaction. In StatPearls. StatPearls Publishing. <http://www.ncbi.nlm.nih.gov/books/NBK541120/>
- Daza, P., Novy, D. M., Stanley, M. A., & Averill, P. (2002). The depression anxiety stress scale-21: Spanish translation and validation with a hispanic sample. *Journal of Psychopathology and Behavioral Assessment*, *24*(3), 195–205. <https://doi.org/10.1023/A:1016014818163>
- de Rooij, S. R., Schene, A. H., Phillips, D. I., & Roseboom, T. J. (2010). Depression and anxiety: Associations with biological and perceived stress reactivity to a psychological stress protocol in a middle-aged population. *Psychoneuroendocrinology*, *35*(6), 866–877. <https://doi.org/10.1016/j.psyneuen.2009.11.011>
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 355–391. <https://doi.org/10.1037/0033-2909.130.3.355>
- Erdem, E., Aydogdu, T., & Akpolat, T. (2011). Validation of the medisana MTP Plus upper arm blood pressure monitor, for self-measurement, according to the European Society of Hypertension International Protocol revision 2010. *Blood Pressure Monitoring*, *16*(1), 43. <https://doi.org/10.1097/MBP.0b013e3283419623>
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175–191. <https://doi.org/10.3758/BF03193146>
- Ferreira, S. O. (2019). Emotional activation in human beings: Procedures for experimental stress induction. *Psicologia USP*, *30*, e180176. <https://doi.org/10.1590/0103-6564e20180176>
- Fonseca-Pedrero, E., Paino, M., Lemos-Giráldez, S., & Muñiz, J. (2010). Propiedades psicométricas de la depression anxiety and stress scales-21 (DASS-21) en universitarios Españoles. [Psychometric properties of the Depression Anxiety and Stress Scales-21 (DASS-21) in Spanish college students]. *Ansiedad y Estrés*, *16*(2–3), 215–226.
- Fonseca-Pedrero, E., Paino, M., Sierra-Baigrie, S., Lennox-Girald, S., & Muniz, J. (2012). Propiedades psicométricas del “Cuestionario de ansiedad estado-rasgo” (STAI) en universitarios. [Psychometric properties of the State-Trait Anxiety Inventory (STAI) in college students.]. *Behavioral Psychology*, *20*(3), 547–561.
- Gianaros, P. J., Derbshire, S. W. G., May, J. C., Siegle, G. J., Gamalo, M. A., & Jennings, J. R. (2005). Anterior cingulate activity correlates with blood pressure during stress. *Psychophysiology*, *42*(6), 627–635. <https://doi.org/10.1111/j.1469-8986.2005.00366.x>
- Glass, D. C., Reim, B., & Singer, J. E. (1971). Behavioral consequences of adaptation to controllable and uncontrollable noise. *Journal of Experimental Social Psychology*, *7*(2), 244–257. [https://doi.org/10.1016/0022-1031\(71\)90070-9](https://doi.org/10.1016/0022-1031(71)90070-9)
- Goswami, N., Lackner, H. K., Papousek, I., Jezova, D., Hinghofer-Szalkay, H., & Montani, J.-P. (2010). Rate of cardiovascular recovery to combined or separate orthostatic and mental challenges. *International Journal of Psychophysiology*, *75*(1), 54–62. <https://doi.org/10.1016/j.ijpsycho.2009.11.005>
- Guillén-Riquelme, A., & Buela Casal, G. (2011). Actualización psicométrica y funcionamiento diferencial de los ítems en el State Trait Anxiety Inventory (STAI). *Psicothema*, *23*(3), 510–515.
