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A systematic and meta-analytic review of the impact of sleep restriction on memory formation

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ABSTRACT

Modern life causes a quarter of adults and half of teenagers to sleep for less than is recommended (Kocevska et al., 2021). Given well-documented benefits of sleep on memory, we must understand the cognitive costs of short sleep. We analysed 125 sleep restriction effect sizes from 39 reports involving 1234 participants. Restricting sleep (3–6.5 hours) compared to normal sleep (7–11 hours) negatively affects memory formation with a small effect size (Hedges' $g = 0.29$, 95 % CI = [0.13, 0.44]). We detected no evidence for publication bias. When sleep restriction effect sizes were compared with 185 sleep deprivation effect sizes (Newbury et al., 2021) no statistically significant difference was found, suggesting that missing some sleep has similar consequences for memory as not sleeping at all. When the analysis was restricted to post-encoding, rather than pre-encoding, sleep loss, sleep deprivation was associated with larger memory impairment than restriction. Our findings are best accounted for by the sequential hypothesis which emphasises complementary roles of slow-wave sleep and REM sleep for memory.

1. Introduction

Sleep restriction refers to a sleep duration that is less than an individual's typical duration in the absence of sleep debt or below the National Sleep Foundation's recommendation for an individual's age group (e.g., Lowe et al., 2017; Reynolds and Banks, 2010), and it ranges from single episodes of short sleep to chronic reduction over months or years. The National Sleep Foundation recommends that adults sleep for roughly 8 hours per night, adolescents for 9 hours, and school-aged children for 10 hours (Hirshkowitz et al., 2015). Yet, one third to one half of adults, teenagers, and school-aged children across Europe, North America, and Asia sleep for less than their recommended duration (Kocevska et al., 2021; Li et al., 2010; Sheehan et al., 2019). These proportions are stark but unsurprising. Educational and employment schedules are synchronised to 9–5 pm routines which curtail sleep times for many who prefer to wake and sleep later (Goldin et al., 2020; Roenneberg et al., 2007), the 24/7 nature of modern life demands night work despite one in four shift workers experiencing sleep loss (Pallesen et al., 2021), and ubiquitous reliance on digital devices can disrupt sleep-wake cycles (Skeldon et al., 2017; Vetter et al., 2011). The common need for more sleep was evident during COVID-19 lockdowns when lack of sleep timing constraints meant populations globally lengthened their nightly sleep durations (Crowley et al., 2023; Korman et al., 2020).

Sleep restriction is clearly pervasive, and it is therefore critical for public health that the consequences of lack of sleep are properly understood. Physical costs have been well-documented including compromised immune system functioning (Cohen et al., 2009), obesity (Cappuccio et al., 2010), cardiometabolic diseases (Cappuccio and Miller, 2017), neuroendocrine stress (Leproult and Van Cauter, 2010), dysregulated inflammatory responses (Van Leeuwen et al., 2009), and mental health issues (Tang et al., 2017). Beyond physical health effects, the impact of short sleep on waking cognitive performance has received ample scientific attention particularly in cognitive domains most susceptible to tiredness. For example, sleep restriction increases lapses in attention, vigilance, and alertness (Balkin et al., 2004; Belenky et al., 2003; Cunningham et al., 2018; Van Dongen et al., 2003) which are major causes of road traffic and workplace accidents (Chattu et al., 2018). Short sleep diminishes executive functioning and decision-making abilities (Harrison and Horne, 2000; Lau et al., 2019; Stojanoski et al., 2019; Tai et al., 2022) which is a particular concern for professions such as healthcare and aviation where critical decisions are made under high sleep debt conditions (Caldwell, 2012; Smithies et al.,

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2021). Sleep loss also impairs emotional regulation (e.g., Palmer and Alfano, 2017), sensory perception (e.g., Killgore, 2010), working memory (e.g., Frenda and Fenn, 2016), problem solving (e.g., Satterfield and Killgore, 2019), and creativity (e.g., Horne, 1988).

In addition, the impact of sleep restriction on long-term memory formation has been intensely researched. Broadly, the formation of long-term memories can be conceptualised into three distinct phases – encoding, consolidation, and retrieval. Encoding tends to refer to the physical change that occurs in the brain in response to a new input which might be considered to represent a short-term memory trace. Consolidation processes then occur at the synaptic or systems level to strengthen and transform learned information into a stored long-term trace. Following this, memory traces can be spontaneously reactivated to induce conscious recollection or retrieval of the learned information.

