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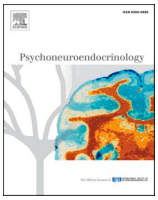

The influence of glucose administration on stress reactivity and long-term memory in adult men and women

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ABSTRACT

Stress and the associated cortisol release have profound effects on long-term memory (LTM). While glucose increases the cortisol stress response and exhibits memory enhancing effects in non-stressful situations, the interaction of glucose and stress on LTM has rarely been studied. The aim of this study was to investigate whether the glucose-related amplification of the cortisol stress response would enhance LTM formation. Overall, $N = 62$ healthy, fasted adults (age $M = 23.13$, $SD = 3.02$; 54.84 % female) participated. They consumed a drink containing water or glucose and underwent a non-stressful control task or the Trier Social Stress Test with Objects, during which panel members interact with certain objects (central) while leaving others untouched (peripheral). At the estimated cortisol peak, they encoded a wordlist. On the next day, they retrieved the objects and the words. We repeatedly assessed subjective stress, salivary cortisol and blood glucose concentrations and recorded an electrocardiogram. Glucose increased blood glucose concentrations, and the stressor led to a significant increase in cortisol as compared with the control task. Changes in cortisol were more pronounced in the glucose as compared with the water groups. Heart rate was elevated in the glucose as compared with the water groups during the recovery. Central objects were better remembered than peripheral objects when encoded during stress. Additionally, emotional words were remembered better as compared with neutral words. These effects were not modulated by glucose. These findings suggest that emotional information is remembered better than neutral information independent of stress and glucose intake. Stress enhances LTM of stressor-relevant information and glucose intake increases the cortisol stress response. However, these factors do not appear to interact. Glucose availability may thus play a less decisive role when memorizing a stressful episode.

1. Introduction

The stress response, mediated by the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis, serves to provide energy, closely linking stress to energy metabolism (Ulrich-Lai and Ryan, 2014). Activation of the SNS and HPA axis triggers the release of noradrenaline, adrenaline, and cortisol, which stimulate the release of glucose from energy storages to prepare the organism for *fight or flight* (Sapolsky, 2000; Taborsky et al., 2021; Ulrich-Lai and Herman, 2009). Stress thereby has a measurable impact on energy metabolism and acutely increases blood glucose concentration (Fellinger et al., 2025).

In turn, the stress response seems to depend on the availability of glucose. In rodents, cortisol stress reactivity is increased in times of food consumption, and decreased in times of food restriction (Choi et al., 1996). In humans, glucose intake prior to a stressor enhances the cortisol stress response (Bentele et al., 2021; Gonzalez-Bono et al., 2002; Kirschbaum et al., 1997; Meier et al., 2021; von Dawans et al., 2020; Zänkert et al., 2020), but the SNS stress response remains unaffected (Bentele et al., 2021; Kirschbaum et al., 1997; von Dawans et al., 2020). While blood glucose concentration and cortisol stress reactivity were positively associated in some studies (Gonzalez-Bono et al., 2002; Kirschbaum et al., 1997), not all could replicate this effect (Meier et al., 2021; Rüttgens and Wolf, 2022; von Dawans et al., 2020). Further, the cortisol amplification seems to be more pronounced in settings evoking psychosocial as compared with physiological stress (Rüttgens and Wolf, 2022; von Dawans et al., 2020). Findings in rodent models suggested

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that the effect of glucose on the stress response may be mediated by its activation of the ventromedial nucleus which in turn activates the paraventricular nucleus (PVN; Choi et al., 1996) – a region centrally involved in modulating HPA axis and SNS stress responses through its projections to the median eminence, the brainstem and the spinal cord (Ulrich-Lai and Herman, 2009; Ulrich-Lai and Ryan, 2014). While the exact mechanisms of increased cortisol stress reactivity following glucose uptake still need to be elucidated, another unanswered question is whether the amplified cortisol stress response after glucose load has any cognitive or behavioral effects.

Cortisol can exert profound effects on behavior and cognition[1] (Lupien et al., 2009). These effects are mediated by an interplay of the high-affinity mineralocorticoid receptor (MR) and the low-affinity glucocorticoid receptor (GR) (Rimmele et al., 2013; Wolf, 2019). While MRs are for example present in the hippocampus, GRs are expressed more ubiquitously, in particular in the PVN, subfields of the hippocampus and the amygdala (Joëls et al., 2012), which are integral for stress and long-term memory (LTM; Roesler et al., 2021; Roozendaal and McGaugh, 2011). Both, rapid nongenomic effects as well as slower genomic effects of cortisol have been related to memory outcomes in previous animal and human studies (Joëls et al., 2012).

Effects of stress on memory are multifaceted and dependent on different modulating factors. Most critically, the timing of the stressor, i.e. whether stress occurred before or during encoding, consolidation or retrieval, shapes effects of stress on memory (Goldfarb et al., 2019; Roozendaal et al., 2009; Schwabe and Wolf, 2013; Wolf, 2019). While stress long before encoding and retrieval impairs memory, post-encoding stress can enhance memory (Schwabe and Wolf, 2013; Shields et al., 2017). However, these effects are modulated by the centrality of the learned information, i.e. whether the content was relevant in the stressful situation or not (Schwabe et al., 2012; Wolf, 2019). When the learning content is closely tied to the stressor, stress enhances memory of this information as opposed to stress-irrelevant information (Schwabe et al., 2012; Wolf, 2019). For example, in stressful interview settings, objects that are interacted with (central objects) are better remembered than objects placed in the background (peripheral objects) and central objects experienced in a control setting (Wiemers, Sauvage, et al., 2013; Wolf, 2019). Conversely, encoding stress-irrelevant information after stress, at the peak of the cortisol stress response, can impair memory (Schwabe et al., 2012; Shields et al., 2017). This is proposed to support memory formation of the stress-relevant information, while suppressing memory formation of stress-irrelevant information (cf. memory formation and memory storage mode; Schwabe et al., 2012b) to support optimal behavior in the future. Lastly, the emotional valence of the learning content, i.e. its potential to evoke arousal, is essential (Shields et al., 2017). Evidence suggests that stress can facilitate memory of emotional (negative and positive) as compared with neutral content (Merz, 2017; Merz et al., 2019; Payne et al., 2007). Taken together, the effects of stress and cortisol on LTM depend on the timing of the stressor as well as the centrality and the emotionality of the encoded information (Klier and Buratto, 2020; Roozendaal et al., 2009; Schwabe et al., 2012; Schwabe and Wolf, 2013; Shields et al., 2017).

If glucose increases cortisol reactivity, one could expect that the effects of cortisol on LTM would indirectly be enhanced. However, the effects of stress and cortisol on memory are not linear but follow an inverted U-shape (Rimmele et al., 2013; Roozendaal et al., 2009; Schilling et al., 2013). For example, memory retrieval is optimal when cortisol levels during retrieval are intermediate, i.e., when MR activation is predominant and GR activation is low. In contrast, very low cortisol levels and a resulting lack of MR activation, as well as very high cortisol levels following stressor exposure and an associated predominance in GR activation, are associated with retrieval impairments (Rimmele et al., 2013; Roozendaal et al., 2009; Schilling et al., 2013). Taken this into account, the prediction of how glucose may modulate effects of stress on memory becomes inherently more complex.

Moreover, glucose itself has been reported to affect cognition in non-stressful contexts. This effect has been described as 'glucose memory facilitation effect' (Benton and Owens, 1993; Foster et al., 1998; Smith et al., 2011). Thereby, glucose can boost hippocampus-dependent memory (Smith et al., 2011), increase LTM retrieval when consumed prior and post encoding (Meikle et al., 2005), and enhance recognition memory (Smith et al., 2011). These effects seem to be driven by direct effects of glucose on the hippocampus. For example, a disruption of hippocampal glucose uptake via the blockade of insulin-dependent glucose transporters impairs LTM in rats (Pearson-Leary and McNay, 2016). While these effects were observed in non-stressful situations, it is unclear whether the facilitating effect of glucose may interact with the effects of cortisol on LTM in stressful situations.

