

The Influence of Baseline Sleep on Responses to New Stressors:  
A systematic Review

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## Highlights

- 86% of studies link baseline sleep to later stress; most show worse sleep → more stress
- Baseline Rapid-Eye-Movement (REM) sleep show mixed effects in humans and animals
- Subjective sleep measures are the most consistent in predicting stress reduction
- Neuroendocrinal levels in animals often show opposite effects (worse sleep → less stress)

## Declaration of Interest

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## Abstract

*Background:* The way we process and memorize stressors has been repeatedly shown to be affected by following sleep periods, particularly rapid eye movement (REM) sleep, but far fewer studies have examined how baseline sleep prior to stressor exposure might shape our stress response after exposure. In this systematic review, we explored how subjectively and objectively measured baseline sleep, including REM sleep, contributes to future stress responses.

*Methods:* We identified relevant publications from Web of Science, PubMed, EMBASE, and PsychInfo through August 2024. Human and animal studies were included if they assessed baseline sleep prior to stress exposure, incorporated a stressor, and statistically analyzed stress outcomes in relation to baseline sleep. Studies that only incorporated populations with existing sleep or stress abnormalities were excluded.

*Results:* Fifty-one independent studies (39 human, 12 animal), consisting of both real-life and experimental stressors met the inclusion criteria. Overall, a majority of studies ( $n = 34$ ) showed that worse baseline sleep predicted stronger stress responses – a result that was most consistent in human studies measuring sleep subjectively. Animal studies showed varying effects, with sleep disruption often predicting reduced physiological stress response ( $n = 6$ ). REM sleep yielded varying stress outcomes as well, across species.

*Conclusions:* Baseline sleep prior to stressor exposure often acts as a predictor of subsequent stress response. Factors like species, stressor type, the sleep assessment tool, and the stress response measure can influence the direction of the effect. Perceived sleep quality might predict stress coping capabilities more than objective measures like REM levels.

Keywords: Sleep, REM sleep, stressor, stress, fear

## 1. Introduction

The effects of sleep on well-being and emotional processing are well documented (Genzel et al., 2015; Tempesta et al., 2018; Walker & van der Helm, 2009). In particular, the bi-directional effects of sleep and stress have been investigated extensively over the last decades, often in relation to conditions such as posttraumatic stress disorder (PTSD) where sleep is not only considered a hallmark symptom (Germain, 2013; Ross et al., 1989) but also a contributor to its development (Pace-Schott et al., 2015, 2023; Seo et al., 2022). Nevertheless, studies of sleep and stress – particularly experimental paradigms like fear conditioning which include the induction of a stressor – have primarily focused on how sleep affects (and is affected by) the memory of a stressful experience that has already occurred beforehand (for review, see Davidson & Pace-Schott, 2020; Schenker et al., 2021). Far fewer studies have investigated how baseline patterns of sleep prior to stressor exposure influence the encoding of that stressor, and fewer still have examined how habitual sleep (i.e., the typical sleeping patterns of an individual measured over multiple nights) contributes to this process. This gap likely stems from the substantial logistical challenges of conducting longitudinal studies, especially when involving objective physiological measures of sleep in humans rather than subjective self-reports.

With recent advancements in mobile sleep monitoring, studies of baseline sleep and its relation to future exposure to stressors are now becoming more abundant. Understanding this relationship is important because it could facilitate identifying modifiable risk factors that could inform early intervention strategies. For example, detecting habitual sleep patterns associated with increased risk of PTSD development in high-stress populations, such as military personnel or emergency responders (Colvonen et al., 2019; Short et al., 2020), could aid in preventive screening and resilience-building programs. Moreover, investigating baseline sleep and stress could help to further extend existing theoretical models developed to account for the effect of sleep on fear recall and extinction. Among those, the “sleep to forget, sleep to remember” approach proposes that sleep, particularly Rapid-Eye-Movement (REM) sleep, reduces the emotional tone attached to encoded stressful memories (Walker, 2009) and the sleep recalibration hypothesis suggests REM sleep modulates tonic norepinephrine (NE) levels in the amygdala, helping to maintain a healthy balance between sensitivity and selectivity to perceived threat (Goldstein & Walker, 2014). These models, however, are largely focused on REM sleep

following exposure to a stressor. Therefore, evaluating current evidence regarding the influence of baseline sleep prior to stressor exposure might help enrich the models to account for the effects of day-to-day-sleep on stress. Nevertheless, to date, no review has focused on exploring this particular relationship.

To this end, the current systematic review explores the current landscape of studies aimed to evaluate if and how baseline sleep is associated with future reaction to stressors. We include both human and animal studies, while distinguishing human studies to those that examined real-life stressors and those that induced stress artificially in experimental settings. Moreover, to gain a broad perspective, we considered studies that employed a variety of sleep and stress measures, including single-night and multiple-night sleep assessments (using either objective or subjective measures, as well as sleep deprivation paradigms); retrospective sleep assessments (e.g., through questionnaire items like “how did you sleep during the last month?”); and behavioral and physiological measures of stress, ranging from subjective stress reports to skin conductance response (SCR), neuroendocrinal outcomes, and brain imaging. We aimed to answer the following questions: (a) Overall, does baseline sleep predict future reaction to stressors? (b) Is baseline REM sleep particularly consequential in this relationship? (c) What methodologies are most effective in identifying this association, and what methodological limitations should be addressed in future studies?

## **2. Materials and Methods**

The review was conducted based on four primary databases, PubMed Web of Science, PsychINFO and Embase, selected for their robust coverage of psychological and sleep-related research. The search strategy was aimed to identify studies that assessed pre-stressor “baseline” sleep characteristics and examined their relationship to subsequent outcomes following exposure to a stressor, ensuring temporal precedence between the specified stressor and sleep.

### *2.1. Search Strategy*

Keywords for the systematic search were designed to cast a wide net to capture as many publications as possible that focus on the two core elements of the current review, sleep (e.g.,

“sleep”, “insomnia”) and stress (e.g., “stress”, “trauma”, “fear”, “PTSD”, “anxiety”), while ensuring there are indications of a temporal direction between the two such that one precedes the other (e.g., “prior”, “precede”, “pre-deployment”, “longitudinal”, “trait-level”). The following query was used for PubMed (see Section S1 in Supplementary Materials for the corresponding queries in all databases):

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(sleep[Title] OR insomnia[Title]) AND (stress[Title] OR fear[Title] OR  
anxi*[Title] OR ptsd[Title] OR posttraumatic[Title] OR post-traumatic[Title] OR  
trauma*[Title] OR predeployment[Title] OR pre-deployment[Title]) AND  
(prior[Title/Abstract] OR subsequent[Title/Abstract] OR  
retrospective*[Title/Abstract] OR prospective*[Title/Abstract] OR  
longitudinal[Title/Abstract] OR precede*[Title/Abstract] OR  
biomarker[Title/Abstract] OR trait-level[Title/Abstract])
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Only peer-reviewed studies were included in the search; search dates extended through August 2024 with no lower bound, to capture both historical and recent research. To validate the sensitivity of our search strategy, we cross-checked results against a benchmark list of 20 pre-identified relevant studies (based on the authors’ familiarity with the topic area), verifying that all studies in the list are retrieved.

## 2.2. Study Selection

Studies were included if they met the following criteria:

1. Were experimental or observational studies involving humans or animals.
2. Assessed sleep prior to stress exposure using subjective (e.g., sleep diaries, self-reports) or objective (e.g., actigraphy, polysomnography (PSG)) methods, or through manipulation of sleep, regardless of whether sleep was also assessed later after stressor exposure.
3. Included a stressor, defined as an inciting psychological, physiological, acute or chronic incident that the study intentionally incorporated to evaluate its impact on the stress system (e.g., combat deployment, car accidents, COVID-19 pandemic, fear conditioning, induced social anxiety, and others).

4. Assessed stress outcomes due to the stressor, whether subjective (e.g., perceived stress scales) or objective (e.g., skin conductance response, cortisol levels, brain activation in relevant regions, heart-rate variability).

Studies were excluded if they:

1. Did not establish a temporal differentiation between sleep and stress, where sleep is clearly measured prior to stressor initiation (regardless of whether sleep was also assessed later after stressor exposure).
2. Lacked statistical analysis assessing the link between baseline sleep parameters and later stress development.
3. Only included populations with pre-existing sleep or stress abnormalities.
4. Had unclear methodologies or insufficient data for extraction.

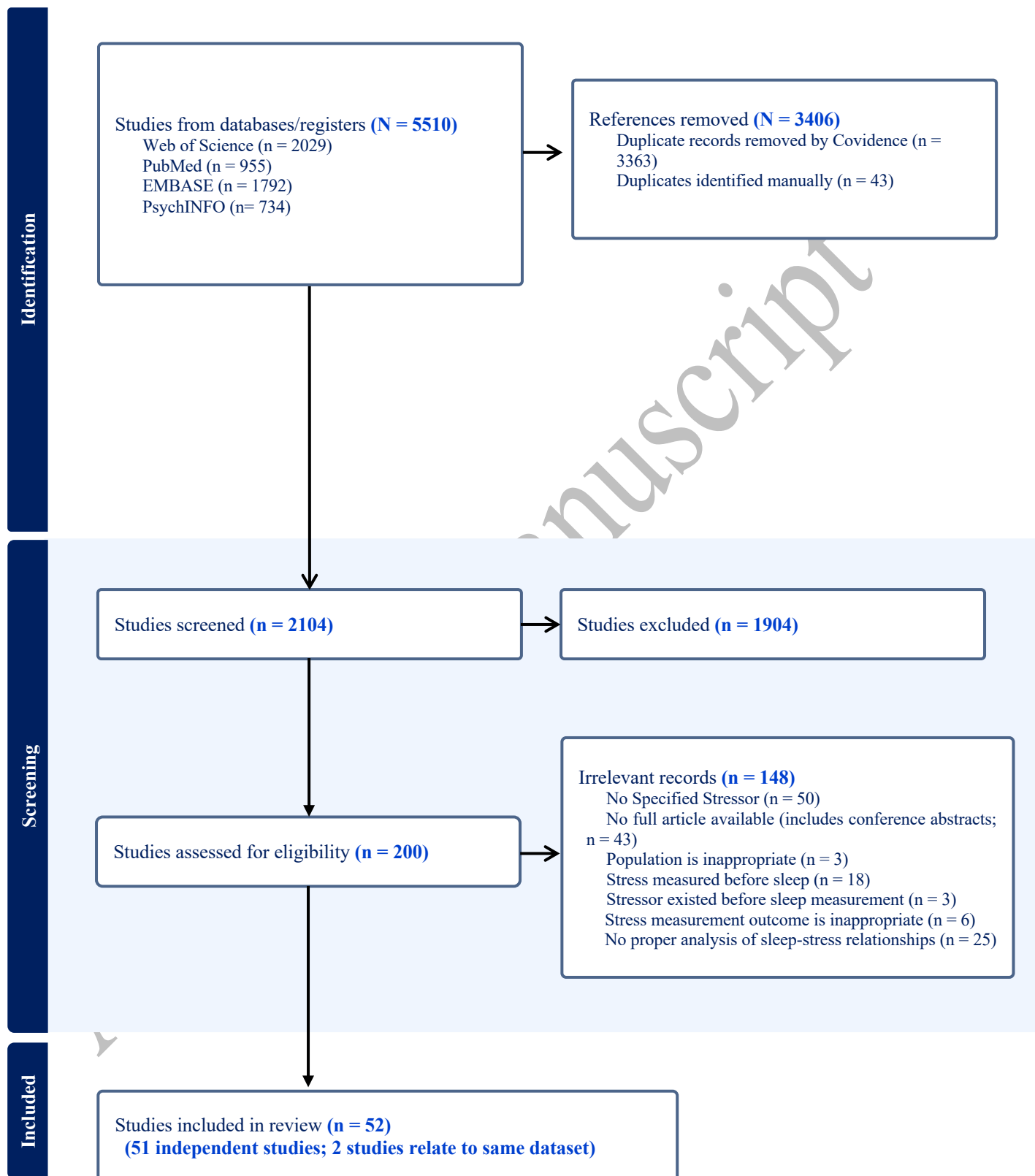
### *2.3. Data Extraction, Managing Risk Bias*