- Hidalgo, V., Villada, C., & Salvador, A. (2020). Post-encoding stress does not enhance memory consolidation: The role of cortisol and testosterone reactivity. *Brain Sciences*, *10*(12), 995. <https://doi.org/10.3390/brainsci10120995>
- Hsieh, A. Y., Tripp, D. A., Ji, L.-J., & Sullivan, M. J. L. (2010). Comparisons of catastrophizing, pain attitudes, and cold-pressor pain experience between Chinese and European Canadian young adults. *The Journal of Pain*, *11*(11), 1187–1194. <https://doi.org/10.1016/j.jpain.2010.02.015>
- Juster, R.-P., Hatzenbuehler, M. L., Mendrek, A., Pfaus, J. G., Smith, N. G., Johnson, P. J., Lefebvre-Louis, J.-P., Raymond, C., Marin, M.-F., Sindi, S., Lupien, S. J., & Pruessner, J. C. (2015). Sexual orientation modulates

- endocrine stress reactivity. *Biological Psychiatry*, 77(7), 668–676. <https://doi.org/10.1016/j.biopsych.2014.08.013>
- Kajantie, E., & Phillips, D. I. W. (2006). The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology*, 31(2), 151–178. <https://doi.org/10.1016/j.psyneuen.2005.07.002>
- Kibler, J. L., & Ma, M. (2004). Depressive symptoms and cardiovascular reactivity to laboratory behavioral stress. *International Journal of Behavioral Medicine*, 11(2), 81–87. https://doi.org/10.1207/s15327558ijbm1102_3
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154. <https://doi.org/10.1097/00006842-199903000-00006>
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The ‘Trier Social Stress Test’—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28(1–2), 76–81. <https://doi.org/10.1159/000119004>
- Kirschbaum, C., Wüst, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine*, 54(6), 648. <https://doi.org/10.1097/00006842-199211000-00004>
- Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology*, 69(1), 113–132. <https://doi.org/10.1016/j.biopsycho.2004.11.009>
- Levine, S., & Ursin, H. (1991). What is stress? In M.R. Brown, G.F. Koob & C. Rivier (Eds.), *Stress: Neurobiology and neuroendocrinology*. Marcel Dekker.
- Light, K. C., Kothandapani, R. V., & Allen, M. T. (1998). Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. *International Journal of Psychophysiology*, 28(2), 157–166. [https://doi.org/10.1016/S0167-8760\(97\)00093-7](https://doi.org/10.1016/S0167-8760(97)00093-7)
- Lim, J.-A., Yun, J.-Y., Choi, Y., Choi, S.-H., Kwon, Y., Lee, H. Y., & Jang, J. H. (2020). Sex-specific differences in severity of depressive symptoms, heart rate variability, and neurocognitive profiles of depressed young adults: Exploring characteristics for mild depression. *Frontiers in Psychiatry*, 11, 217. <https://doi.org/10.3389/fpsy.2020.00217>
- Liu, J. J. W., Ein, N., Peck, K., Huang, V., Pruessner, J. C., & Vickers, K. (2017). Sex differences in salivary cortisol reactivity to the Trier Social Stress Test (TSST): A meta-analysis. *Psychoneuroendocrinology*, 82, 26–37. <https://doi.org/10.1016/j.psyneuen.2017.04.007>
- Lovallo, W. (1975). The cold pressor test and autonomic function: A review and integration. *Psychophysiology*, 12(3), 268–282. <https://doi.org/10.1111/j.1469-8986.1975.tb01289.x>
- Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: omparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy*, 33(3), 335–343. [https://doi.org/10.1016/0005-7967\(94\)00075-U](https://doi.org/10.1016/0005-7967(94)00075-U)
- Lundberg, U., & Frankenhaeuser, M. (1980). Pituitary-adrenal and sympathetic-adrenal correlates of distress and effort. *Journal of Psychosomatic Research*, 24(3), 125–130. [https://doi.org/10.1016/0022-3999\(80\)90033-1](https://doi.org/10.1016/0022-3999(80)90033-1)
- McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology*, 583(2), 174–185. <https://doi.org/10.1016/j.ejphar.2007.11.071>
- McHugh, M. L. (2011). Multiple comparison analysis testing in ANOVA. *Biochimica Medica*, 21(3), 203–209. <https://doi.org/10.11613/bm.2011.029>