Even though both glucose and cortisol have been shown to influence LTM, we are aware of only one study that investigated the interaction of glucose and stress, respectively cortisol, on memory performance to date (Rüttgens and Wolf, 2022). In this study, fasted participants either consumed a drink containing glucose or stevia (a non-nutritive sweetener that acted as placebo for the glucose drink) before being subject to the socially evaluated cold-pressor test (SECPT; Schwabe et al., 2008). Participants encoded a wordlist prior to stress and retrieved the words directly after. Furthermore, they performed a working memory task at the estimated cortisol peak. Stress impaired working memory performance, but it did not affect word recall. Glucose neither affected the cortisol stress response, nor performance in the memory tasks (Rüttgens and Wolf, 2022). This suggests that glucose may not affect stress after encoding, yet potential effects of glucose on stress *during* and *before* encoding remain unclear. Also, as the study did not incorporate a nightly consolidation phase, effects of stress on retrieval and consolidation were intertwined in this design, warranting further investigation. While the experiment was well controlled and used established paradigms, the cortisol stress response to the SECPT might not have been strong enough to evoke the hypothesized effects. Stressors that rely on speech tasks in combination with social evaluation, such as the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), are known to induce stronger elevations in cortisol as compared with merely physical stressors (Dickerson and Kemeny, 2004; Skoluda et al., 2015). Additionally, using sweetener as control might have affected the results, as sweetener has previously been found to affect cortisol reactivity in women (Meier et al., 2021). Considering this, we felt that a re-evaluation of the effects of glucose on memory under stress was warranted in a slightly adjusted design, incorporating social-evaluative stress during and before encoding, a nightly consolidation phase, and a water control group.

The aim of the current, exploratory project was therefore to test the effect of glucose on cortisol stress reactivity and LTM. We employed two memory tasks, to test effects on encoding *during the stressful situation* and *after the stressful situation*. To do so, fasted participants either consumed a *glucose* drink or *water* and performed an adapted version of the TSST or a friendly control condition, during which target objects are placed in the room, some of which are interacted with (central objects) whereas others are placed in the background (peripheral objects; Wiemers, Sauvage, et al., 2013). At the estimated cortisol peak, participants encoded a list of neutral, positive and negative words that was unrelated to the stressor. On the subsequent day, they were asked to recognize the objects present during the stressor and to retrieve the wordlist.

Based on previous psychosocial stress studies (Bentele et al., 2021; Gonzalez-Bono et al., 2002; Kirschbaum et al., 1993; Meier et al., 2021; von Dawans et al., 2020; Zänkert et al., 2020), we hypothesized that glucose enhances the cortisol stress response to the TSST relative to water. As results regarding an association between blood glucose and cortisol reactivity were heterogeneous in the past, we further examined

[1] As glucose intake primarily affected stress reactivity of the HPA axis as opposed to the SNS, we focus on effects of cortisol on cognition in the following, even though noradrenaline and adrenaline exhibit cognitive effects as well (Roesler and Schröder, 2011; Sara, 2009).

whether blood glucose concentrations were positively associated with cortisol reactivity on a continuous scale. Furthermore, we hypothesized that *stress during encoding* – mediated by the rising cortisol levels in interaction with norepinephrine – enhances LTM of objects as compared with a friendly control condition (Wolf, 2009), especially if the objects were central to the stressor (Wolf, 2019). We expected this effect to be stronger in the *glucose* group exposed to *stress* as compared with the *water* group, as glucose consumption before encoding enhances memory in non-stressful situations (Meikle et al., 2005). Lastly, we hypothesized that high cortisol concentrations during encoding reduces memory formation of words (Schwabe et al., 2012; Wolf, 2009). As a high glucose concentration during encoding enhances memory (Meikle et al., 2005), we expected that the effect was stronger in the *water* group as compared with the *glucose* group. As such, we expected that participants that encoded the wordlist under stress after consuming water would retrieve the least number of words. As evidence suggests that emotional content is remembered better than neutral content (Merz, 2017; Merz et al., 2019; Payne et al., 2007), we further tested the effect of valence in our models.

2. Methods

2.1. Participants

Healthy participants aged between 18 and 40 years were recruited via flyers posted at the University of Konstanz and the city of Konstanz as well as via the university's participant pool management software (SONA Systems). Eligibility for the study was screened in an online survey to control for the influence of various factors known to impact SNS or HPA axis activity (Quintana et al., 2016; Strahler et al., 2017). We excluded participants fulfilling any of the following criteria: (1) body mass index (BMI) below 18.5 or above 30 kg/m^2, (2) smokers (> 5 cigarettes per day), (3) disturbed circadian rhythm due to night shift or jetlag, (4) mental or somatic disorder(s), (5) medication intake, (6) alcohol or drug abuse, (7) clinically relevant symptoms of depression (based on Beck's Depression Inventory II sum score >18; Kühner et al., 2007). In addition, female participants were excluded if they reported an irregular, very short (<21 days) or long (>37 days) menstrual cycle, current pregnancy, being in the menopause or using hormonal contraceptives (Schmalenberger et al., 2021). Overall, $N = 62$ eligible men and women (mean age = 23.13; $SD = 3.02$, range = 18 – 37; 54.84 % female) participated in the study.

2.2. Design and experimental procedure

The study comprised a 2×2 design with the between-subjects factors *drink* (2 levels: water, glucose) and *stress* (2 levels: control, stress) and participants were assigned randomly to the four study groups. The resulting group sizes were the following: (1) water, control ($n = 14$), (2) glucose, control ($n = 16$), (3) water, stress ($n = 18$), (4) glucose, stress ($n = 14$). The experiment was carried out in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the University of Konstanz (IRB 45/2022). Participants received 40€ or course credits for participation.

The experimental sessions were performed on two consecutive days in the afternoon between 1:30 and 5:00 PM to control for the circadian rhythm (Miller et al., 2016). On average, sessions started at 3:25 PM ($SD = 68$ min), with $n = 29$ participants starting at 2:30 PM, $n = 12$ starting at 3:00 PM and $n = 21$ starting at 5:00 PM. To standardize the time between visits, each participant's sessions were always scheduled at the same time of day. On day one, participants were invited to the laboratory after a 5 h fast, as postprandial glucose concentrations may take up to 5 h to return to baseline (Moebus et al., 2011). After they were equipped with an electrocardiogram (MindWare Mobile device, Mindware Technologies, Gahanna, OH), a physiological baseline was recorded for 5 min. Then, they consumed a drink (glucose or water, see 2.3.1.). After an absorbance period, during which participants completed questionnaires, they either underwent the TSST (stress condition) or the friendly TSST (friendly control condition; Wiemers, Sauvage, et al., 2013; Wiemers, Schoofs, et al., 2013). Later, participants were asked to memorize a list of words. The session ended with a recovery period during which participants sat quietly for 5 min to obtain a physiological recording at rest and afterwards filled out questionnaires.

On the subsequent day, participants were invited to the laboratory to perform the memory retrieval tests. After an introduction, participants were again equipped with an electrocardiogram, and a physiological baseline was recorded. Then, they performed an object recognition test, and a free word recall test, both of which were interrupted by a questionnaire. The session ended with a paced breathing exercise (cf. Meier et al., 2025), results of which will be reported elsewhere. At the end of day two, participants were debriefed and received compensation. The procedure is depicted in Fig. 1.

2.3. Materials

2.3.1. Glucose and water drink

Participants consumed either a drink containing 75 g of glucose dissolved in 200 mL of non-sparkling mineral water (*glucose*), or 200 mL of non-sparkling mineral water (*water*). Similar amounts of glucose have been used in previous studies investigating the effect of glucose on the cortisol stress response (Gonzalez-Bono et al., 2002; Rüttgens and Wolf, 2022; von Dawans et al., 2020; Zänkert et al., 2020). Drinks were prepared by a third person not involved in testing and cooled to 4°C. Participant and experimenter were blind to the assigned drink content prior to the session.

2.3.2. Stress and friendly control condition

The Trier Social Stress Test (TSST; Kirschbaum et al., 1993) is a widely used standardized protocol that reliably induces psychosocial stress with marked increases in vegetative and endocrine stress markers (Goodman et al., 2017). The standard TSST involves a 10-minute anticipation period in which participants can prepare for a mock job interview. The preparation is followed by a videotaped 5-minute free speech and a 5-minute mental arithmetic task that are performed while standing in front of a two-person committee wearing white lab coats.