Two reviewers independently extracted and evaluated all data to determine study inclusion. Discrepancies between reviewers were resolved through discussion and reaching consensus and then validated again for relevance by a third reviewer. Only data related to the link between baseline sleep and future reaction to a stressor were considered, even if the study included additional periods of sleep following stressor exposure. Specifically, for each included study, we systematically extracted:

- Study design: Longitudinal vs experimental, within vs. between-subject design.
- Population characteristics: sample size, age group (e.g., children, adolescents, adults), specific subgroups (e.g. military personnel, healthy populations, clinical populations), and relevant psychiatric or medical conditions (e.g., insomnia, PTSD, anxiety).
- Baseline sleep measurement methods: objective (e.g., actigraphy, PSG) vs subjective (e.g., sleep diaries, self-reports), duration of assessment (single night vs. multiple nights), and specific variables examined (e.g., REM sleep, total sleep time, sleep efficiency).
- Specified Stressors: Real life vs. experimentally induced stressors.

- Stress measurement methods: subjective (e.g., perceived stress scales) vs. objective (e.g., SCR, cortisol, heart rate variability, brain activation).
- Statistical methods: analytical approaches, key covariates controlled for (e.g. baseline anxiety).
- Key finding: Main results related to the association between baseline sleep and stress response, including classification of effect size to weak, medium or strong (based on standards for Cohen's d and related metrics if reported, or derived using standard techniques appropriate for the statistical methods used; see Table S1).

In addition, four reviewers ran a risk of bias assessment for all included studies, with one reviewer compiling all results to ensure consistency of decisions. These assessments provided a descriptive overview of methodological strengths and weaknesses in the evidence base and were not used to determine study inclusion or weighting in the synthesis. For human studies, Parallel-group randomized controlled trials (RCTs) were evaluated using the Cochrane risk-of-bias tool for randomized trials (RoB 2), and crossover trials using the RoB 2 Crossover extension, each covering five domains: randomization, deviations from intended interventions, missing outcome data, outcome measurement, and selective reporting (Sterne et al., 2019). RCT studies in animals were assessed using the SYRCLE tool, an adaptation of RoB principles for animal intervention studies, and translated to the same 5 domains (Hooijmans et al., 2014). Non-randomized human studies were assessed with the Risk Of Bias In Non-randomized Studies – of Interventions, Version 2 (ROBINS-I V2), which evaluates bias due to seven domains including confounding, participant selection, intervention classification, deviations from intended interventions, missing data, outcome measurement, and selective reporting; domain variants were selected based on whether exposures were baseline-only or time-varying (Sterne et al., 2016). Overall risk-of-bias judgment for each study was derived using the standard decision rules of each tool based on the individual ratings of its corresponding domains.



**Figure 1: PRISMA Flowchart**

### 3. Results

A PRISMA flow diagram illustrating the search and screening process is outlined in Figure 1. The initial database search yielded 5,510 studies (PubMed = 955, Web of Science = 2,029; Embase = 1792; PsychINFO = 734). After removing duplicates ( $n = 3,363$ ) using Covidence, 2,104 studies remained for title and abstract screening. Of these, 1,904 studies were excluded for not meeting the inclusion criteria. The full texts of 200 studies were assessed for eligibility, resulting in 51 independent studies being included in the final synthesis (two studies, Feng et al. 2018a and 2018b, relate to the same dataset and are therefore counted as one); 148 studies were excluded for the following reasons: no full article available ( $n = 43$ ), unspecified stressor ( $n = 50$ ), assessment of stress only prior to sleep ( $n = 18$ ), no direct or appropriate analysis of the relations between baseline sleep and stress outcomes ( $n = 25$ ), or other factors ( $n = 12$ ; see Figure 1 for details). Representative examples of excluded studies are shown in Supplementary Table S2, illustrating the main exclusion categories. The complete list of included studies is shown in Table 1.

#### 3.1. Overall Characteristics of Included Studies

Overall, there were 39 human studies and 12 animal studies included in our review. Most human studies examined adults of both sexes, with only a few focusing on children and adolescents or including participants of only one sex. Moreover, the majority of human studies were of healthy populations, although a few included, alongside a healthy control group, participants with insomnia symptoms or examined populations with specific diagnoses such as bipolar disorder, non-suicidal self-injury, and generalized anxiety. Studies with animals were almost evenly divided between rats and mice, mostly male, with one study investigating prairie voles.

Baseline sleep across studies was monitored in a variety of ways, including objective measures like polysomnography (PSG) in both humans and animals and actigraphy (humans only), and subjective measures like sleep diaries and surveys (humans). In the case of sleep surveys, the majority were taken before the introduction of the stressor and only a few assessed baseline sleep habits retroactively after stressor exposure. When baseline sleep was monitored,

the duration varied from a single night to a week, with only some animal studies extending beyond one week. In studies where the baseline sleep effect was examined through manipulation of sleep using sleep deprivation (8 in humans, 10 in animals), total sleep deprivation was the most common method in both humans and animals, followed by partial sleep deprivation and finally circadian disruption (one human study) and early-life sleep disruption (one animal study).

The types of stressors examined were also varied across studies. In humans, the stressors examined were almost evenly distributed between real-life and artificial stressors. Real-life stressors included combat deployment, COVID-19 pandemic, occupational stress and work demands, final exams, traumatic childbirth, infant inoculation, motor vehicle accident, and traumatic injury. Artificial stressors often included an electric shock or a Trier Social Stress Test (TSST), with other studies using a speech task, failure induction task, Stroop color-word interference task, and a trauma film paradigm. In animal studies, the types of experimental stressors often included electric foot shocks or restraint stress, with four studies employing different techniques like Chronic Social Defeat, single prolonged stress, and open field exposure.

The risk-of-bias assessment for randomized trials (RoB 2; Supplementary Table S3) showed that bias arising from to the randomization process, missing outcome data, and measurement of the outcomes (Domains 1, 3 and 4) was mostly low. However, bias due to deviations from the intended intervention (Domain 2) and, particularly, selective reporting of results (Domain 5), were common, resulting in the majority of studies exhibiting “some concerns” in their final overall rating. These patterns were consistent across both human and animal studies. Only three studies (two animal, one human) were judged to be at high overall risk of bias, again primarily due to concerns in Domains 2 and/or 5. For the non-randomized studies (ROBINS-I; Supplementary Table S4), approximately half were characterized as having an overall moderate risk of bias and the other half as serious risk. No study was judged to have an overall “Critical” risk. Moderate overall ratings were mainly driven by moderate concerns about the existence of confounding factors (Domain 1) and selective reporting of results (Domain 7), whereas studies with serious risk of bias almost always showed serious concerns about confounding, sometimes accompanied by serious concerns in additional domains. Biases due to deviations from the intended interventions or the measurement of the outcomes (Domains 4 and 6) were generally low. When stratifying by whether sleep was measured subjectively or objectively (see Table 1), non-randomized studies employing subjective measures were more

often classified as serious risk (13 of 20) whereas those using objective measures were more commonly rated as moderate risk (8 of 10). Overall, non-randomized studies seemed to be more vulnerable to bias than randomized trials (compare Supplementary Tables S3 and S4).

### *3.2. Baseline Sleep Effects Across All Studies*

Of the 51 total human and animal studies reviewed, 44 studies (86%) found a significant association between sleep and stress outcomes, while 7 studies (14%) reported null effects. Percentages were quite consistent across species, with 85% and 92% of studies reporting significant effects in humans and animals, respectively. Of those reporting significant findings, 34 studies (77%) found that poor sleep predicted increased stress response while only 8 studies (18%; 2 humans, 6 animals) reported the opposite effect and two animal studies found effects in both directions depending on sleep measure (Radwan et al., 2021; Sgoifo et al., 2006). Among studies with significant findings, reported effect sizes in humans were typically in the medium-to-high range, particularly in military, occupational, and trauma-related contexts; whereas in animals, effect sizes were often large, especially when outcome variables were related to fear conditioning, hypothalamic-pituitary-adrenal (HPA) axis response, or neurochemical regulation.

Given that the majority of studies found significant effects, it is warranted to consider whether results might have been skewed due to publication bias. One way to address this possibility is by restricting our analysis to only those studies that considered the association between baseline sleep and future stress as secondary to other research objectives (thus reducing the risk of null effects being shelved). Applying this criterion, six studies were identified (based on the reported experimental design or by personal knowledge of the study's objectives), 5 human and 1 animal. Of these, 4 (66%) found significant association between poor sleep and increased stress response whereas 2 (33%) had null effects. While it is difficult to draw strong conclusions from such a small sample, the general trend of an existing relationship between poor baseline sleep and increased future stress response seems to be maintained, though a publication bias may have also played a role given the smaller percentages of studies finding any effect compared to the full literature.

### 3.3. Baseline REM Sleep Effects

Out of the studies reviewed, seven human studies used PSG or electroencephalography (EEG) during baseline sleep monitoring, allowing reliable REM sleep detection. Of these, three studies reported a significant association between baseline REM sleep and subsequent stress responses. Two of the three studies found that increased REM sleep is generally associated with more adaptive stress responses, including fewer intrusive memories (Alkalame et al., 2024), and weaker neural activity in the amygdala during fear learning (Lerner et al., 2017). In contrast, one study found that greater REM percentage predicted increased test anxiety (Larios and Lerner, 2024), suggesting that habitual REM sleep may enhance stress response to anticipatory stress. Four other studies did not report significant effects, either because no effects were found (Brand et al., 2018; Marshall et al., 2014), or because the focus of the studies was total sleep deprivation rather than REM sleep (Goldstein et al., 2013; Franzen et al., 2011).

Among the animal studies, six studies employed methodologies that could provide REM-specific conclusions. Four studies employed REM sleep deprivation (RSD) to directly assess the role of REM sleep in stress response. RSD was associated with altered HPA-axis regulation through blunting of the normal stress-induced adrenocorticotropic hormone (ACTH) response (Machado et al., 2008) as well as the corticosterone response (Oyola et al., 2019), and decreased levels of fear of exploration (Hicks & Moore, 1979). Conversely, chronic REM disruption early in life was associated with increased ethanol consumption after stress exposure and heightened amygdala activation, indicating a developmental vulnerability to stress (Jones et al., 2020). Two other studies measured baseline REM sleep patterns without manipulation and examined their relationship to later stress. One found that low REM sleep continuity prior to the stress exposure predicted greater startle response (Polta et al., 2013), while the other did not find an effect of REM sleep but found that increased NREM time and fragmentation predicted stress susceptibility (Radwan et al., 2021).