- Meyer, T., Smeets, T., Giesbrecht, T., Quaedflieg, C. W. E. M., & Merckelbach, H. (2013). Acute stress differentially affects spatial configuration learning in high and low cortisol-responding healthy adults. *European Journal of Psychotraumatology*, 4(1), 19854. <https://doi.org/10.3402/ejpt.v4i0.19854>
- Midway, S., Robertson, M., Flinn, S., & Kaller, M. (2020). Comparing multiple comparisons: Practical guidance for choosing the best multiple comparisons test. *PeerJ*, 8, e10387. <https://doi.org/10.7717/peerj.10387>
- Miller, R., & Kirschbaum, C. (2019). Cultures under stress: A cross-national meta-analysis of cortisol responses to the Trier Social Stress Test and their association with anxiety-related value orientations and internalizing mental disorders. *Psychoneuroendocrinology*, 105, 147–154. <https://doi.org/10.1016/j.psyneuen.2018.12.236>
- Mirete, M., Molina, S., Villada, C., Hidalgo, V., & Salvador, A. (2021). Subclinical social anxiety in healthy young adults: Cortisol and subjective anxiety in response to acute stress. *Anales de Psicología*, 37(3), 432–439. <https://doi.org/10.6018/analesps.483411>
- Moses, T. E. H., Gray, E., Mischel, N., & Greenwald, M. K. (2023). Effects of neuromodulation on cognitive and emotional responses to psychosocial stressors in healthy humans. *Neurobiology of Stress*, 22, 100515. <https://doi.org/10.1016/j.ynstr.2023.100515>
- Muñiz, J., & Bartram, D. (2007). Improving international tests and testing. *European Psychologist*, 12(3), 206–219. <https://doi.org/10.1027/1016-9040.12.3.206>
- Muñiz, J., Elosua, P., & Hambleton, R. K. (2013). Directrices para la traducción y adaptación de los tests: Segunda edición. *Psicothema*, 25.2, 151–157. <https://doi.org/10.7334/psicothema2013.24>
- Nash, D., & Heiss, J. (1967). Sources of anxiety in laboratory strangers. *The Sociological Quarterly*, 8(2), 215–221. <https://doi.org/10.1111/j.1533-8525.1967.tb01049.x>
- Nayak, S., Shiflett, S. C., Eshun, S., & Levine, F. M. (2000). Culture and gender effects in pain beliefs and the prediction of pain tolerance. *Cross-Cultural Research*, 34(2), 135–151. <https://doi.org/10.1177/106939710003400203>
- Nierop, A., Wirtz, P. H., Bratsikas, A., Zimmermann, R., & Ehlert, U. (2008). Stress-buffering effects of psychosocial resources on physiological and psychological stress response in pregnant women. *Biological Psychology*, 78(3), 261–268. <https://doi.org/10.1016/j.biopsycho.2008.03.012>
- Ortuño Sierra, J., García Velasco, L., Inchausti, F., Debbané, M., & Fonseca Pedrero, E. (2016). Nuevas aproximaciones en el estudio de las propiedades psicométricas del STAI. *Actas Españolas de Psiquiatría*, 44(3), 83–92.
- Ortuño-Sierra, J., Santarén-Rosell, M., de Albéniz, A. P., & Fonseca-Pedrero, E. (2015). Dimensional structure of the Spanish version of the Positive and Negative Affect Schedule (PANAS) in adolescents and young adults. *Psychological Assessment*, 27(3), e1–e9. <https://doi.org/10.1037/pas0000107>
- Phillips, A. C., Hunt, K., Der, G., & Carroll, D. (2011). Blunted cardiac reactions to acute psychological stress predict symptoms of depression five years later: Evidence from a large community study. *Psychophysiology*, 48(1), 142–148. <https://doi.org/10.1111/j.1469-8986.2010.01045.x>
- Popa, B., Guillet, L., & Mullet, E. (2014). Cultural differences in the appraisal of stress. *Psicológica*, 35(3), 745–760.
- Rubeking, B. (2017). Boring is bad: Effects of emotional content and multitasking on enjoyment and memory. *Computers in Human Behavior*, 72, 488–495. <https://doi.org/10.1016/j.chb.2017.03.015>
- Ruiz, F. J., Martín, M. B. G., Falcón, J. C. S., & González, P. O. (2017). The hierarchical factor structure of the Spanish version of Depression Anxiety and Stress Scale-21. *International Journal of Psychology and Psychological Therapy*, 17(1), 97–105.
- Sandin, B., Chorot, P., Lostao, L., Joiner, T., Santed, M., & Valiente, R. (1999). The PANAS scales of positive and negative affect: Factor analytic validation and cross-cultural convergence. *Psicothema*, 11(1), 37–51.