We used an adapted version of the TSST that was described in previous publications (Bierbrauer et al., 2021; Wiemers, Sauvage, et al., 2013) and allows to specifically test the memory of the stressful situation (TSST with Objects). The TSST with Objects involves a 5-minute anticipation phase and an 8-minute speech part, without incorporating a mental arithmetic part (Bierbrauer et al., 2021; Wiemers, Sauvage, et al., 2013). During the TSST, 24 objects (e.g., stapler, ruler) were placed in the TSST room, half of which were used by the committee in a standardized manner and order (central objects). The other half was not used but placed in direct proximity of the committee (peripheral objects). As described in detail before (Bierbrauer et al., 2021), we used two parallel versions of the task that differed in the subset of objects that the committee manipulated during the TSST. An overview of the setup and the objects can be retrieved from the supplemental material. This version of the TSST has successfully induced psychosocial stress in previous studies (Bierbrauer et al., 2021; Herten et al., 2017; Wiemers, Sauvage, et al., 2013), albeit cortisol stress responses might be lower as compared with the original protocol.

Participants in the control condition underwent a friendly control condition (friendly TSST), in which the committee was following the same procedure as in the modified TSST version described above (Wiemers, Schoofs, et al., 2013). As such, the same objects were placed in the room and the committee interacted with the objects but reacted friendly and supportive to statements of the participants. The friendly TSST was not videotaped and no white coats were worn. Participant and experimenter were blind to the assigned condition prior to the session.

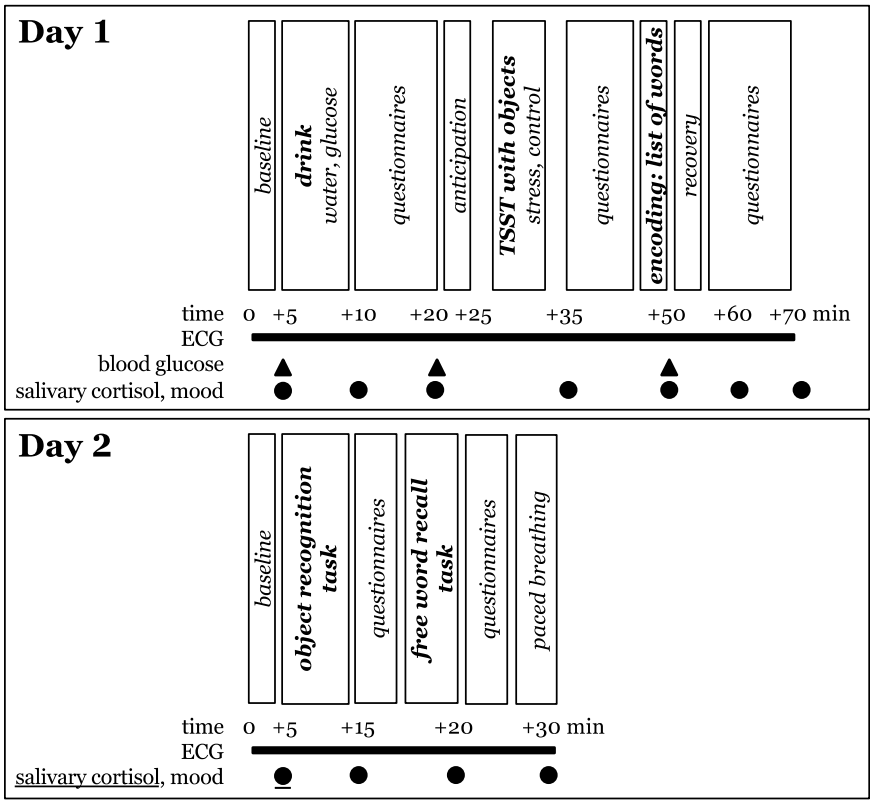

Fig. 1. Experimental procedure. ECG = electrocardiogram. TSST = Trier Social Stress Test.

2.3.3. Long-term memory tasks

The study comprised two separate LTM tasks. First, as described in 2.3.2, 24 objects were placed in the TSST room during participants' speech performance. While participants were not notified about the purpose of the objects on day one, their memory of the objects was tested on the subsequent day. This object recognition task included pictures of the 24 objects present in the TSST room (e.g., stapler), 24 'difficult distractors' that were similar to the objects presented (e.g. stapler in a different color), and 24 'easy distractors' that were not present in the room (Bierbrauer et al., 2021). On day two, the objects were presented on a grey background on an Apple iPad Pro 11" and participants were asked to indicate whether they had seen the object on the previous day (yes, no) and how confident they were in their answer (1 = very confident that they saw the object on the previous day, 6 = very confident that they did not see the object on the previous day). There was no time limit for the response and participants took approximately 10 min to complete the task. For the statistical analyses, we computed participant's *hit rate (HR), false alarm rate (FAR)* and *recognition performance (Pr)* for central and peripheral objects. *HR* was defined as the conditional probability of indicating that one had seen the object on the previous day ('yes'), when the object was indeed present, P(yes/present), whereas *FA* represented the conditional probability of responding that one had seen the object on the previous day ('yes'), when the object was indeed *not* present, P(yes/not present) (Snodgrass and Corwin, 1988). *Pr* was indexed by subtracting *FAR* from *HR*.

Second, participants were asked to memorize a list of 30 words (Merz et al., 2019; Rüttgens and Wolf, 2022) at the estimated peak of the cortisol stress response approximately 20 min after stressor onset (Miller et al., 2013). This list contained 10 neutral (e.g., survey, function, document), 10 negative (e.g., abuse, accident, murder), and 10 positive words (e.g., kiss, beauty, harmony). As performed in previous studies (Merz et al., 2019), we used two parallel versions of the word list and participants had two minutes to memorize the words. Word frequency, length, or semantic cohesion did not differ between neutral, negative and positive words (Kuhlmann et al., 2005; Merz et al., 2019). Participants were told that their memory of the words will be tested on the subsequent day, on which they performed a free retrieval of the list for 5 min. We counted the *number of correctly remembered words* per emotional valence (neutral, negative, positive) for later statistical analysis (theoretical range: 0 – 10 for each valence).

2.4. Measures

2.4.1. Blood glucose concentrations

At three scheduled timepoints on day one (cf. Fig. 1), blood glucose concentration (in dg/mL) was assessed from capillary blood of the fingertip using disposable lancets (Roche Diabetes Care, Mannheim, Germany) and a glucometer (GlucoMen areo, A. Menarini diagnostics, Berlin, Germany). Due to the presence of outliers that exceeded the mean of the sample by more than 3 *SD*, we winsorized blood glucose concentrations. Further, due to a violation of normality as indicated by a significant Shapiro Wilk test, we log transformed blood glucose concentrations. For correlational analyses, we computed a change score by subtracting the first from the individual's maximum winsorized blood glucose value (*blood glucose reactivity*). Additionally, we computed the area under the curve with respect to increase (AUCi; Pruessner et al., 2003). While transformed values were used in the statistical analyses, raw values are presented in the descriptive analyses, tables and figures.

2.4.2. Salivary cortisol

At seven scheduled timepoints on day one, and once as a baseline on day two (cf. Fig. 1), saliva was sampled with Cortisol Salivettes® (Nümbrecht, Germany). Samples were stored at −20°C until further analyses in the biochemical laboratory of the University of Konstanz. After thawing, samples were centrifuged at 2500 rpm for 10 min. To determine salivary cortisol concentrations (in nmol/L), samples were analyzed in duplicates using the commercially available Cortisol Saliva ELISA (RE52611, IBL international GmbH, Hamburg, Germany). The

intra- and inter-assay coefficients were 10.61 % and 20.12 % respectively. Four samples did not contain enough saliva to determine cortisol; one sample only contained enough saliva for a single determination. A subset of samples with high inter-assay CVs (> 20 %) in the first duplicate determination were reassessed and the more plausible value from the first determination was kept as single determination to avoid a bias from alterations in assay batch, storage and rethawing cycles. Due to the presence of outliers that exceeded the mean of the sample by more than 3 *SD*, we winsorized salivary cortisol concentrations. Moreover, due to a violation of normality as indicated by a significant Shapiro Wilk test, we log transformed the values for the statistical analysis. For correlational and descriptive analyses, we computed a *cortisol reactivity* change score by subtracting the winsorized baseline cortisol value before the TSST (+20 min) from the winsorized individual cortisol peak determined as the maximum value of the post-TSST samples (+50, +60, +70 min), to decide whether individuals were cortisol responders (increase > 1.5 nmol/L) or not (Miller et al., 2013). Additionally, we computed the AUCi (Pruessner et al., 2003). While transformed values were used in the statistical analyses, raw values are presented in the descriptive analyses, tables and figures.