Overall, across humans and animal studies, there were mixed results with 62% (8 of 13) of the studies finding baseline REM sleep is linked to future stress response (with four of the eight showing a reduction of stress with more REM) and 38% (5 of 13) finding no associations with baseline REM sleep (see Table 2).

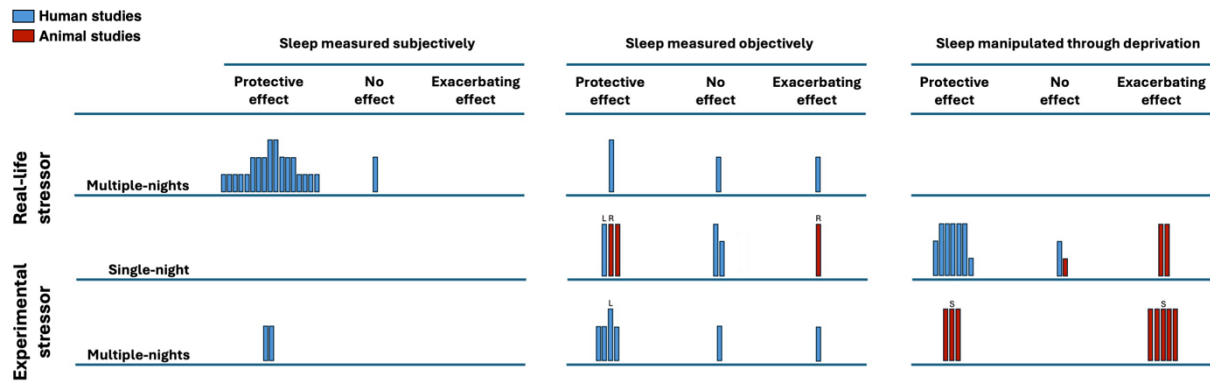
**Table 2.** Summary of Studies by Type of Baseline REM Sleep Effect on Subsequent Stress Response.

Protective Effect	Exacerbating Effect	No Effect
Alkalame et al., 2024* <sup>†</sup>	Larios & Lerner, 2024* <sup>†</sup>	Brand et al., 2018* <sup>†</sup>
Jones et al., 2020 <sup>††</sup>	Machado et al., 2008 <sup>††</sup>	Goldstein et al., 2013* <sup>†</sup>
Lerner et al., 2017* <sup>†</sup>	Hicks & Moore, 1979 <sup>††</sup>	Marshall et al., 2014* <sup>†</sup>
Polta et al., 2013 <sup>†††</sup>	Oyola et al., 2019 <sup>††</sup>	Radwan et al., 2021 <sup>†</sup>
		Franzen et al., 2011* <sup>†</sup>

*Note.* \* denotes Human Studies. Tildas denote the main type of REM sleep measure:  
<sup>†</sup> REM duration/percent; <sup>††</sup> REM deprivation; <sup>†††</sup> REM continuity/fragmentation

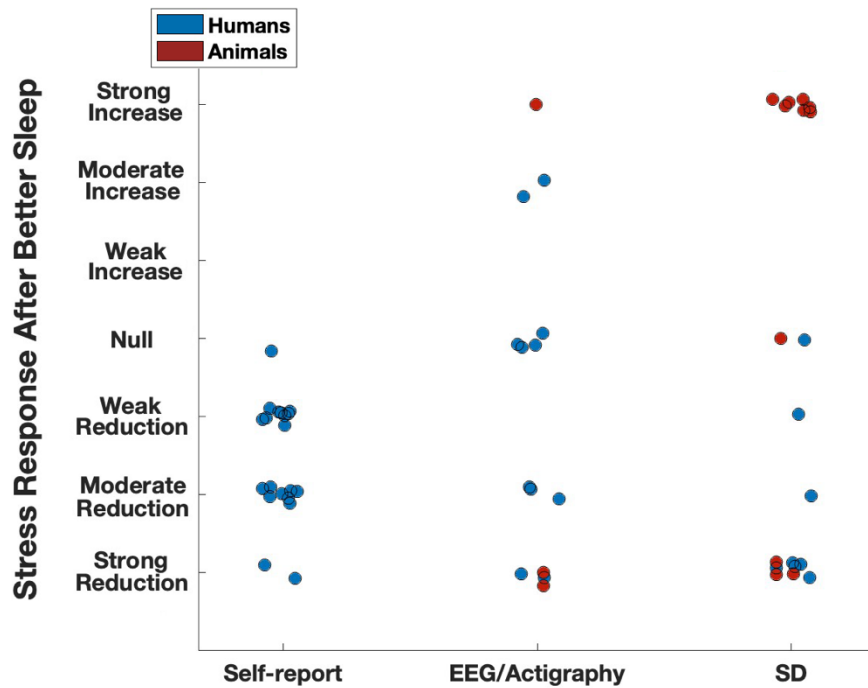
### 3.4. Factors Influencing the Associations Between Baseline sleep and Future Stress Response

Across studies, several finer patterns could be identified when taking into consideration the direction and size of the effects (see harvest plot in Figure 2). First, all but one of the 20 human studies measuring sleep through subjective measures (self-reports, caregiver reports, or sleep diaries) suggested a protective effect of sleep. This was by far the most consistent finding across the literature reviewed, with effects showing considerably more variability in both humans and animals when sleep was measured using objective measures or when sleep deprivation was employed. Figure 3 displays a scatter plot of all studies differentiated by the sleep measurement tool (subjective, objective or sleep deprivation), with each study assigned a numeric values based on its effect size (from -3 indicating strong reduction of stress to 3 indicating strong increase in stress, with null effect being 0; among the very few animal studies that yielded two opposite effects, each effect was treated as a separate study for simplicity). A clear pattern was evident, with studies using subjective sleep measures showing the most consistent sleep-protective effects, followed by studies using objective measures that occasionally yield null or exacerbating (i.e., opposite of protective) effects of sleep on stress, and finally sleep deprivation studies yielding the highest heterogeneity of results with both protective and exacerbating effects of sleep being almost equally prevalent.



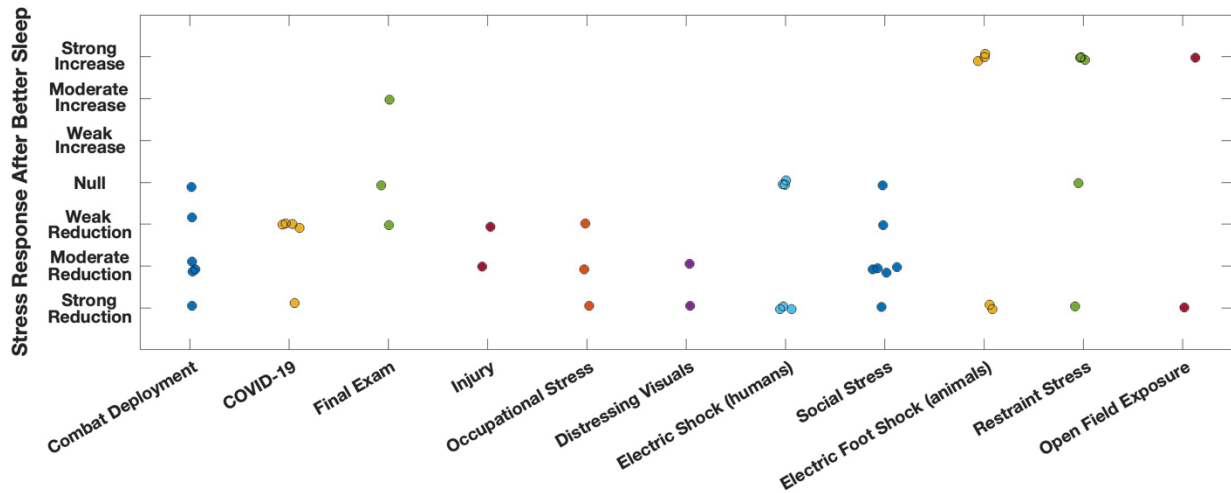
**Figure 2:** Harvest plot of studies included in the review. Each bar represents a study, with its location corresponding to the study design (sleep measuring method, stressor type and number of nights) and the reported sleep effect (protective from stress, exacerbating stress, or null effect). Blue bars correspond to human studies and red bars to animal studies. Three studies produced effects requiring representation by two bars, in which case a letter above the corresponding bars indicates the pairing (L - Lerner et al., 2017, including one single-night and one multiple-night experiments; R – Radwan et al., 2021, S – Sgoifo et al., 2006, including both protective and exacerbating effects). Height of the bar represents the strength of the evidence (strong, medium, weak). For studies with significant effects, this was determined based on effect size, whereas for null effects this was determined using equivalence testing against the smallest effect size of interest (see Table 1 and Supplementary Table S1).

Another notable pattern appeared when contrasting sleep deprivation studies between humans and animals (compare blue and red dots on the right column of Figure 3). Whereas almost all of the eight human sleep deprivation studies suggested a protective effect of baseline sleep on future stress, animal studies showed much more varied results and even a slight tendency toward an exacerbating effect of sleep. This difference partly stemmed from a relatively large number of animal studies measuring stress using HPA response, which consistently showed elevated reaction with more sleep (Machado et al., 2018; Meerlo et al., 2002; Oyola et al., 2019; Sgoifo et al., 2006). Nevertheless, at least two more animal studies using different stress measures showed the same effect (Ruskin & LaHoste, 2009; Pinho et al., 2013) while the one human sleep deprivation study measuring HPA response showed a protective effect (Minkel et al., 2014), in line with the rest of the human sleep deprivation studies and contrasting with the animal sleep deprivation studies. Therefore, it is possible that sleep deprivation, at least with current protocols, differs in its relationship with future stress response across species (however, see Discussion for alternative explanations).



**Figure 3:** The relation between baseline sleep and future stress response as a function of sleep measurement method and species. Stress response is categorized based on effect size, ranging from Strong Reduction (i.e., reduced stress response due to more/better sleep) to Strong Increase (increases stress response following more/better sleep; see Table S1 for how effect size was determined for each study). Each dot represents a study (green – human study; red – animal study). Dots were plotted with small jitter to allow better visibility. Each of the few animal studies that yielded two contrasting effects are treated as two separate studies for simplicity. SD - sleep deprivation.

Finally, we examined whether stressor type had any influence on the outcome. Figure 4 compares effects of stressors that appeared in at least two studies (to avoid reaching conclusions based on single experiments). Among the real-life stressors, protective effects were consistently observed across combat deployment, COVID-19, occupational stress, and injury, whereas one notable exception was test anxiety, which yielded mixed results. Among human lab stressors, electric shock yielded mixed results whereas social stress tasks like the TSST almost consistently showed protective effects. Lastly, in animal studies, all three types of stressors – electric foot shocks, open field exposure and restraint stress tests – yielded very disparate results, often showing either strong increase or strong reduction of the stress response following better sleep.



**Figure 4:** The relationship between baseline sleep and future stress response as a function of stressor type. Categories of stress response are the same as in Figure 3. Each dot represents a study (only studies employing stressors that were used in at least two publications are included). Dots were plotted with small jitter to allow better visibility. One animal study that yielded two contrasting effects is plotted as two separate studies for simplicity. The three rightmost stressors are animal studies, the rest are human studies.