- Santos-Carrasco, D., & De la Casa, L. G. (2024). Stressing out! Effects of acute stress on prepulse inhibition and working memory. *Psychophysiology*, 61(9). <https://doi.org/10.1111/psyp.14599>
- Schwabe, L., Haddad, L., & Schachinger, H. (2008). HPA axis activation by a socially evaluated cold-pressor test. *Psychoneuroendocrinology*, 33(6), 890–895. <https://doi.org/10.1016/j.psyneuen.2008.03.001>
- Schwabe, L., & Wolf, O. T. (2009). The context counts: Congruent learning and testing environments prevent memory retrieval impairment following stress. *Cognitive, Affective, & Behavioral Neuroscience*, 9(3), 229–236. <https://doi.org/10.3758/CABN.9.3.229>
- Shields, G. S. (2020). Stress and cognition: A user’s guide to designing and interpreting studies. *Psychoneuroendocrinology*, 112, 104475. <https://doi.org/10.1016/j.psyneuen.2019.104475>
- Shilton, A. L., Laycock, R., & Crewther, S. G. (2017). The Maastricht Acute Stress Test (MAST): Physiological and subjective responses in anticipation, and post-stress. *Frontiers in psychology*, 8, 567. <https://doi.org/10.3389/fpsyg.2017.00567>
- Skoluda, N., Strahler, J., Schlotz, W., Niederberger, L., Marques, S., Fischer, S., Thoma, M. V., Spoerri, C., Ehlert, U., & Nater, U. M. (2015). Intra-individual psychological and physiological responses to acute laboratory stressors of different intensity. *Psychoneuroendocrinology*, 51, 227–236. <https://doi.org/10.1016/j.psyneuen.2014.10.002>

- Smeets, T.** (2011). Acute stress impairs memory retrieval independent of time of day. *Psychoneuroendocrinology*, *36*(4), 495–501. <https://doi.org/10.1016/j.psyneuen.2010.08.001>
- Smeets, T., Cornelisse, S., Quaedflieg, C. W. E. M., Meyer, T., Jelicic, M., & Merckelbach, H.** (2012). Introducing the Maastricht Acute Stress Test (MAST): A quick and non-invasive approach to elicit robust autonomic and glucocorticoid stress responses. *Psychoneuroendocrinology*, *37*(12), 1998–2008. <https://doi.org/10.1016/j.psyneuen.2012.04.012>
- Soto, J. A., Roberts, N. A., Pole, N., Levenson, R. W., Bursleson, M. H., King, A. R., & Breland-Noble, A.** (2012). Elevated baseline anxiety among African Americans in laboratory research settings. *Journal of Psychophysiology*, *26*(3), 105–115. <https://doi.org/10.1027/0269-8803/a000073>
- Souza-Talarico, J. N., Plusquellec, P., Lupien, S. J., Fiocco, A., & Suchecki, D.** (2014). Cross-country differences in basal and stress-induced cortisol secretion in older adults. *PLoS One*, *9*(8), e105968. <https://doi.org/10.1371/journal.pone.0105968>
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E.** (1982). *Cuestionario de ansiedad estado-rasgo*. TEA Ediciones.
- Stephens, A., Willemsen, G., Kunz-Ebrecht, S. R., & Owen, N.** (2003). Socioeconomic status and hemodynamic recovery from mental stress. *Psychophysiology*, *40*(2), 184–191. <https://doi.org/10.1111/1469-8986.00020>
- Trofimova, I.** (2013). A study of the dynamics of sex differences in adulthood. *International Journal of Psychology*, *48*(6), 1230–1236. <https://doi.org/10.1080/00207594.2012.756981>
- Ulrich-Lai, Y. M., & Herman, J. P.** (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience*, *10*(6), 397–409. <https://doi.org/10.1038/nrn2647>
- Urizar, G. G., Yim, I. S., Kofman, Y. B., Tilka, N., Miller, K., Freche, R., & Johnson, A.** (2019). Ethnic differences in stress-induced cortisol responses: Increased risk for depression during pregnancy. *Biological Psychology*, *147*, 107630. <https://doi.org/10.1016/j.biopsycho.2018.12.005>
- Watson, D., Clark, L. A., & Tellegen, A.** (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070. <https://doi.org/10.1037/0022-3514.54.6.1063>