2.4.3. Heart rate

To assess the autonomic stress response, we recorded an electrocardiogram (ECG) using a portable MindWare Mobile device (Mindware Technologies, Gahanna, OH) with a sampling rate of 500 Hz. The electrode setup included seven spot electrodes (ECG electrodes ASF50, Asmuth Gmbh, Minden, Germany) and combined the standard Lead II with the standard tetrapolar system (Sherwood et al., 1990). We defined four events of interest for the analysis: (1) the 5-min baseline, (2) the 5-min anticipation of the TSST, (3) the 8-min TSST, (4) the 5-min recovery period. Heart rate was analyzed in 1 min epochs (windowing) and averaged across each event of interest. This approach closely matches averages of longer recordings, while allowing to exclude single minutes of noisy data if needed (Quigley et al., 2024). The ECG signal was analyzed using MindWare Heart Rate Variability Analysis Software version 3.2.3. After inspection, artifacts were removed, and ectopic beats were corrected manually. Additionally, we computed *heart rate reactivity* as the change from baseline to TSST and the AUCi (Pruessner et al., 2003). While transformed values were used in the statistical analyses, baseline corrected values are presented in the figures.

2.4.4. Questionnaires and potential covariates

We assessed demographic variables such as self-reported *sex* assigned at birth, age, height and weight (for the calculation of body mass index [BMI]) using a questionnaire on the platform Qualtrics. Subjective stress responses were assessed using the affect grid (Russell et al., 1989). The affect grid is a single item scale that assesses current mood on two dimensions, pleasure and arousal. The scores on each dimension range from 1 (low arousal, and low pleasure) to 9 (high arousal, and high pleasure). As in previous work (Meier et al., 2020, 2021), we multiplied arousal and the inverted pleasure scores to obtain a single-item score, with higher scores indicating higher levels of subjective stress (theoretical range 1–81).

2.5. Statistical analysis

Statistical analysis was performed in R version 4.4.0 (2024–04–24) with RStudio version 2024.9.0.375 utilizing the packages *tidyverse* (Wickham et al., 2019), *reshape2* (Wickham, 2007), *nlme* (Pinheiro et al., 2018), *table1* (Rich, 2023), *sjPlot* (Lüdecke, 2024), and *performance* (Lüdecke et al., 2021).

To determine potential covariates, we first tested whether the four experimental groups differed in respect to demographics and state variables that have been shown or are likely to be associated with stress reactivity (Strahler et al., 2017). These variables included sex, age, BMI and fasting duration.

Using nonlinear mixed models, we then tested whether the *glucose* drink significantly increased blood glucose concentrations as compared with *water*. Using the same approach, we tested whether the *stress* condition led to an increase in cortisol as compared with the *friendly control* condition and whether the cortisol response was more pronounced in the *glucose* as compared with the *water* group exposed to stress. The same analysis was conducted with *subjective stress* and *heart rate* as an outcome.

As correlational analyses have traditionally been conducted in previous research on this topic (Kirschbaum et al., 1997; Meier et al., 2021; von Dawans et al., 2020), we computed a Pearson's correlation coefficient between *cortisol reactivity* and *blood glucose reactivity* as well as between the cortisol, heart rate and glucose AUCi.

Next, we tested whether LTM for peripheral and central objects and for negative, positive and neutral words was influenced by the experimental manipulations. To do so, we used linear mixed models to evaluate whether *recognition performance* of objects (*Pr*) was predicted by *object type* (peripheral, central), *stress* (control, stress) and *drink* (water, glucose) and their respective interaction terms. Similarly, we used a linear mixed model to evaluate whether retrieval of words was predicted by *valence* (within-subjects factor: neutral, positive, negative), *stress* (control, stress) and *drink* (water, glucose) and their respective interaction terms.

Lastly, we tested whether *cortisol reactivity*, *glucose reactivity* and their interaction were significant predictors of the memory outcomes (*Pr* for central and peripheral objects and the *number of correctly remembered words* per emotional valence) in multiple regression analyses.

3. Results

3.1. Descriptives and preliminary analyses

A demographic description of the sample and the results of the group comparisons can be found in Table 1. Participants did not significantly differ in relevant demographic or baseline characteristics (i.e., age, sex, BMI, glucose baseline, cortisol baseline on day 1 and 2), except for heart rate baseline of day 1, which was significantly higher in the *stress glucose* condition as compared with the *control water* and *control sugar* condition (see Table 1). Cortisol baseline did not significantly differ on the two consecutive testing days, $t(61) = 1.25$, $p = .217$, $d = 0.16$.

On average, participants fasted 6.42 h ($SD = 2.9$) before entering the session and exhibited a mean blood glucose level of 103.41 mg/dL ($SD = 11.43$). The experimental groups differed significantly regarding the average fasting duration, $F(3, 58) = 3.77$, $p = .015$, $\text{eta}^2_{\text{part}} = .16$, with the friendly-control water group having fasted significantly longer than the stress water group (Bonferroni corrected t-test $p = .025$). As the inclusion of fasting duration as a covariate did not change the interpretation of the statistical models reported below (cf. supplemental material), we report the models without covariates in the main text.

Participants consuming water liked the drink significantly more, rated the taste as significantly less sweet and had a higher desire to drink more as compared with participants consuming glucose (see Table 1).

The two versions of the word list, $Chi^2(3) = 4.08$, $p = .253$, and the two TSST versions, $Chi^2(3) = 0.63$, $p = .890$, were evenly distributed across the experimental groups. Word recall performance did not differ between the two word lists used for positive, $F(1, 59) = 0.77$, $p = .383$, $\text{eta}^2_{\text{part}} = .01$, negative, $F(1, 59) = 0.35$, $p = .556$, $\text{eta}^2_{\text{part}} < .01$, and neutral words, $F(1, 59) = 1.06$, $p = .308$, $\text{eta}^2_{\text{part}} = .02$.

3.2. Effect of drink on blood glucose concentrations

The nonlinear mixed model in which we predicted blood glucose concentrations included 186 observations nested in 62 participants. We included a random intercept, random slope, a linear, quadratic and cubic effect of *time* as well as the main and interaction effects of *drink* and *stress*.

Table 1
Description of the sample.

	control water (N = 14)	control sugar (N = 16)	stress water (N = 18)	stress sugar (N = 14)	Test statistics
Age (in years)	23.6 (4.31) [range: 19–37]	22.8 (2.01) [range: 20–27]	23.1 (2.62) [range: 18–31]	23.1 (3.18) [range: 18–37]	$F(3, 58) = 0.16$, $p = .925$, $eta^2_{part} < .01$
Sex (female/male)[f]	7/7	8/8	11/7	8/6	$Chi^2(3) = 0.60$, $p = .897$, *Cramer's V* = 0.10
BMI (in kg/m^2)	23.6 (2.27)	22.6 (3.25)	21.7 (2.01)	22.5 (3.00)	$F(3, 58) = 1.29$, $p = .287$, $eta^2_{part} = .06$
Glucose baseline	104.0 (9.24)[a]	107.0 (12.5)	104.0 (8.55)	99.6 (15.0)[b]	$F(3, 55) = 0.90$, $p = .446$, $eta^2_{part} = .04$
Cortisol baseline Day 1 (nmol/L)	5.36 (2.90)	4.05 (2.75)	4.72 (5.41)	5.29 (3.96)	$F(3, 58) = 0.35$, $p = .788$, $eta^2_{part} = .02$
Cortisol baseline Day 2 (nmol/L)	5.08 (3.42)	4.37 (3.04)	3.83 (2.07)	3.98 (3.28)	$F(3, 58) = 0.54$, $p = .657$, $eta^2_{part} = .03$
Heart rate baseline Day 1	71.1 (8.59)	70.5 (9.49)	73.5 (9.32)	81.1 (10.5)	$F(3, 55) = 3.37$, $p = .025$, $eta^2_{part} = .16$
Drink rating: taste	71.4 (19.6)	36.3 (29.2)[a]	73.4 (18.5)	16.7 (16.7)	$F(1, 58) = 65.04$, $p < .001$, $eta^2_{part} = .53$
Drink rating: sweetness	5.30 (9.79)[c]	86.6 (24.3)	1.80 (3.19)[d]	88.4 (16.5)	$F(1, 53) = 380.36$, $p < .001$, $eta^2_{part} = .88$
Drink rating: desire	79.9 (23.9)	24.9 (21.3)[a]	80.5 (21.6)	11.2 (14.2)	$F(1, 58) = 132.87$, $p < .001$, $eta^2_{part} = .70$
Fasting duration	8.11 (4.56)	5.65 (0.91)	5.21 (0.51)	7.17 (3.43)[b]	$F(3, 57) = 3.68$, $p = .017$, $eta^2_{part} = .16$
Cortisol response in nmol/L [responder rate in %]	-0.10 (1.97) [14 %]	0.91 (1.66) [33 %]	2.21 (4.38) [39 %]	4.51 (6.57) [71 %]	$Chi^2(3) = 10.21$, $p = .017$