#### 4. Discussion

Overall, our review suggests that baseline sleep was a robust predictor of future stress vulnerability across species, study designs and stressor types, with most studies showing that poor sleep quality predicts worse stress outcomes. These findings align with theoretical models, such as the REM recalibration hypothesis, which posits that REM sleep modulates noradrenergic tone to recalibrate emotional reactivity and render stress-encoding centers in the brain, such as the amygdala, to become less sensitive and more selective in their response (Goldstein & Walker, 2014); and the “sleep to forget, sleep to remember” model, which proposes that REM sleep helps decouple the emotional tone from memory consolidation (Walker, 2009). Our results indicate that those models, which were mainly conceived to account for the effect of sleep after exposure to a stressor, can be extended to consider sleep as a preparatory modulator of stress response, not just a recovery mechanism. Nevertheless, this general finding was more common for some study types than others, as detailed in Table 3 and explored in depth below.

**Table 3.** Summary of main findings

Effect	Supportive Evidence
Overall, better sleep is most commonly associated with reduced future stress response	36/51 of studies showing protective effects, rest divided almost equally between exacerbating and null effects (Figure 2; section 3.2)
Protective effects mostly evident in humans when sleep is measured subjectively through self-reports	19/20 studies with subjective sleep reports show protective effects (Figure 2, 3; section 3.4)
Associations between REM sleep and future stress response are variable across studies	Among studies examining habitual REM sleep and stress, 5, 4 and 4 showed null, exacerbating, and protective effects respectively (Section 3.3)
In humans, better sleep might not be as protective against anticipatory stress like test-anxiety.	4 studies using anticipatory stress (test-anxiety, childbirth) found baseline sleep to be weakly or not protective at all (Figure 4; Section 3.4)
In animals, habitual sleep often has strong protective or exacerbating effects on future stress. Exacerbation is common when stress is indexed by neuroendocrine measures.	9/10 animal studies showed strong protective or exacerbating effects on future stress, with all 4 studies using HPA-axis stress measures showing exacerbation (Figure 3; Section 3.4).

#### 4.1. Subjective Versus Objective Habitual Sleep Measures and their Relation to Future Stress

Among studies where habitual sleep was measured subjectively, better sleep consistently indicated stress protection. However, it is important to note that since most of the studies using subjective sleep measures were also longitudinal studies examining real-life stressors (18 of 20), it is possible that other elements (study design, stressor type) were the true driving force behind the effect. Still, several factors speak against this possibility (see harvest plot in Figure 2). First, among the studies examining real-life stress, two of the three studies using objective measures of sleep (Gruber et al., 2021; Larios & Lerner, 2024; Simon et al., 2024) did not find a protective effect, a clear divergence from the consistent pattern and one that more resembles the varied effects found in studies using objective sleep measures to predict responses to experimental stressors (Figure 2, middle column). At the same time, among studies implementing a lab-design with an experimental stressor, the two studies measuring sleep subjectively (Grove et al., 2023; Short & Schmidt, 2018; Figure 2, left column, lower row) were consistent with the protective pattern, finding a medium-size stress reduction effect following better sleep despite the overall trend of these lab-based studies to yield mixed results. Another potential confound was the type of baseline sleep targeted, either habitual sleep (i.e., regular day-to-day sleep over a period of time) or single-night sleep just prior to stressor exposure. Since most studies employing subjective sleep measures also targeted habitual sleep (for example, using a survey asking participants about their sleep habits over the last month or recording sleep diaries for a week), it

is possible that this factor was driving the results. However, once again, this possibility is undermined by the fact that quite a few studies used objective measures to target habitual sleep as well (i.e. employing actigraphy or mobile EEG for a week or more; Bottary et al., 2020; Casement et al., 2019; Gruber et al., 2021; Lerner et al., 2017; Larios & Lerner, 2024; Simon et al., 2024; Wright et al., 2007; see Figure 2, middle column, upper and lower rows), with far more varied results. Finally, another potential alternative explanation was the number of subjects tested: the majority of studies implementing subjective sleep reporting were also examining large cohorts (e.g., 100 participants or more; see Table 1); still, there were several exceptions to this rule (e.g., Gruber et al., 2021; Grove et al., 2023), and among the studies with large cohorts, there was no consistent relation between the size of the cohort and effect size (e.g., no indications of larger cohorts → stronger protective effect of sleep). Therefore, among all factors considered, measuring sleep through subjective evaluation was the most consistent in predicting sleep-induced protection against stress.

Why would such a difference between subjective and objective sleep measures emerge to begin with? Potentially, it might reflect the fact that objective physiological metrics could overlook important personal aspects such as perceived sleep quality, and these aspects may be more predictive of the emotional reserves available for the individual to cope with stress when it arrives. For example, it is not uncommon for individuals to perceive their sleep as insufficient despite their objective measures showing standard values (Kaplan et al., 2012; Lehrer et al., 2022; McCall et al., 1995), and this perception in and of itself may drive them to a poor emotional response when facing stressors. Moreover, since subjective habitual sleep measures reflect an individual's ongoing appraisal of their sleep, their sense of restfulness, and their confidence in their ability to recover overnight, these evaluative components may more closely track the psychological resources that determine stress resilience. Subjective sleep reports may also implicitly capture daytime functioning such as fatigue, general mood and attentional capacity, which are tightly linked to the stress system (Edinger et al., 2015). In contrast, objective metrics primarily index total sleep and sleep-stages' quantity and continuity, which may not fully capture the qualitative aspects of sleep that are most relevant for how the body and mind respond to stress, and are also less likely to reflect daytime functioning. In this sense, subjective sleep quality may act as a proximal summary of both nighttime experience and daytime consequences, offering a more holistic indicator of the individual's current regulatory

state. Additionally, subjective sleep measures tend to be more trait-like than objective measures. Instruments such as sleep quality questionnaires often target stable patterns in sleep behavior and perception over weeks or months. Objective sleep, on the other hand, even when measured over multiple nights, can fluctuate markedly across days due to idiosyncratic factors unrelated to inherent stress resilience (e.g., transient environmental noise, atypical schedules, or even short-term mood variation), potentially diluting its predictive value. This broader coverage of subjective habitual sleep measures could explain why, across heterogeneous study designs and stressor types, it emerges as the more consistent predictor of subsequent stress reactivity.

Nevertheless, it is important to remember that subjective sleep measures also have clear limitations. Chief among them is the fact that they are collected retrospectively, either through surveys asking participants how their sleep has been over the past several weeks, or using sleep diaries filled out in the morning following overnight sleep. These retroactive assessments are susceptible to memory errors and could be influenced by participants' thoughts about their sleep rather than directly measuring it, making them imperfect proxies of actual sleep. Moreover, reliance on questionnaires could in and of itself explain why subjective assessments are more correlated with future stress measurements than objective measures, as many studies that employ subjective sleep measures use questionnaires to collect stress measurements. Consequently, correlations between stress and sleep measured through questionnaires could simply reflect shared method variance (i.e., a correlation between different measures that stems from similarities between the tools used to collect them, often stemming from the two measures being affected by a third factor – like mood or response style affecting both sleep and stress questionnaires), rather than a direct effect of one measure on another.

#### *4.2. The Role of Stressor Type in Moderating Baseline Sleep–Stress Associations*

Overall, stressor type was not a strong modulator of the sleep-stress relationship (see Figure 4), with the majority of studies showing the same pattern: In humans, the response to almost all stressors was predominantly reduced following better sleep, whereas in animals the stress response for the three stressor types repeatedly used (foot shock, restraint, open field exploration) was either strongly reduced or strongly elevated after better sleep. However, within the human studies, this general pattern is qualified by two observations.

First, when real-life settings are considered, although the protective sleep effect was found across many natural stressor types like childbirth, combat deployment, COVID-19, and injury, test anxiety seems to be an exception and was not consistently associated with worse sleep whether reported by objective or subjective measures. Although based on only three studies, it is tempting to speculate that this pattern stems from one unique property of test anxiety compared to most other natural stressors (with the exception of childbirth): its predictability. Potentially, this property, which characterizes the stress caused by tests as anticipatory, could result in the involvement of different types of coping mechanisms that are less affected by sleep compared to non-anticipatory stress. It is noteworthy that the one other study using an anticipatory real-life stressor, childbirth, also found only a weak, indirect protective effect of sleep (Deforges et al., 2021).

Second, under experimental settings, the type of stressor did seem to make a difference when taking into consideration the specific method used for stress measurement. Fear-conditioning tasks employing electric shocks consistently yielded a protective effect of sleep on amygdala activity (Lerner et al., 2017; Feng et al., 2018a, b; 2023) but not on other physiological measures of stress like SCR and eyeblink startle (Bottary et al., 2020; Marshall et al., 2014; Peters et al., 2014). In contrast, studies that used social-evaluative tasks, such as the TSST, consistently showed protective effects of sleep for nearly all physiological and behavioral measures tested (though note that brain activity was not examined with this paradigm). This pattern of results suggests that some stress-response systems may be more sensitive to sleep than others. Specifically, the sympathetic and HPA system appear more affected by sleep in anticipatory psychosocial contexts (e.g., facing a speech test as part of the TSST task) than in phasic, physical stress contexts (e.g., electric shock). Taking together, these observations highlight the need to re-examine the impact of baseline sleep on test anxiety using physiological measures in conjunction with subjective measures, given the anticipatory nature of test-related stress. More broadly, these findings underscore the importance of understanding the effects of sleep on bottom up stressors (e.g. physical threats) versus top down stressors (threat to identity or performance). Rather than a simple hierarchy of stressor types, these results point to potentially divergent sleep-stress pathways that may operate through distinct mechanisms.

### *4.3. Sleep Deprivation Prior to Stress*

Nearly all human sleep deprivation (SD) studies linked SD to increased neural, physiological, or emotional stress responses following exposure to stressor, including heightened amygdala reactivity, blood pressure, and cortisol (Feng et al., 2023, Franzen et al., 2011; Minkel et al., 2024). In addition, partial SD was associated with more system-specific effects, like elevated alpha amylase, but no change in fear learning (O’Leary et al., 2015; Peters et al., 2014). In contrast, animal studies often showed the opposite effects, with the most consistent result being a reduction of stress-induced HPA response following SD (Machado et al., 2008; Meerlo et al., 2002; Oyola et al., 2019; Sgoifo et al., 2006). Thereby, while in humans SD might act as an amplifier of stress systems, in animals SD may induce neuroendocrine fatigue or blunting. Nevertheless, some of these discrepancies could be due to the variation, length and chronicity in SD methods, where human SD was typically acute (typically 1 night of total or partial SD) whereas animal SD studies ranged from 24h total SD to 72h RSD or chronic sleep deprivation periods. Therefore, longer or more intense SD in animals may trigger compensatory downregulation of stress systems compared to humans. Another possibility is that differences in stressor type and intensity contributed to the discrepancy in results across species: Whereas human stressors included weaker manipulations like mild electric shock or social-evaluative stress, animal studies used foot shocks and restraint stress that yield strong physical threat. Finally, given that many of the stress measures in animals focused on HPA responses, it is possible that this system in particular is more prone to blunting following SD compared to other systems such as amygdala activity or behavioural response (though note that the one study examining HPA response in humans did find increases following SD, in line with the main pattern in humans using different measures).