Notes. For continuous variables, mean (*SD*) is reported and a one-way ANOVA was calculated to test whether the four experimental groups differed in respect to the listed variable. For count variables, Pearson's Chi-squared test was calculated.
[a] $n = 2$ missing, [b] $n = 1$ missing, [c] $n = 4$ missing, [d] $n = 3$ missing, [f] self-reported sex assigned at birth.

The results indicated a significant main effect of *drink*, $b = .39$, $SE = .04$, $p < .001$, and a significant *time* x *drink* interaction effect, $b = 2.64$, $SE = .31$, $p < .001$, as well as a significant *time*^{2} x *drink* interaction effect, $b = -2.34$, $SE = .31$, $p < .001$. Bonferroni corrected post-hoc t-tests confirmed that the glucose drink led to a significant increase in blood glucose concentrations, while water did not (see Fig. 2). Blood glucose concentrations did not differ at baseline, $p > .05$, $d = 0.07$, but were significantly higher in the glucose groups as opposed to the water groups at timepoints + 20, $p < .001$, $d = -3.47$, and + 50 min, $p < .001$, $d = -3.85$.

3.3. Effect of stress and glucose on subjective stress

The nonlinear mixed model that predicted subjective stress included 404 observations nested in 59 participants. We included a random intercept, a linear, and quadratic effect of *time* as well as the main and interaction effects of *drink* and *stress*. We found a significant *time* x *stress*, $b = 56.11$, $SE = 23.83$, $p = .019$, and a *time*^{2} x *stress* interaction effect, $b = -91.16$, $SE = 23.61$, $p < .001$. Other main or interaction effects did not reach the level of significance (for detailed results see supplementary material).

Post-hoc *t*-tests confirmed that the stress group experienced a significant increase in subjective stress from before (timepoint +20 min) to directly after the stressor (+35 min), $t(30) = -7.75$, $p < .001$, $d = -1.39$, with a decline in subjective stress thereafter (+50 min), $t(28) = 5.40$, $p < .001$, $d = 1.00$. A similar picture was observed in the friendly control condition, where participants also reported increases in subjective stress from pre (+20 min) to post TSST (+35 min), $t(27) = -3.58$, $p = .001$, $d = -0.68$, and a decline thereafter (+50 min), $t(26) = 2.14$, $p = .042$, $d = 0.41$. The increase in subjective stress was significantly higher in participants who underwent the stressful as compared with the friendly TSST, $t(43.43) = -4.80$, $p < .001$, $d = -1.21$ (see Fig. 3A).

3.4. Effect of stress and glucose on salivary cortisol

The nonlinear mixed model in which we predicted salivary cortisol concentrations included 434 observations nested in 62 participants. We included a random intercept, random slope, a linear, quadratic and cubic effect of *time* as well as the main and interaction effects of *drink* and *stress*.

The results indicated a significant linear effect of *time*, $b = -3.63$, $SE = 1.25$, $p = .004$, and a significant quadratic effect of *time*, $b = 1.79$, $SE = 0.58$, $p = .002$. Moreover, we observed a significant *time* x *stress* interaction effect, $b = 3.33$, $SE = 1.67$, $p = .047$, a *time*^{2} x *stress* interaction effect, $b = -1.71$, $SE = 1.67$, $p = .027$, and a significant *time*^{2} x *drink* interaction effect, $b = -1.56$, $SE = 0.77$, $p = .049$. The three-way interaction *time* x *stress* x *drink* did not reach the level of significance (see Fig. 3B; for detailed results see supplementary material).

Concerning the significant *time* x *stress* interaction effect, post-hoc *t*-tests confirmed that the TSST led to a significant increase in salivary cortisol from prior to the TSST (timepoint +20 min) to peak concentrations (+50 min) in the stress condition, $t(31) = -3.14$, $p = .004$, $d = -0.56$, but not in the friendly control condition, $t(29) = 0.15$, $p = .878$, $d < 0.03$. Moreover, salivary cortisol decreased significantly between timepoint + 50 min and + 60 min in the stress condition, $t(31) = 2.67$, $p = .012$, $d = 0.47$, but not in the friendly control condition, $t(29) = -0.64$, $p = .527$, $d = -0.12$.

Regarding the significant *time* x *drink* interaction effect, post-hoc *t*-tests showed a significant increase in salivary cortisol between timepoints + 20 and + 50 min, $t(29) = -3.56$, $p = .001$, $d = -0.65$, as well as a significant decrease in salivary cortisol between timepoints + 50 min and + 60 min in the *glucose* group, $t(29) = 3.01$, $p = .005$, $d = 0.55$, with no significant changes between those timepoints in the *water* group, $t(31) = 0.21$, $p = .837$, $d = 0.04$ and $t(31) = 0.13$, $p = .898$, $d = 0.02$.

3.5. Effect of stress and glucose on heart rate

The nonlinear mixed model in which we predicted heart rate included 235 observations nested in 59 participants. We included a random intercept, a linear, quadratic and cubic effect of *time* as well as the main and interaction effects of *drink* and *stress* (see Fig. 3C; for detailed results see supplementary material).

The results indicated a significant quadratic, $b = -1.06$, $SE = 0.13$, $p < .001$, and cubic effect of *time*, $b = -0.66$, $SE = 0.13$, $p < .001$. Moreover, we observed a significant *time* x *glucose* interaction effect, $b = 0.37$, $SE = 0.19$, $p = .046$. No other effects reached the level of significance (see Fig. 3C; for detailed results see supplementary material).

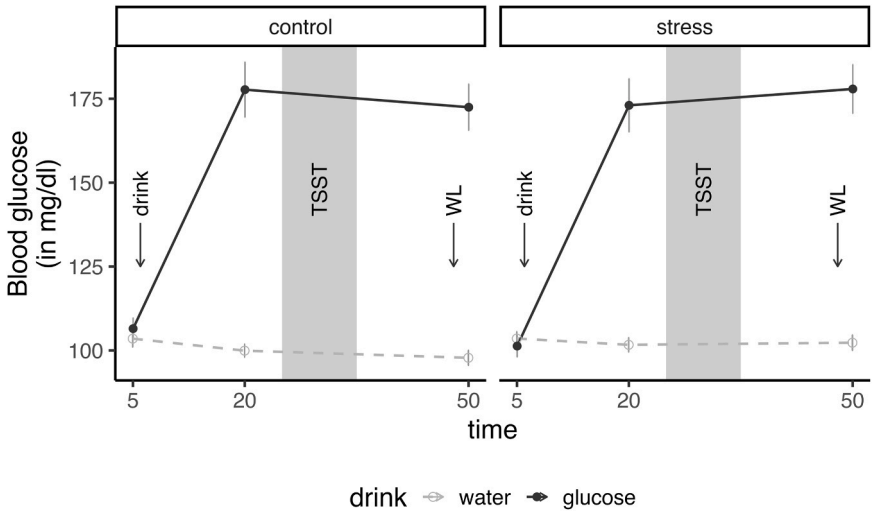

Fig. 2. Changes in blood glucose concentrations (raw values in mg/dL) over time for the four different experimental groups. Blood glucose increased significantly in the glucose groups but did not change in the water groups. Shaded area = Trier Social Stress Test (TSST) with Objects or friendly control condition. WL = wordlist encoding.

Post-hoc *t*-tests confirmed that both the stressful and the friendly control TSST led to a significant increase in heart rate from baseline to anticipation, $t(58) = -5.03$, $p < .001$, $d = -0.65$, and from anticipation to TSST, $t(57) = -10.04$, $p < .001$, $d = -1.32$. While heart rate significantly decreased from TSST to recovery in the overall sample, $t(57) = 13.58$, $p < .001$, $d = 1.78$, it was significantly higher in the glucose as compared with the water groups during the recovery period, $t(53.07) = 3.14$, $p = .003$, $d = 0.83$.

3.6. Correlation between glucose, heart rate and cortisol reactivity

The correlation between *glucose reactivity* and *cortisol stress reactivity* just missed statistical significance in the overall sample, $r(60) = .25$, $p = .050$ (see Fig. 4), and glucose AUCi and cortisol AUCi, $r(60) = .18$, $p = .169$. In exploratory subgroup analyses, we found a significantly positive association in the *stress and water* group (reactivity: $r(16) = .50$, $p = .033$, AUCi: $r(16) = .46$, $p = .053$), but not in the other groups (all $p > .05$).

Further, we neither found a significant correlation between *glucose reactivity* and *heart rate reactivity*, $r(56) = .11$, $p = .421$, and glucose and heart rate AUCi in the overall sample, $r(57) = .20$, $p = .122$, nor in any subgroup analyses (all $p > .05$).

3.7. Effect of stress and glucose on long-term memory performance

The linear mixed model that predicted object recognition performance included 124 observations nested in 62 participants. We included a random intercept, random slope, the main and interaction effects of *object type*, *stress* and *drink* (cf. supplemental material). The results indicated a significant interaction effect of *object type central x stress*, $b = 0.18$, $SE = 0.09$, $p = .045$, indicating that in the stress conditions, central objects were remembered significantly better than peripheral objects, $t(60) = -2.74$, $p = .008$, $d = .63$, but no difference was detected in the friendly control conditions (see Fig. 5A). No other effects were statistically significant (for detailed results see supplementary material). Exploratory *t*-tests showed that the *water* group exposed to *stress* showed significantly better memory for central objects than the *water* group exposed to the *friendly control* condition, $t(29.96) = 2.94$, $p = .006$, $d = 1.01$. This difference was not significant when comparing the groups who consumed *glucose*, $t(27.65) = 0.65$, $p = .522$, $d = 0.24$.

A regression model that predicted object recognition performance by *object type* and the continuous glucose and cortisol reactivity (124 observations, $R^2 = 0.07$) showed that higher *cortisol reactivity* was associated with higher object recognition performance, $b = 0.03$, $SE = 0.01$, $p = .031$. The interaction between *cortisol reactivity* and *glucose reactivity* just missed the threshold for significance, $b = 0.01$, $SE = 0.01$, $p = .051$. No other significant predictors were found (for detailed results see supplementary material).

The linear mixed model in which we predicted word recall performance included 183 observations nested in 61 participants. We included a random intercept, random slope, and the main and interaction effects of *valence*, *stress* and *drink*. The results indicated a significant main effect of *valence* (positive), $b = 1.42$, $SE = 0.50$, $p = .006$, and *valence* (negative), $b = 1.50$, $SE = 0.48$, $p = .002$, indicating that both positive and negative words were remembered significantly better than neutral words (see Fig. 5B). The enhancing main effect of *stress* just missed the significance level, $b = 1.10$, $SE = 0.57$, $p = .058$, and no other main or interaction effects were statistically significant (for detailed results see supplementary material).

The regression model that predicted word recall performance by *valence* and the continuous glucose and cortisol predictors (183 observations, marginal $R^2 = 0.09$, conditional $R^2 = 0.45$) confirmed that *positive*, $b = 0.86$, $SE = 0.33$, $p = .009$, and *negative* words, $b = 1.46$, $SE = 0.33$, $p < .001$, were better remembered than *neutral* words. No other predictors were statistically significant, but a trend for an interaction between *glucose reactivity* and *negative word recall* was observed, $b = -0.01$, $SE = 0.01$, $p = .068$ (for detailed results see supplementary material).

3.8. Sensitivity analysis

We performed sensitivity analyses in G*Power (Faul et al., 2007) to determine the minimum effect size that we could reliably detect with 80 % statistical power given the current sample size of $N = 61$, *alpha* = .05, as well as an observed correlation between object recognition performance (central and peripheral) of $r = .14$ and an observed correlation between word recall performance (neutral, negative, positive) of $r = .39$, respectively. In the analysis using *stress* and *drink* as predictors for object recognition performance, we had 80 % power to detect the highest order interaction effect at a minimum effect size of $f = .29$ (medium effect). In the analysis using *stress* and *drink* as predictors for word recall performance, we had 80 % power to detect the highest order interaction effect at a minimum effect size of $f = .22$ (medium effect).