### *4.4. Effects of REM Sleep (Observed or Manipulated)*

In the thirteen studies that examined baseline REM sleep, results were quite varied with 31% suggesting a protective role against future stress, 31% demonstrating increased stress response with more REM, and 38% showing no association (See Table 2). These findings, and particularly the variation in the direction of effects once they were found, is clearly different than

the overall pattern of results found for baseline sleep across the entire reviewed literature. This variation matches similarly mixed results found in studies examining how REM sleep following exposure to emotional stimuli affects fear recall and reactivity (e.g., Gujar et al., 2011; Menz et al., 2013, 2016; Wagner et al., 2002). Moreover, even the few studies covered in this review that examined, in addition to baseline REM sleep, how REM sleep *after* the exposure to stressor affected stress response, found mixed results (Bottary et al., 2020; Machado et al., 2008; Marshall et al., 2014; Polta et al., 2013; Radwan et al., 2021). In short, REM sleep often affects stress response in both protective and amplifying directions, whether measured before or after stress exposure. Given the methodological differences across studies, it is difficult to come to strong conclusions regarding these discrepancies; however, it is tempting to see these findings as aligning with recent perspectives claiming REM sleep serve functions that go far beyond the reduction of emotional tone attached to experiences. Specifically, it was previously shown that REM sleep following fear conditioning could both increase and decrease the fear response to stimuli resembling the ones exposed to during acquisition, depending on how predictive they were of upcoming threat (Lerner et al., 2021). Similarly, a recent model proposed that REM sleep assists in refining encoded memories and rescuing weak memories from elimination, thus suggesting that REM sleep may affect the consolidation of memories, both neutral and stressful, in more than one way (Shuster et al., 2025). While these studies focused on the effect of REM sleep on already-encoded memories, it is reasonable to hypothesize that baseline REM sleep occurring before encoding (e.g., before the exposure to a stressor, as is the focus of this review) could have a divergent effect on the initial response to new experiences as well. In other words, a trait-level tendency for having more or less habitual REM sleep may prone the individual to higher or lower sensitivity to stressful experiences, depending on the specific characteristics of the experience. The exact nature of these characteristics, which may depend on the predictability of the threat, remain to be explored in future studies.

#### *4.5 Limitations and Future Directions*

Although the prevailing pattern in the literature we reviewed suggests baseline sleep could provide a protective effect against heightened stress responses to future stressors, several caveats should be discussed. First, regarding the methodology of our review, it is important to

reiterate that our literature search was limited to peer-reviewed studies and did not include grey literature or preprints; the review was not pre-registered; and although our findings addressed effects sizes, we did not conduct formal meta-analysis due to substantial methodological heterogeneity across studies (including differences in populations, sleep assessments/manipulations, and stress measures) and because many reports did not consistently provide the variance estimates required for quantitative analysis. Future reviews may limit their focus on studies employing more homogeneous methodologies (e.g., animal sleep deprivation paradigms) to enable a pre-registered meta-analysis.

Second, as noted earlier, there was some risk of both internal validity and publication biases in the reviewed literature. With respect to publication bias, some concern emerged when examining the subset of papers in which the relationship between baseline sleep and future stress response was not the primary research focus: these studies tended to yield somewhat more null effects than the full set of studies. However, this evidence is weak, as only six studies fell into this category, and their overall pattern did not dramatically diverge from that of the remaining studies. Turning to internal validity bias, the study-by-study assessment showed that most studies were classified as having at least moderate risk, and, most importantly, half of the non-randomized trials exhibited an overall serious risk of bias. While such proportions are common in the broader literature (Igelström et al., 2021), in our review they were mainly concentrated in studies using subjective measures of sleep. This poses an interpretational challenge: the same studies that yielded the most consistent evidence for a protective effect of habitual sleep are also those most vulnerable to confounding and selective reporting. Consequently, the risk of bias assessment suggests that while the association between sleep and future stress response appears robust, the strength and consistency of the protective effects observed in subjective sleep studies should be interpreted with caution. This does not necessarily imply that the observed associations are spurious, but rather reflects the need for future pre-registered longitudinal studies with pre-specified analytic plans, comprehensive reporting of outcomes, stronger control of confounders, and combined subjective-objective sleep assessment to more definitively establish the strength and specificity of baseline sleep as a predictor of subsequent stress responses.

Third, although several factors seem to contribute to deviations from the general protective pattern, these are yet to be investigated systematically. While some studies incorporated multiple measures, none have focused on comparing them in the context of baseline

sleep and subsequent stress responses, and future research making such explicit comparisons is warranted. These include: (a) comparing outcome measures building on anticipatory stress, like test anxiety, to non-anticipatory stressors; (b) contrasting objective versus subjective measures of baseline sleep; and (c) comparing the impact of human and animal sleep deprivation protocols on future stress response, while controlling for deprivation methods, stressor intensity, and the stress outcome measure (e.g., HPA-axis related or not). In addition, emerging metrics such as sleep regularity, which has recently been linked to health and mortality outcomes (e.g., Windred et al., 2024), are yet to be explored in relation to stress responses and could be readily integrated into future studies.

Finally, it should be noted that our review revealed that some studies collect baseline sleep data as part of standard protocols but omit analyses linking it to stress outcomes when unrelated to their primary goals. We believe that analysis of such relationships should become routine whenever relevant data is available.

## **5. Conclusion**

In the current review – to our knowledge, the first of its kind – we showed that, overall, baseline sleep prior to an exposure to a stressor can act as a key predictor of subsequent stress response. While various factors such as stressor type and the assessment tools used to measure sleep and stress may influence the direction of the effect, the synthesis of human and animal studies reviewed here highlights baseline sleep as an often-overlooked determinant of stress resilience. In particular, humans' subjective perception of their own habitual sleep quality emerges as an accessible behavioral marker that may help forecast vulnerability to stress long before exposure occurs. This finding could have practical or even clinical implications. For example, brief, routine subjective sleep assessment (e.g., monthly administration of a short insomnia/sleep-quality screener) could be integrated into existing occupational health workflows of populations at risk for exaggerated stress responses such as military personnel, healthcare workers, and emergency responders, to flag individuals whose baseline sleep falls below clinically meaningful thresholds. These data could be used to trigger additional follow-ups including a focused clinical interview, targeted monitoring during subsequent high-stress intervals, or referral for a sleep evaluation when warranted. Beyond screening, individuals identified as higher risk could be

offered low-burden interventions (e.g., guidance for structured sleep scheduling or cognitive-behavioral therapy for insomnia) before major stress exposures. Because sleep is modifiable, relatively low-cost, and scalable, such targeted implementation could provide a practical pathway for strengthening adaptive capacity and reducing the likelihood of exaggerated stress responses in the face of predictable stressors. Turning to future research, while the association between baseline sleep and stress has been more variable when evaluated using objective sleep measures, sleep deprivation, or in general when tested in animals, these inconsistencies may stem from divergent neurobiological pathways linking sleep and stress regulation across species and experimental models. Integrating endocrine, neural, and behavioral measures across human and animal studies might be essential to identify shared mechanisms and improve translational validity, while simultaneously refining measurement and modeling approaches to enhance standardization of paradigms and assessment tools. Taken together, our findings underscore the need to treat sleep not merely as a recovery state but as a proactive target for stress prevention and resilience-building.

#### **Data statement**

All data analyzed in this review are from previously published studies and are publicly available through the cited sources.

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**Table 1.** Summary of included studies by stressor type.

Study	Design	Subjects (N female, % ethnic/racial minority)	Baseline Sleep Measurement	Stressor	Main outcome of sleep's effect on subsequent stress response
<b>Human Studies: Associations between Habitual Sleep and Response to a Future Real-life Stressor</b>					
Deforges et al., 2021	Longitudinal	1,160 pregnant women (1,160 F)	Self-report	Childbirth	Prenatal sleep quality → risk factor for CB-PTSD development ↓
Acheson et al., 2019	Longitudinal	2,404 male military personnel (0 F)	Self-report <sup>a</sup>	Combat deployment	Sleep quality → re-experiencing symptoms ↓
Gehrman et al., 2013	Longitudinal	15,204 military personnel (3,079 F, 29.7%)	Self-report	Combat deployment	Sleep quality → anxiety and PTSD diagnoses ↓↓
Koeffel et al., 2013	Longitudinal	172 National Guard Soldiers (60 F)	Self-report	Combat deployment	Sleep quality → PTSD symptoms and diagnosis ↓
Simon et al., 2024	Longitudinal	20 male combat soldiers (0 F, 0%)	Fitbit for 1 week	Combat deployment	Sleep duration and efficiency → PCL-5 scores ×
Van Liempt et al., 2013	Longitudinal	453 military personnel (32 F)	Self-report	Combat deployment	Sleep quality → PTSD diagnosis ↓
Wang, et al., 2019	Longitudinal	4,645 military personnel (246 F, 43.9%)	Self-report	Combat deployment	Sleep quality → PTSD diagnosis ↓
Bauducco et al., 2024	Longitudinal	2,781 adolescents (1,473 F)	Self-report	COVID-19 pandemic	Sleep efficiency, sleep quality, and total sleep time → overall anxiety symptoms ↓
Gruber et al., 2021	Longitudinal	62 adolescents (45 F, 12.3%)	Actigraphy, sleep logs for 7 nights	COVID-19 pandemic	Sleep duration & efficiency (Actigraphy) → perceived stress ↓↓
Kiss et al., 2022	Longitudinal	3,193 adolescents (1,562 F, 19.4%)	Parent/caregiver reports	COVID-19 pandemic	Sleep quality → psychological distress ↓
Wang et al., 2022	Longitudinal	1,790 adolescents (880 F, 0%)	Self-report	COVID-19 pandemic	Sleep quality → anxiety symptoms ↓
Wang et al., 2024	Longitudinal	7900 children (3820 F, .81%)	Self-report	COVID-19 pandemic	Sleep duration → anxiety ↓
Hamilton et al., 2021	Single Group	167 college students (125 F, 26%)	Sleep log for 6 days	Final exam in statistics	Sleep quality → test anxiety ↓

Study	Design	Subjects (N female, % ethnic/racial minority)	Baseline Sleep Measurement	Stressor	Main outcome of sleep's effect on subsequent stress response
Larios & Lerner, 2024	Longitudinal	52 college students (31 F, 55.8%)	5-day Mobile EEG at start of semester	Final exams	%REM → test anxiety ↑
Nordberg et al., 2022	Longitudinal	637 college students (439 F, 26.2%)	Self-report	Final exams	Habitual sleep quality → acute stress before exam ×
Lucas-Thompson et al., 2008	Longitudinal	92 mother-infant dyads (92 F, 48%)	Mother reports	Infant inoculation	Sleep continuity → cortisol reactivity ↓
Bryant et al., 2010	Longitudinal	898 injured, traumatic patients (289 F, 12%)	Self-report <sup>b</sup>	Traumatic injury	Sleep quality → psychiatric disorder diagnoses ↓
Neylan et al., 2020	Survey Data	666 adults (455 F)	Self-report <sup>c</sup>	Motor vehicle accident	Sleep quality and sleep efficiency → PTSD diagnosis ↓
Kalmbach et al., 2015	Longitudinal	91 workers (60 F, 34.4%)	Self-report	Shift work	Sleep resilience → risk for shift work disorder development ↓↓
Kalmbach et al., 2019	Longitudinal	1,336 medical interns (600 F, 38.2%)	Self-report	Occupational stress	Sleep quality / efficiency → anxiety levels / vulnerability to anxiety ↓
Wolkow et al., 2024	Police officers vs. Control	342 police officers and 85 control (107 F)	Self-report	Police work	Sleep difficulties → stress and in police officers ↓
<b>Human Studies: Associations Between Habitual Sleep and Response to a Future Experimental Stressor</b>					
Alkalame et al., 2024	Single Group <sup>d</sup>	27 healthy adults (15 F)	Actigraphy and sleep log for 7 days, then in-lab PSG for 4 days.	Trauma Film	REM efficiency, % REM, TST → intrusive memories ↓
Bottary et al., 2020	Insomnia Patients vs. Healthy Control	48 adults (36 F)	Actigraphy for 14 days	Electric shock	Sleep quality → fear conditioning ×
Lerner et al., 2017	Single Group per experiment <sup>e</sup>	34 college students (10 F)	Mobile EEG / PSG for 1 week / 1 night	Electric shock	REM duration, %REM → fear-related brain activity during conditioning ↓↓
Marshall et al., 2014	Single Group	42 adults (17 F, 37%)	EEG for 2 nights	Electric shock	Change in REM between 2 nights → stress during fear acquisition ×
Wright et al., 2007	Single Group	53 women (37.3%)	Actigraphy, sleep log for 7 days	Mental Stress Task	sleep quality (Actigraphy) → cortisol reactivity ↑