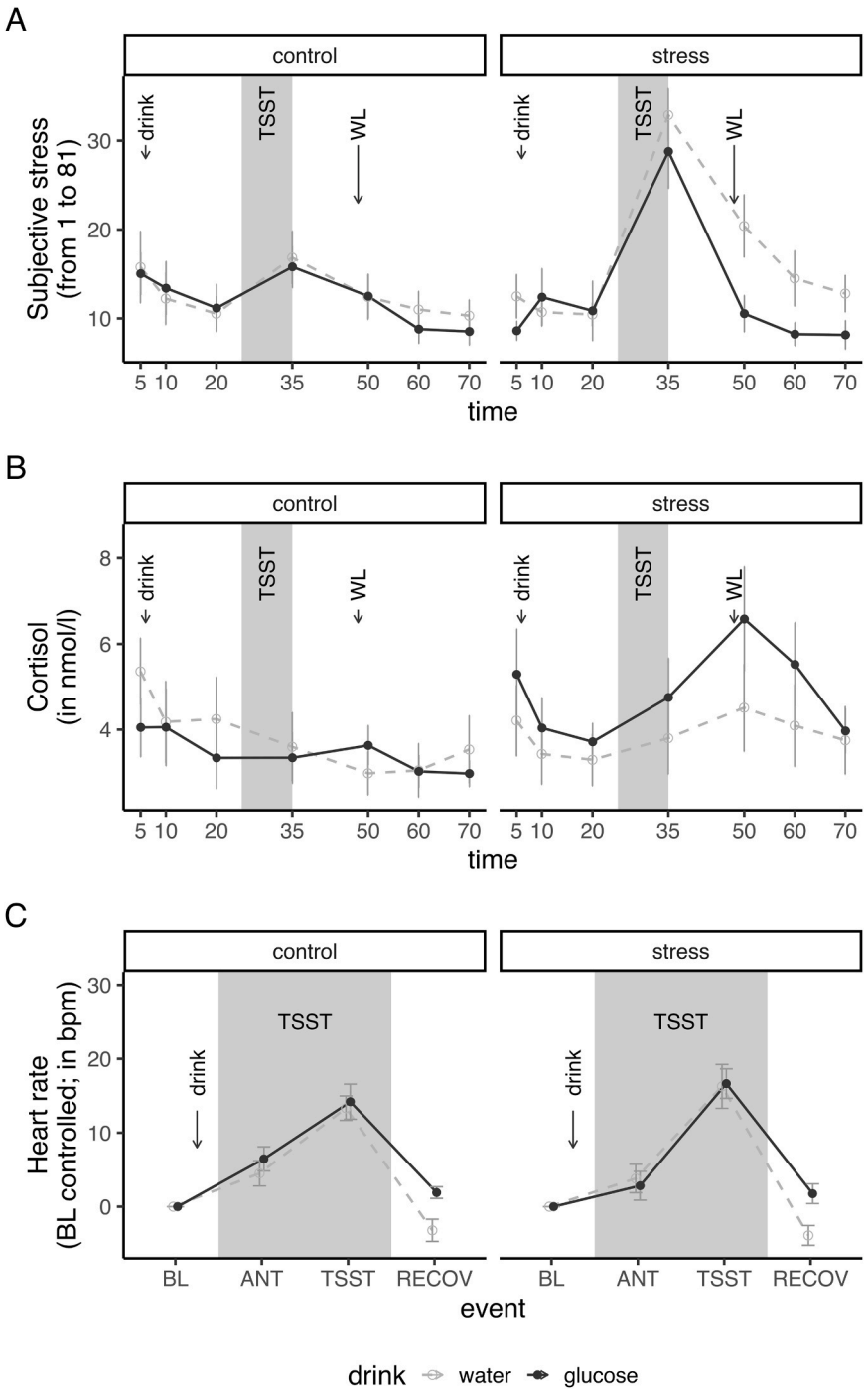

Fig. 3. Changes in subjective stress (A), salivary cortisol concentrations (raw values in nmol/L; B), and heart rate (baseline controlled in beats per minute (bpm); C) over time for the four different experimental groups. Subjective stress increases were more pronounced in the stress as compared with the control group. The TSST increased cortisol concentrations as compared with the friendly control version. Cortisol changes were more pronounced in the glucose groups as compared with the water groups. The stressful and friendly control TSST increased heart rate in a similar manner. Heart rate was elevated in the glucose groups as compared with the water groups during the recovery. Shaded area = Trier Social Stress Test (TSST) with Objects or friendly control condition. WL = wordlist encoding. BL = baseline. ANT = anticipation. RECOV = recovery.

4. Discussion

In this experiment, we investigated the effect of glucose and stress on long-term memory (LTM) of objects presented during a stressful situation and words obtained at the associated cortisol peak. Our analyses confirmed the successful manipulation of blood glucose concentrations through the glucose drink and a cortisol stress response in the groups exposed to stress as compared with the friendly control condition. Even though glucose did not alter autonomic reactivity to the stressful and friendly interview, heart rate was elevated during the recovery period in those who consumed glucose as opposed to water. Memory of information central to the stressor was remembered better than peripheral information. This seemed to be driven by a significant difference in the groups who consumed water, where stress significantly enhanced the memory of central information as opposed to the friendly control setting. Emotional information was generally remembered better than

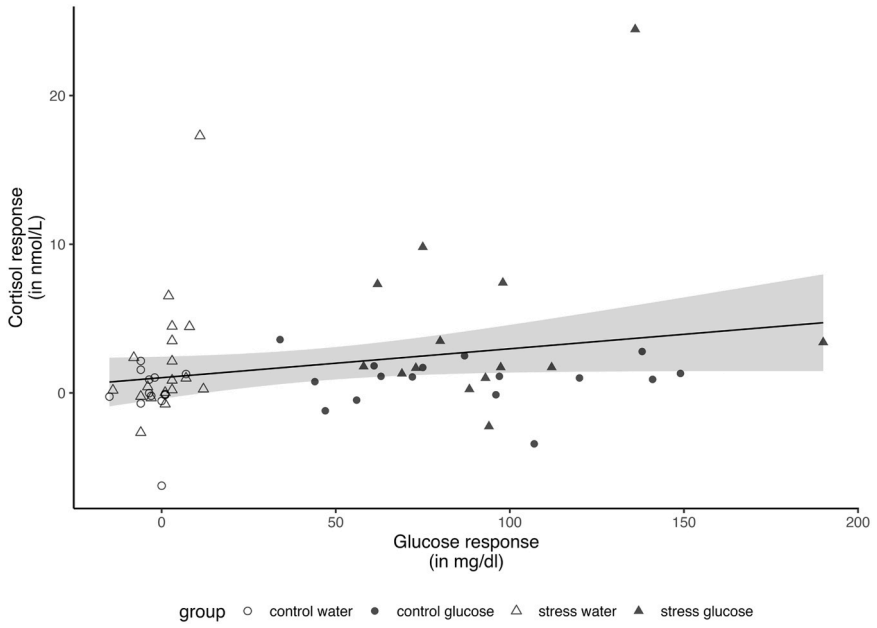

Fig. 4. Scatterplot depicting the correlation between cortisol reactivity (in nmol/L) and glucose reactivity (in mg/dL). The Pearson's correlation coefficient in the overall sample was not significant.

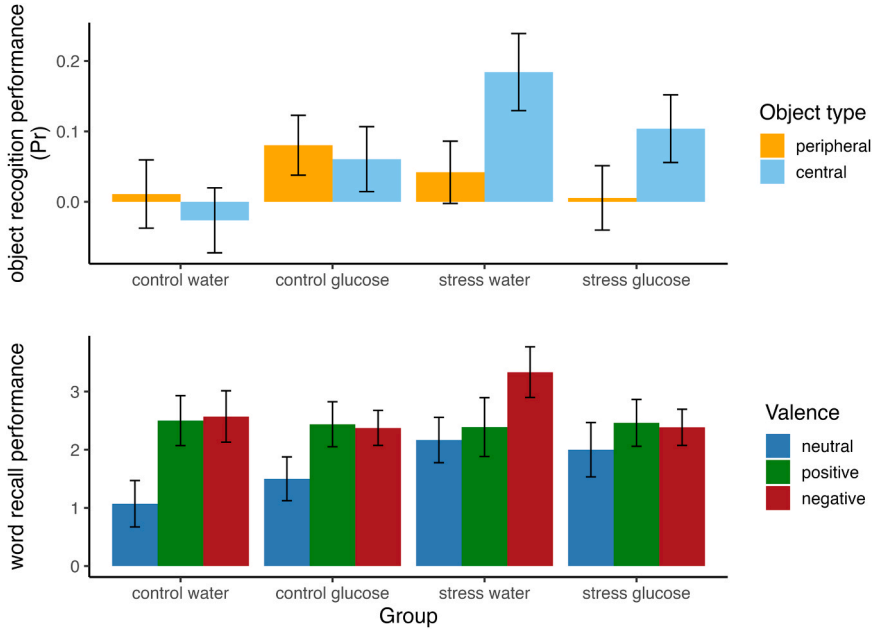

Fig. 5. (A) Object recognition performance for peripheral and central objects in the stress and the friendly control condition. (B) word recall performance for neutral, positive and negative words in the stress and the friendly control condition. In the stress condition, central objects were remembered significantly better than peripheral objects. Negative and positive words were remembered significantly better than neutral words.

neutral information. A higher cortisol stress reactivity was associated with better object recognition. Glucose did however not modulate these effects, suggesting that it may not have a strong influence on the formation of LTM across stressful and friendly contexts in this sample.

In line with previous psychosocial stress studies (Bentele et al., 2021; Gonzalez-Bono et al., 2002; Kirschbaum et al., 1997; Meier et al., 2021; von Dawans et al., 2020; Zänkert et al., 2020) and a recent meta-analysis (Kördel et al., 2025), glucose increased the cortisol stress response compared with water. Of note, glucose also led to a slight increase in cortisol dynamics in the friendly interview condition. On the one hand, previous research demonstrated that glucose intake alone does not activate the HPA axis (Kirschbaum et al., 1997). On the other hand, stimulatory effects of high concentrations of peripheral insulin on the HPA axis have been reported (Fruehwald-Schultes et al., 1999, 2001), which might also explain the cortisol lunch peak (Legler et al., 1982). As a dampening effect of fasting on HPA axis reactivity has been reported in animals (Choi et al., 1996), it is not entirely clear whether the effect observed is due to an increased cortisol response after glucose consumption, or a dampening effect of fasting. In the current experiment, participants' blood glucose concentrations were at the upper limit of the fasting range when entering the laboratory and remained well above hypoglycemia throughout the study in the water groups. This may indicate that it was the glucose manipulation rather than the fasting that drove the effect.