Study	Design	Subjects (N female, % ethnic/racial minority)	Baseline Sleep Measurement	Stressor	Main outcome of sleep's effect on subsequent stress response
Short & Schmidt, 2018	Sleep Habits Survey	99 college students (77 F, 33.1%)	Self-report	Speech task	Sleep quality → anticipatory anxiety/reactivity ↓
Brand et al., 2018	Single Group	39 children (14 F)	In-home EEG for 1 night	TSST	Sleep parameters → cortisone reactivity ×
Casement et al., 2019	Bipolar Diagnosis vs. Healthy Control	49 adolescents (33 F, 26.5%)	Actigraphy up to 14 days	TSST	Moderate sleep duration → cardiac-based vulnerability to stress ↓
Grove et al., 2023	NSSI vs. Healthy Controls	72 adults (47 F, 22%)	Self-report	TSST	Sleep quality → worrisome stress response in those at risk for NSSI ↓
Massar et al., 2017	Single Group	59 adult males (0 F)	Actigraphy for 7 consecutive days	TSST	Sleep efficiency → reactivity to acute stress, HPA-axis and cardiovascular reactivity ↓

#### Human Studies: Effects of Total/Partial Sleep Deprivation on Response to a Future Experimental Stressor

Goldstein et al., 2013	SD vs. Control within-subject	18 healthy adults (9 F)	24h PSG monitored SD	Emotionally negative images	Amygdala activity anticipating threat in sleep vs. SD ↓↓
Feng et al., 2018a, 2018b	SD vs. Control	70 college students (27 F)	24h researcher monitored SD	Electric shock	Fear memory during acquisition, amygdala activation during consolidation in sleep vs. SD ↓↓
Feng et al., 2023	SD vs. Control	68 college students (33 F)	24h researcher monitored SD	Electric shock	SCR and amygdala activity for fear acquisition in sleep vs. SD ↓↓
Peters et al., 2014	PSD vs. Control	32 adults (17 F)	24h Actigraphy monitored PSD	Electric shock	Physiological fear response to shocks in sleep vs. PSD ×
Yang et al., 2012	SD vs. Control	28 healthy adults (14 F); 30 healthy adults (15 F)	24h researcher monitored SD	Mental stress + cold pressor test	Increased heart rate during stressor and recovery in sleep vs. SD ↓
Franzen et al., 2011	SD vs. Control within-subject	20 adults (11 F, 40%)	24h PSG monitored SD	Stress protocol <sup>f</sup>	Increased Systolic blood pressure following stress in sleep vs. SD ↓↓
Minkel et al., 2014	SD vs. Control	26 healthy adults (12 F, 53.8%)	24h Actigraphy monitored SD	TSST	HPA response to stress in sleep vs. SD ↓↓
O'leary et al., 2015	SD vs. Control	108 college students (80 F)	7-day Sleep log, 24h actigraphy monitored PSD	TSST	Basal Salivary $\alpha$ -Amylase activity (sAA) and sAA immediately following stress in sleep vs. SD ↓

#### Animal Studies: Associations Between Habitual Sleep and Response to a Future Experimental Stressor

Study	Design	Subjects (N female, % ethnic/racial minority)	Baseline Sleep Measurement	Stressor	Main outcome of sleep's effect on subsequent stress response
Radwan et al., 2021	Stress Resilient vs. Stress Susceptible <sup>g</sup>	48 male mice (0 F)	EEG and EMG for 1 day	Chronic Social Defeat	Time in NREM (and TST) → stress vulnerability following stressor ↑↑ NREM continuity → stress vulnerability following stressor ↓↓
Polta et al., 2013	Shock vs. Control	16 male mice (0 F)	EEG and EMG for 1 day	Electric foot shocks	REM continuity → acoustic startle response 1 month after shock exposure ↓↓
<b>Animal Studies: Effects of Total/Partial Sleep Deprivation on Response to a Future Experimental Stressor</b>					
Jones et al., 2020	ELSD vs. Control	130 prairie voles (55 F)	ELSD 14 to 21 days after birth <sup>h</sup> .	Electric foot shocks	Ethanol intake, amygdala activity after shocks in sleep vs. ELSD ↓↓
Machado et al., 2008	RSD vs. Control	27 rats (0 F)	96h EEG monitored RSD	Electric foot shocks	HPA response to single footshock <sup>i</sup> in sleep vs. RSD ↑↑
Pinho et al., 2013	SD vs. Control	84 male rats (0 F)	72h researcher monitored SD	Electric foot shocks	Freezing behavior and amygdala pCREB levels in sleep vs. SD ↑↑
Ruskin & LaHoste, 2009	SD vs. Control Shock vs. Control	18 male mice (0 F)	24h researcher monitored SD	Electric foot shocks	Contextual freezing behavior in sleep vs. SD ↑↑
Hakimeh & Vahid, 2017	SD vs. Control vs. Exercise vs. Exercise/SD vs. Wide platform Vs. Sham exercise	48 female rats (48 F)	72h researcher monitored SD	Open field exposure	Anxiety behavior in sleep vs. SD ↓↓
Hicks & Moore, 1979	2 day RSD vs. Control 4 day RSD vs. Control	44 male rats (0 F)	2 and 4 days researcher monitored RSD	Open field exposure	Exploration-fear emotionality in sleep vs. RSD ↑↑
Meerlo et al., 2002	SD vs. Control PSD vs. Control	54 male rats (0 F)	48h SD / 160h PSD	Restraint stress	HPA response to a novel stressor in sleep vs. SD ↑↑
Oyola et al., 2019	RSD vs. Control RS vs. Control	28 mice (16 F)	12h researcher monitored RSD	Restraint stress	Corticosterone reactivity in sleep vs. RSD females ↑↑
Sgoifo et al., 2006	SD vs. Control	40 male rats (0 F)	48h researcher monitored SD	Restraint stress	cardiac-based vulnerability to stress in sleep vs. SD ↓↓

Study	Design	Subjects (N female, % ethnic/racial minority)	Baseline Sleep Measurement	Stressor	Main outcome of sleep's effect on subsequent stress response
Davis et al., 2021	SPS vs. SD vs. SPS + SD vs. Control	41 male rats (0 F)	12h EEG+EMG monitored SD	SPS	HPA response to stress in sleep vs. SD ↑↑ Fear-associated memory impairments in sleep vs. SD ×

Note: † ↑ †† Better baseline sleep associated with higher stress response; ‡ ↓ ‡‡ Better baseline sleep associated with lower stress response; dashed arrow – small effect size; full arrow – medium effect size; double arrow – strong effect size ; × No significant association between baseline sleep and stress response. Self- (or parent's) reports refer to questionnaires prompting participants to subjectively evaluate their (or their child's) sleep retrospectively for a predefined period of time. Percentage of ethnic/racial minority is indicated for human studies when the information was available. CB-PTSD=childbirth post-traumatic stress disorder, EEG=electroencephalography, ELSD=early-life sleep disruption, F=female, HPA=hypothalamic–pituitary–adrenal axis, NSSI=non-suicidal self-injury, pCREB = phosphorylation of Cyclic adenosine monophosphate responsive element binding protein, PCL-5=PTSD checklist for DSM-5, PSD=partial sleep deprivation, PSG=polysomnography, PTSD=post-traumatic stress disorder, REM=rapid eye movement sleep, %REM = percentage of REM sleep out of total sleep time, NREM = non-REM sleep, RS=restraint stress, RSD=REM-sleep deprivation, SCR = skin conductance response, SD=sleep deprivation, SPS=single prolonged stress, TSST=Trier Social Stress Test, TST=total sleep time

<sup>a</sup>: Sleep habits assessed during clinical interviews.

<sup>b</sup>: Sleep habits for 2 weeks prior to traumatic injury retroactively assessed after traumatic injury occurred.

<sup>c</sup>: Sleep habits prior to motor vehicle accident retroactively assessed after motor vehicle accident.

<sup>d</sup>: Participants were divided between a control group, a circadian disruption group provided melatonin, and a circadian disruption group provided a placebo, but analyzed as a single group for REM sleep effects.

<sup>e</sup>: Two Experiments. Exp. 1: Baseline sleep monitored through PSG headband 1 week before stressor. Exp. 2: Baseline sleep measured via PSG night before stressor.

<sup>f</sup>: Stress protocol consisted of four 10-minute periods: (1) resting baseline; (2) Stroop color-word naming interference task; (3) speech preparation and delivery; and (4) a recovery period.

<sup>g</sup>: Stress was measured by sorting mice into stress-resilient or stress-susceptible groups based on behavior following stressor exposure.

<sup>h</sup>: ELSD has been shown to lead to fragmented NREM and REM sleep.

<sup>i</sup>: Another experimental condition examined multiple foot shocks, but there was no clear temporal differentiation between their administration and the REM sleep manipulation.

## Supplementary Materials

### S1. Search Strategy

Below we detail the complete search strategies for each database included in the review. All searches were conducted independently using tailored Boolean operators and field tags. No registries, preprints, or grey literature sources were searched. Search queries were run up to and including August 2024, including truncations, Boolean logic, and filters.

Duplicates were removed using Covidence (Veritas Health Innovation, Melbourne, Australia) based on title, author, and DOI matching. After deduplication, 2,104 unique records remained.

#### PubMed (NCBI)

(sleep[Title] OR insomnia[Title]) AND (stress[Title] OR fear[Title] OR anxiety\*[Title] OR ptsd[Title] OR posttraumatic[Title] OR post-traumatic[Title] OR trauma\*[Title] OR predeployment[Title] OR pre-deployment[Title]) AND (prior[Title/Abstract] OR subsequent[Title/Abstract] OR retrospective\*[Title/Abstract] OR prospective\*[Title/Abstract] OR longitudinal[Title/Abstract] OR precede\*[Title/Abstract] OR biomarker[Title/Abstract] OR trait-level[Title/Abstract])

Records retrieved: 955

#### Web of Science (Clarivate Analytics)

(TI=(sleep OR insomnia)) AND (TI=(stress OR fear OR anxiety\* OR ptsd OR posttraumatic OR "post-traumatic" OR trauma\* OR predeployment OR "pre-deployment")) AND (TI=(prior OR subsequent OR retrospective\* OR prospective\* OR longitudinal OR precede\* OR biomarker OR "trait-level") OR AB=(prior OR subsequent OR retrospective\* OR prospective\* OR longitudinal OR precede\* OR biomarker OR "trait-level"))

Records retrieved: 2,029

#### PsycINFO (APA PsycNet)

(TI(sleep) OR TI(insomnia)) AND (TI(stress) OR TI(fear) OR TI(anxiety\*) OR TI(ptsd) OR TI(posttraumatic) OR TI("post-traumatic") OR TI(trauma\*) OR TI(predeployment) OR TI("pre-deployment")) AND (TI(prior) OR TI(subsequent) OR TI(retrospective\*) OR TI(prospective\*) OR TI(longitudinal) OR TI(precede\*) OR TI(biomarker) OR TI("trait-level") OR AB(prior) OR AB(subsequent) OR AB(retrospective\*) OR AB(prospective\*) OR AB(longitudinal) OR AB(precede\*) OR AB(biomarker) OR AB("trait-level"))

Records retrieved: 734

#### Embase (Elsevier)

(sleep:ti OR insomnia:ti) AND (stress:ti OR fear:ti OR anxiety\*:ti OR ptsd:ti OR posttraumatic:ti OR "post-traumatic":ti OR trauma\*:ti OR predeployment:ti OR "pre-deployment":ti) AND (prior:ti,ab OR subsequent:ti,ab OR retrospective\*:ti,ab OR prospective\*:ti,ab OR longitudinal:ti,ab OR precede\*:ti,ab OR biomarker:ti,ab OR "trait-level":ti,ab)

Records retrieved: 1,792

**Table S1.** Summary of effect size/strength of evidence determinations.

Study	Effect Size	Main outcome of sleep's effect on subsequent stress response
Acheson et al., 2019	Small	$r = .18$ (calculated from $\beta = .18$ using $r = B/\sqrt{B^2+1}$ )
Alkalame et al., 2024	Medium*	IRR = 0.73 per 1 SD of %REM (from paper)

Bauducco et al., 2024	Small	$\eta_p^2 = .013$ (calculated from $\chi^2(3,2733) = 108.52$ ); or Hedge's $g = 0.177$ (calculated from $M(SE) = 499(2.38)$ , $N = 2080$ vs $M(SE) = 479.75(7.86)$ , $N = 323$ ).
Bottary et al., 2020	None	Null findings, medium evidence strength based on $\beta = 0.007$ , $SD = 0.128$ , $CI = [-0.266, 0.251]$ (from paper)
Brand et al., 2018	None	Null findings, medium evidence strength based on REM sleep data: $r(26) = 0.05$ (from paper) with calculated $CI = [-0.33, 0.42]$ .
Bryant et al., 2010	Medium*	OR = 2.89 for PTSD or OR = 2.5 for any anxiety disorder (from paper)
Casement et al., 2019	Medium*	$\beta = -.27$ (from paper)
Davis et al., 2021	None	Null findings, weak evidence strength based on comparing SDS ( $72.99 \pm 9.5\%$ ) and SPS+SD ( $64.6 \pm 11.3\%$ ) groups (from paper), with calculated $d = 0.22$ and $CI = [-0.55, 0.77]$ .
Deforges et al., 2021	Small*	$f^2 = 0.0043$ (from paper)
Feng et al., 2018a, 2018b	Strong*	$r = 0.50$ (fear memory during acquisition, from paper; $r = 0.67$ (amygdala activation during consolidation in sleep vs. SD, from paper).
Feng et al., 2023	Strong*	$d = 0.77$ for SCR, $d = 0.84$ for amygdala activation (from paper)
Franzen et al., 2011	Strong*	$d = 0.73$ (from paper)
Gehrman et al., 2013	Strong*	OR = 4.33 for Group 1, OR = 4.14 for Group 2 for anxiety outcome (from paper)
Goldstein et al., 2013	Strong	$\eta_p^2 = .20$ (calculated from $F(2,34) = 4.15$ , condition $\times$ cue type interaction in right anterior insula).
Grove et al., 2023	Medium	$\beta = 1.11$ (from paper); $\beta_{simple} = .96$ , $t = 2.75$ , $p = .007$ (NSSI group, from paper)
Gruber et al., 2021	Strong*	$\Delta R^2 = .18$ (from paper)
Hakimeh & Vahid, 2017	Strong	$\eta_p^2 = .34-.36$ (calculated from $F(11,76) = 3.5-3.8$ for rearing and center time)
Hamilton et al., 2021	Small	$\beta = -.166$ (from paper)
Hicks & Moore, 1979	Strong	$\eta_p^2 = .37-.69$ (calculated from $F(3,40) = 7.92-29.94$ ; defecation, urination, freezing)
Jones et al., 2020	Medium	$\eta_p^2 = .048$ (calculated from $F(1,115) = 5.823$ , $p = .017$ )
Kalmbach et al., 2015	Strong*	OR=5.59 for FIRST $\rightarrow$ SWD and $\beta = 0.81$ for SWD $\rightarrow$ anxiety (from paper)
Kalmbach et al., 2019	Medium*	RR = 2.83 (from paper)
Kiss et al., 2022	Small	SHAP = 0.08 (from paper)
Koeffel et al., 2013	Medium*	$r = .17 - 0.4$ (from paper)
Larios & Lerner, 2024	Medium*	$r = 0.34$ (from paper)
Lerner et al., 2017	Strong	$r = -.78$ (from paper)
Lucas-Thompson et al., 2008	Medium	$r = .27$ (6-month night waking $\rightarrow$ cortisol reactivity, from paper); $r = .29$ (12-month night waking $\rightarrow$ post-inoculation cortisol, from paper).
Machado et al., 2008	Strong*	$r = 0.83$ (from paper)
Marshall et al., 2014	None	Null findings, CI unavailable, strong evidence based on $p = 0.98$ (from paper).
Massar et al., 2017	Medium*	$r = -0.27$ for sleep efficiency % and salivary cortisol, blood pressure reactivity, $r = -0.34$ , blood pressure recovery, $r = -$

		0.31, HR reactivity, $r = -0.03$ , and HR recovery, $r = -0.04$ (from paper)
Meerlo et al., 2002	Strong	$\eta_p^2 = .15$ (calculated from $F(2,56) = 4.80, p = .012$ ); $\eta^2 = .167$ (interaction effect, calculated from $F(2,44) = 4.42, p = .018$ ).
Minkel et al., 2014	Strong*	$d = .89$ and $d = 1.15$ for increased cortisol response five minutes and twenty minutes after stressor, respectively (from paper)
Neylan et al., 2020	Small*	OR = 1.3–1.5 (from paper).
Nordberg et al., 2022	None	Null findings, medium evidence strength based on: $\beta = -0.05$ , $SE = 0.1$ (from paper), with calculated $CI = [-0.25, 0.15]$ .
O’leary et al., 2015	Small*	$\eta_p^2 = .038$ (from paper)
Oyola et al., 2019	Strong	$\eta_p^2 = .225$ (calculated from $F(1,26) = 7.54$ ).
Peters et al., 2014	None	Null findings, CI unavailable, medium evidence based on $p > 0.1$ (from paper)
Pinho et al., 2013	Strong	$\eta_p^2 = .18$ (calculated from $F(3,44) = 3.18, p = .033$ )
Polta et al., 2013	Strong*	$r^2 = 0.62$ (from paper)
Radwan et al., 2021	Strong	$\eta_p^2 = .163$ (calculated from $F(22,202) = 1.79, p < .05$ )
Ruskin & LaHoste, 2009	Strong	$\eta_H^2 = 0.633 - 0.947$ (calculated from $H = 11.5 - 16.2, k = 3, N = 18$ ).
Sgoifo et al., 2006	Strong	$\eta_p^2 = .193$ (calculated from $F(3,54) = 4.3, p < .02$ ).
Short & Schmidt, 2018	Medium	$sr^2 = .07-.08$ (from paper).
Simon et al., 2024	None	Null findings, CI unavailable, medium evidence based on $p = 0.402$ (from paper)
Van Liempt et al., 2013	Medium*	OR = 2.992 (from paper)
Wang et al., 2022	Small*	standardized $\beta$ estimate = 0.061 (at T2, from paper); standardized $\beta$ estimate = 0.117 (at T3, from paper)
Wang et al., 2024	Small*	$\beta = -0.05$ (from paper)
Wang, et al., 2019	Medium*	Adjusted OR = 3.14 (from paper)
Wolkow et al., 2024	Small	Partial $R^2 = .027$ , calculated from $\beta = 2.784, SE = 1.191$ and $df = 195$ .
Wright et al., 2007	Medium	$r = .41$ (from paper)
Yang et al., 2012	Medium	Cohen’s $d_{av} = 0.57$ (average and SDs estimated from figure at 120s, maximal NS–TSD difference; calculated with Lakens’ average standardizer as $\Delta M / (SD_1 + SD_2) / 2$ ; Laken, 2013)

Note: \* Denotes effect size reported by articles. Across studies, multiple effect size metrics were reported or derived. Cohen’s  $d$  was interpreted as small ( $\approx .20$ ), medium ( $\approx .50$ ), and large ( $\approx .80$ ). Cohen’s  $f^2$  was interpreted as small ( $\approx .02$ ), medium ( $\approx .15$ ), and large ( $\approx .35$ ). Pearson’s  $r$  values and  $\beta$  regression values, either reported directly or derived from standardized regression coefficients, were interpreted as small ( $\approx .10$ ), medium ( $\approx .30$ ), and large ( $\approx .50$ ). Eta squared measures such as partial eta squared ( $\eta_p^2$ ), calculated from F tests, and  $\eta_H^2$ , calculated from Kruskal–Wallis H statistic, were interpreted as small ( $\approx .01$ ), medium ( $\approx .06$ ), and large ( $\approx .14$ ). Odds ratios (ORs) were taken directly from papers and interpreted using Chen, Cohen & Chen (2010) guidelines: small ( $\approx 1.5$ ), medium ( $\approx 2.5$ ), and large ( $\approx 4.3$ ). Risk ratios (RRs) were reported directly and interpreted similarly, with values  $> 1$  indicating increased risk and  $< 1$  indicating protective effects. Partial  $R^2$  and Semi-partial  $R^2$  ( $sr^2$ ) values from linear regression models and  $\Delta R^2$  values from hierarchical regressions, were interpreted as small ( $\approx .01$ ), medium ( $\approx .09$ ), and large ( $\approx .25$ ). Incidence rate ratios (IRRs) were presented as relative rate changes (e.g.,  $IRR = 0.73$  indicating a 27% reduction) without universal benchmarks and were interpreted subjectively. SHAP values from machine learning models reflected relative feature importance, not standardized effect sizes, and were interpreted subjectively. For the seven studies with null effects, evidence strength for the null effect was determined as follows: When Confidence intervals (CI) were available or calculable from the data,

evidence strength was determined based equivalence evidence relative to a pre-specified smallest effect size of interest (SESOI; Lakens, Scheel, & Isager, 2018). SESOI was set at  $\delta = 0.20$ , corresponding to Cohen's conventional threshold for a small effect (i.e., effects within  $\pm 0.20$  SD units of zero were considered negligible; Cohen, 1988; Chen, Cohen, & Chen, 2010). Evidence was coded as Strong if the point estimate and entire 95% CI lay within  $\pm\delta$ , Medium if the point estimate was within  $\pm\delta$  but the CI exceeded  $\pm\delta$  on at least one side, and Weak if the point estimate itself lay outside  $\pm\delta$  while the CI also extended beyond  $\pm\delta$ . When CI were unavailable, we determined the strength of evidence for a null effect based on a qualitative evaluation of the relevant  $p$  value and study design.