Cortisol responder rates were highest in the glucose group exposed to stress (71 %) and lowest in the water control group (14 %), with the remaining groups lying between the two (control sugar: 31 %; stress water: 39 %). Like some previous studies (Meier et al., 2021; von Dawans et al., 2020), and unlike others (Gonzalez-Bono et al., 2002; Kirschbaum et al., 1997), we found no convincing evidence for a linear link between blood glucose and cortisol reactivity. This casts doubt on the notion that the rise in blood glucose following glucose consumption is the driving force behind the increased cortisol reactivity (Gonzalez-Bono et al., 2002) and warrants further investigation. While the stress manipulation was successful, the cortisol response in the stress groups (mean increase = 3.21 nmol/L, *SD* = 5.48) was lower than in previous TSST studies that included the speech and the mental arithmetic task. This might be related to the omission of the mental arithmetic task (Goodman et al., 2017). As our study confirmed a glucose enhancement for the cortisol stress response despite the relatively low cortisol reactivity, previous speculations that the glucose effect depends on stressor intensity are challenged (Rüttgens and Wolf, 2022), making it plausible that the effect depends on the nature of the stressor (physiological vs. psychosocial) and neural correlates thereof (Kogler et al., 2015); yet, these are mere speculations and the effect of glucose on various types of stressors should be tested more systematically in future studies. Interestingly, the glucose group exposed to stress also showed a higher variance in cortisol reactivity as compared to the water group. These results imply that other factors beyond the increase in blood glucose concentration contribute to the increased cortisol stress reactivity following glucose consumption. Potential targets for future studies may be the glucose-induced release of insulin, or other nutrition related factors that might act on the PVN through energy availability and satiety signals (Choi et al., 1996) and thereby activate the HPA axis (Rohleder and Kirschbaum, 2007). As there is currently a lack of experiments that test causal effects of metabolic hormones on stress reactivity (Kördel et al., 2025), even though interactions between nutritional factors and the HPA axis are manifold (Rohleder and Kirschbaum, 2007; Ulrich-Lai and Ryan, 2014), this blind spot should be targeted in future research to enhance our understanding of the stress-metabolism crosstalk.

The current study replicated that information central to a stressful episode is remembered better as compared with peripheral information (Schwabe et al., 2012; Wolf, 2019), which might be related to alterations in neural representations (Bierbrauer et al., 2021). This effect seemed to be driven by the fact that in the groups who consumed water, stress significantly enhanced the memory of central information as opposed to the friendly control setting; yet this was not the case in the groups that consumed glucose. While being exploratory, these results suggest that glucose may not add to the memory-enhancing benefit of cortisol. Despite this, centrality did not seem to play a role in the friendly context, a result that opposes previous findings (Wiemers et al., 2013). Results from the continuous approach further highlighted that higher cortisol reactivity was associated with remembering more objects, which opposes prior meta-analytic findings (Shields et al., 2017).

Further, independent from the stress manipulation, emotional words were remembered better than neutral words (Talmi, 2013). Although previous studies suggested that stress before encoding evokes better memory of emotional content (Merz, 2017; Merz et al., 2019; Payne et al., 2007), the enhancing effect of stress on word recall just failed to reach the level of statistical significance in our analysis, which might be because we could not consider potential sex and menstrual cycle phase related effects (Merz, 2017) or potential effects of age (Niu et al., 2024; Roelfsema et al., 2017), which should be addressed in the future.

Lastly, and in contrast to previous studies (Benton and Owens, 1993; Foster et al., 1998; Meikle et al., 2005; Smith et al., 2011), our findings did not support the 'glucose memory facilitation effect'. Previously, an inverted U-shape for the effect of glucose on memory has been discussed, with a dose of 25 g being considered optimal to facilitate memory outcomes (Sünram-Lea et al., 2011). Our dose was three times as high and might thus have hindered to observe the enhancing effect. Even though our results indicated that glucose intake affected cortisol stress reactivity, this effect did not seem to transfer to the LTM outcomes, even though they have often been shown to be affected by cortisol in the past (Wolf, 2009). This calls into question whether the enhancement of the cortisol stress response through glucose is strong enough to cause observable cognitive alterations. In fact, a recent meta-analysis concluded that while glucose robustly increases cortisol stress reactivity, the effect is small in nature (Kördel et al., 2025). As our stressor seemed to evoke rather modest cortisol responses, a final conclusion is at this point difficult to establish. Future studies may investigate potential dose-response effects more systematically (Sünram-Lea et al., 2011).

Our study has some limitations that need to be considered when interpreting the results. Our friendly TSST condition evoked a mild subjective and cortisol stress response, with heart rate reactivity being comparable to the stressful version. This implies that the situation was arousing, even though not capable of mounting a full HPA axis response, with potential effects on LTM outcomes (Hoscheidt et al., 2014). Moreover, this implies that choosing a less arousing control condition in future studies may increase the likelihood to detect effects. Due to the explorative nature of this study, the sample size was relatively small and mainly driven by feasibility. Although it is similar to a previous study that explored effects of glucose on stress and memory (Rüttgens and Wolf, 2022), it might have been too small to detect smaller sized effects. This critically limited the statistical power to detect significant interaction effects in the linear mixed models that predicted LTM performance despite significant pairwise group comparisons being present as shown in exploratory *t*-tests. At the same time, increasing the number of stimuli used in the memory tasks and considering stimulus arousal would further increase precision of the LTM estimates (Nebe et al., 2023), which should be considered in the future. Further, the random allocation of participants to experimental groups resulted in unbalanced sample sizes that might have reduced statistical power calling for a replication. Additionally, the experimental groups were not balanced regarding biological sex, and displayed a surplus of female participants, preventing us from performing analyses of potential sex differences that are well documented for both the cortisol stress response (Liu et al., 2017) and carbohydrate metabolism (Wismann and Willoughby, 2006). Also, the sample primarily consisted of young adults, preventing us to draw conclusions about potential effects of age, which are well documented in the memory (Niu et al., 2024) and stress literature (Roelfsema et al., 2017). Even though the participant and the experimenter were blind to the drink content prior to the experiment, participants could guess by the sweet taste and the experimenter could infer from the blood glucose measurements whether participants consumed glucose or water. This could have biased the results and could have (partly) been prevented by using a sweetened, non-caloric control drink. Due to effects of sweetener on the cortisol stress response which we observed in a previous study (Meier et al., 2021) we decided against this option. Future studies should carefully consider which control drink to include with regards to the respective design. Also, some covariates that may influence stress reactivity or LTM performance, such as sleep (Niu et al., 2025; Payne and Kensinger, 2018), socio-economic status or education were neither assessed nor controlled for in the current study and should be considered in the future.

This study is the first to investigate the effect of glucose and psychosocial stress on LTM in a controlled laboratory experiment with well-established paradigms. In line with a previous report that focused on the effects of stress on retrieval (Rüttgens and Wolf, 2022), our results on effects of stress during and prior to encoding suggest that glucose may not have a strong effect on memory formation of stressful episodes in healthy adults under the conditions tested. Previous research suggested that the memory enhancing effects of glucose in non-stressful contexts are mediated by effects of glucose on the hippocampus. As the blockade of insulin-dependent glucose transporters impairs LTM in rats (Pearson-Leary and McNay, 2016), studying central effects of insulin more systematically may be a promising next step. Indeed, several

reports suggest a modulating effect of central (Bohringer et al., 2008) and peripheral insulin on the HPA axis (Fruehwald-Schultes et al., 1999, 2001) and an effect of central insulin on memory (Hallschmid, 2021; Stockhorst et al., 2004). Furthermore, clinical reports suggest that glucose intake after a traumatic event enhances context learning (Glenn et al., 2014) and central insulin enhances fear extinction (Ferreira de Sá et al., 2020). In light of this, further exploring the diverging central and peripheral effects of insulin in the context of how memories of a stressful episode are formed may be an attractive target for further evaluation. Bettering our basic understanding of these relations may support the development of interventions that contribute to the prevention of fear- and stress-related disorders.

Supplementary information

Supplementary information, data and the analysis code can be retrieved from the following Open Science Framework (OSF) project: https://osf.io/te5ng/.

CRediT authorship contribution statement

Maria Meier: Writing – review & editing, Writing – original draft, Visualization, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Oliver T. Wolf:** Writing – review & editing, Resources, Methodology. **Jens C. Pruessner:** Writing – review & editing, Resources, Methodology, Funding acquisition, Conceptualization. **Christian J. Merz:** Writing – review & editing, Resources, Methodology. **Tobias Rüttgens:** Writing – review & editing, Resources, Methodology.

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Declaration of Competing Interest

The authors declare no competing interests relevant to this publication.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2025.107741.

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