**Table S2.** Representative Examples of Excluded Studies at Full-Text Review

Exclusion Reason	Representative Study	Study Title	Key Feature Causing Exclusion
No specified stressor	Chen et al., 2017	<i>Relapse insomnia increases greater risk of anxiety in chronic insomnia patients</i>	Did not specify a stressor or experimental manipulation intended to elicit stress.
No full article available (conference abstract/unpublished)	Drummond et al., 2024	<i>The Relationship Between REM Sleep Prior to an Experimental Stressor and Stress Reactivity</i>	Available only as an abstract; full text could not be retrieved for review.
Population is inappropriate	Bai et al., 2020	<i>Longitudinal study of sleep and internalizing symptoms in children with ADHD</i>	Involved populations with existing clinical sleep or stress disorders but without a suitable control group or experimental manipulation of stress. Such studies could not establish temporal or comparative effects of baseline sleep on stress outcomes.
Stress measured before sleep	Akram et al., 2015	<i>Anxiety mediates the relationship between</i>	Examined stress/anxiety outcomes prior to sleep measures, reversing temporal order.

Exclusion Reason	Representative Study	Study Title	Key Feature Causing Exclusion
Stressor existed before sleep measurement	<i>Allen et al., 2021</i>	<i>perfectionism and insomnia symptoms Stress and burnout among graduate students: Moderating role of coping strategies</i>	Stressor was chronic/ongoing before baseline sleep assessment, preventing temporal sequencing.
Stress measurement outcome is inappropriate	<i>Bauducco et al., 2022</i>	<i>Trajectories of insomnia symptoms and insufficient sleep across adolescence</i>	Used indirect measures (insomnia/mood) rather than validated stress outcomes.
No proper analysis of sleep–stress relationships	<i>Akerstedt et al., 2014</i>	<i>Do sleep, stress, and illness explain daily variations in fatigue?</i>	Measured sleep and stress but did not test their statistical association.

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**Table S3.** Risk of Bias Assessment for Randomized Controlled Trials for Human & Animal Studies

	Risk of bias domains						Overall
	D1	DS	D2	D3	D4	D5	
Alkalame et al., 2024	+	○	+	-	+	-	-
Davis et al., 2021*	-	○	-	+	+	+	-
Feng et al., 2018	+	○	+	+	+	-	-
Feng et al., 2023	+	○	+	+	+	-	-
Franzen et al., 2011**	+	+	+	+	-	-	-
Goldstein et al., 2013**	+	+	+	+	-	-	-
Hakimeh & Vahid, 2017*	+	○	-	+	+	+	+
Hicks & Moore, 1979*	+	○	-	+	+	-	-
Jones et al., 2020*	-	○	-	+	+	+	-
Machado et al., 2008*	-	○	-	+	+	+	-
Meerlo et al., 2002*	-	○	-	+	+	-	✗
Minkel et al., 2014	+	○	+	-	+	-	-
O'Leary et al., 2015	+	○	+	+	+	-	-
Oyola et al., 2019*	+	○	+	+	+	-	-
Peters et al., 2014	+	○	+	+	+	-	-
Pinho et al., 2013*	+	○	-	+	+	+	-
Polta et al., 2013*	+	○	+	+	+	-	+
Radwan et al., 2021	-	○	+	+	+	-	-
Ruskin & LaHoste, 2009*	+	○	+	+	+	+	+
Sgoifo et al., 2006*	-	○	✗	+	+	-	✗
Yang et al., 2012	+	○	+	-	+	✗	✗

Domains:  
D1: Bias arising from the randomization process.  
DS: Bias arising from period/carryover effects in crossover designs.  
D2: Bias due to deviations from intended intervention.  
D3: Bias due to missing outcome data.  
D4: Bias in measurement of the outcome.  
D5: Bias in selection of the reported result.

Judgement  
✗ High  
- Some concerns  
+ Low  
○ Not applicable

*Note.* Human studies were assessed using the Cochrane risk-of-bias tool for randomized trials (RoB 2), with Cross-over studies (marked with \*\*) assessed using the RoB 2 extension for crossover trials, which adds additional Domain S to address potential bias arising from carryover effects in crossover designs (Sterne et al., 2019). Animal studies (marked with \*) were assessed using the Systematic Review Centre for Laboratory Animal Experimentation (SYRCLE; Hooijmans et al., 2014) risk-of-bias tool. The ten SYRCLE items were reorganized to parallel the RoB 2's bias assessment domains as follows: Domain 1 included Items 1–3 (sequence generation, baseline characteristics, allocation concealment); Domain 2 included Items 4–5 (random housing, caregiver/investigator blinding); Domain 3 included Items 6 and 8 (random outcome assessment, incomplete outcome data); Domain 4 included Item 7 (blinding of outcome assessor); and Domain 5 included Items 9–10 (selective reporting, other bias). Across all tools, domain ratings were conservative: all items within a domain needed to be low for a Low risk rating; any item with high rating resulted in a High-risk domain; and combinations involving “Unclear” were rated as Some concerns. Overall bias was determined based on standard decision rules over the domain ratings (Sterne et al., 2019).

**Table S4.** Risk of Bias Assessment for Non-Randomized Human Studies

Study	Risk of bias domains							Overall
	D1	D2	D3	D4	D5	D6	D7	
Acheson et al., 2019	⊗	+	-	+	-	+	-	⊗
Bauducco et al., 2024	⊗	+	-	+	-	+	-	⊗
Bottary et al., 2020	+	+	+	+	+	+	-	-
Brand et al., 2018	⊗	+	-	+	-	+	-	⊗
Bryant et al., 2010	-	+	+	+	+	+	-	-
Casement et al., 2019	+	+	+	+	-	+	-	-
Deforges et al., 2021	-	⊗	-	+	⊗	+	-	⊗
Gehrman et al., 2013	-	+	+	+	+	+	-	-
Grove et al., 2023	⊗	+	-	+	-	+	-	⊗
Gruber et al., 2021	⊗	+	-	+	+	+	-	⊗
Hamilton et al., 2021	-	+	+	+	+	-	-	-
Kalmbach et al., 2015	-	+	+	+	+	+	-	-
Kalmbach et al., 2019	-	+	+	+	-	+	-	-
Kiss et al., 2022	⊗	-	+	+	⊗	-	-	⊗
Koeffel et al., 2013	-	+	+	+	+	+	-	-
Larios & Lerner, 2024	-	+	+	+	+	+	-	-
Lerner et al., 2017	-	+	+	+	+	+	-	-
Lucas-Thompson et al., 2009	⊗	-	⊗	+	⊗	+	-	⊗
Marshall et al., 2014	-	+	+	+	+	+	-	-
Massar et al., 2017	⊗	+	-	+	-	+	-	⊗
Neylan et al., 2020	-	+	+	+	-	+	-	-
Nordberg et al., 2022	⊗	+	+	+	-	+	+	⊗
Short & Schmidt, 2018	⊗	-	+	+	-	-	-	⊗
Simon et al., 2024	-	+	+	+	+	+	-	-
Van Liempt et al., 2013	-	⊗	-	+	⊗	+	-	⊗
Wang et al., 2019	-	+	-	+	+	+	-	-
Wang et al., 2022	⊗	-	-	⊗	-	-	-	⊗
Wang et al., 2024	-	-	-	+	⊗	-	⊗	⊗
Wolkow et al., 2024	⊗	+	+	-	⊗	+	-	⊗
Wright et al., 2007	-	+	+	+	+	+	-	-

Domains:  
D1: Bias due to confounding.  
D2: Bias due to selection of participants.  
D3: Bias in classification of interventions.  
D4: Bias due to deviations from intended interventions.  
D5: Bias due to missing data.  
D6: Bias in measurement of outcomes.  
D7: Bias in selection of the reported result.

Judgement  
⊗ Serious  
- Moderate  
+ Low

*Note.* Studies were assessed using the Risk Of Bias In Non-randomized Studies – of Interventions, Version 2 (ROBINS-I V2; Sterne et al., 2016). See Table S3 for decision rules.

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## S2. PRISMA 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
<b>TITLE</b>			
Title	1	Identify the report as a systematic review.	Included
<b>ABSTRACT</b>			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	See below
<b>INTRODUCTION</b>			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Pg. 2-3
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Pg. 3
<b>METHODS</b>			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Pg. 4-5
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Pg. 3-4
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Supp. S1
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 5
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Pg. 5
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Pg. 5-6
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Pg. 5-6
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 5-6
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Table 1; Table S1
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Figure 1
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	n/a
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Table 1, Figure 2
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Table S1; Figure 2-4
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Table 1
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	n/a

## S2. PRISMA 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Pg. 10
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Table S1
<b>RESULTS</b>			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Pg. 7, Figure 1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Figure 1; Table S2
Study characteristics	17	Cite each included study and present its characteristics.	Table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	n/a
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Table 1; Figures 2-4
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Pg. 8-10
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	n/a
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Pg. 10-14
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	n/a
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Pg. 10, Tables S3, S4
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Figure 2
<b>DISCUSSION</b>			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Pg. 15-20
	23b	Discuss any limitations of the evidence included in the review.	Pg. 21-22
	23c	Discuss any limitations of the review processes used.	Pg. 21
	23d	Discuss implications of the results for practice, policy, and future research.	Pg. 21-23
<b>OTHER INFORMATION</b>			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	n/a
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	n/a
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	n/a

## S2. PRISMA 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Included
Competing interests	26	Declare any competing interests of review authors.	Included
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	Pg. 23

*From:* Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ* 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit <https://creativecommons.org/licenses/by/4.0/>

Note: when information was included during submission but was not part of the manuscript file (due to the requirement for deidentification), it was simply noted as “included” in the table.

Accepted Manuscript

## S2. PRISMA 2020 Abstract Checklist

Table 1

Section and Topic	Item #	Checklist item	Reported (Yes/No)
<b>TITLE</b>			
Title	1	Identify the report as a systematic review.	Y
<b>BACKGROUND</b>			
Objectives	2	Provide an explicit statement of the main objective(s) or question(s) the review addresses.	Y
<b>METHODS</b>			
Eligibility criteria	3	Specify the inclusion and exclusion criteria for the review.	Y
Information sources	4	Specify the information sources (e.g. databases, registers) used to identify studies and the date when each was last searched.	Y
Risk of bias	5	Specify the methods used to assess risk of bias in the included studies.	N
Synthesis of results	6	Specify the methods used to present and synthesise results.	N
<b>RESULTS</b>			
Included studies	7	Give the total number of included studies and participants and summarise relevant characteristics of studies.	Y
Synthesis of results	8	Present results for main outcomes, preferably indicating the number of included studies and participants for each. If meta-analysis was done, report the summary estimate and confidence/credible interval. If comparing groups, indicate the direction of the effect (i.e. which group is favoured).	Y
<b>DISCUSSION</b>			
Limitations of evidence	9	Provide a brief summary of the limitations of the evidence included in the review (e.g. study risk of bias, inconsistency and imprecision).	N
Interpretation	10	Provide a general interpretation of the results and important implications.	Y
<b>OTHER</b>			
Funding	11	Specify the primary source of funding for the review.	N
Registration	12	Provide the register name and registration number.	N

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ* 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit <https://creativecommons.org/licenses/by/4.0/>