The benefit of sleep for encoding and consolidation has been heavily researched and widely accepted since the seminal work of Jenkins and Dallenbach (1924) one century ago. Many theoretical accounts have since been put forward to explain the cognitive and neural mechanisms underlying these effects (for reviews, see Cordi and Rasch, 2021; Diekelmann and Born, 2010; Feld and Born, 2017; Rasch and Born, 2013; Paller et al., 2021; Stickgold and Walker, 2013). Sleep benefits extend beyond strengthening memories as they were learned to transforming them such that qualitative properties emerge including integration into existing networks (e.g., Tamminen et al., 2010), generalisation (e.g., Tamminen et al., 2012), detecting statistical regularities (e.g., Durrant et al., 2011), and uncovering hidden rules (e.g., Lewis et al., 2018). It follows that lack of sleep should impair memory processes, and recent large-scale meta-analyses provide support for this prediction (Newbury et al., 2021). Specifically, Newbury et al. (2021) extracted 31 reports (55 effect sizes, 927 participants) investigating the effect of losing an entire night of sleep (total sleep deprivation) before memory encoding compared to a night of normal sleep, and 45 reports (130 effect sizes, 1616 participants) investigating total sleep deprivation compared to normal sleep after encoding. Results found that missing a night of sleep before encoding impairs memory and is associated with a medium effect size ($g = 0.62$). Missing a night of sleep after encoding also impairs memory and is associated with a small effect size ($g = 0.28$).

Complete lack of sleep, as is the case in total sleep deprivation, is clearly detrimental for preparing the brain for new encoding and consolidation of recently learned information, but whether sleep length is a boundary condition for such effects is however much less clear. Under conditions of sleep restriction, some sleep is present, but the duration is less than a normal night of sleep. It could be that impairments on memory processes emerge only under total sleep deprivation conditions (e.g., Roehrs et al., 2003) or alternatively short sleep over the longer term could have cumulative effects (e.g., Banks and Dinges, 2007; Pilcher and Huffcutt, 1996) that result in similar detriments as total sleep deprivation. Given the prevalence of sleep restriction in modern life, a more thorough understanding of the consequences of short sleep is now warranted. Furthermore, an investigation of sleep restriction may be more informative in adjudicating between competing theories of sleep and memory given that sleep restriction manipulations can target specific stages of sleep. Here we use a systematic and meta-analytic approach to provide the first comprehensive overview of sleep restriction effects on memory, to quantify the magnitude of the associated effect size and its moderating variables, and to compare it to that associated with total sleep deprivation.

1.1. Why might restricting sleep before encoding impair memory?

1.1.1. The synaptic homeostasis hypothesis

The need for sleep before encoding is emphasised in the literature. According to the synaptic homeostasis hypothesis (Cirelli and Tononi, 2021; Tononi and Cirelli, 2003, 2006, 2012), learning is achieved through increased synaptic strength (potentiation). Synaptic potentiation cannot accumulate indefinitely though. As the synaptic strength of a neuron increases so does the amount of energy consumed by it. Additionally, neurons with higher synaptic strengths fire more easily, but this means that a greater range of signals can now cause them to fire making them less selective and less able to distinguish between signal and noise (plasticity-selectivity dilemma). The consequence of this is a continual reduction in the brain's ability to encode new information as it is encountered during the day. To solve this problem, a downscaling process is thought to occur during slow-wave sleep (SWS) – the deepest stage of human sleep, characterised by slow neural oscillations (1–4 *Hz*) - whereby average synaptic strengths are reduced. This causes stronger synapses which activated most consistently during wake to survive whereas weaker synapses that were less consistently activated during wake are weakened such that they do not survive. As a consequence, saturation of learning is prevented and encoding ability is restored after sleep.

In support of the synaptic homeostasis hypothesis, molecular, structural and electrophysiological evidence is consistent with the occurrence of net synaptic potentiation during wake and net synaptic weakening during sleep (for reviews, see Cirelli and Tononi, 2020; Rasch and Born, 2013; Tononi and Cirelli, 2020). On a behavioural level, Mander et al. (2011) demonstrated that memory encoding ability decreases across a 6-hour daytime wake period but is restored by a nap. In fact, encoding ability correlated positively with NREM sleep and fast sleep spindles during the nap (see March et al., 2023, for similar findings). Moreover, neuroimaging evidence has demonstrated reduced encoding-related hippocampal activation following one night of sleep deprivation (Yoo et al., 2007) or one night of suppressed slow wave activity (with preserved total sleep time; Van der Werf et al., 2009). It follows therefore that short sleep before encoding might impair memory given that sufficient downscaling would be prevented and subsequent learning capacity would be impaired.

Crucially, the synaptic homeostasis hypothesis assumes that the mechanism behind synaptic downscaling is the slow firing rates that occur across the entire cortex during up states of slow oscillations in SWS (Tononi and Cirelli, 2006). In support, the amplitude and synchronisation of slow oscillations decrease across a sleep episode, and this is thought to reflect decreasing global synaptic strengths (Esser et al., 2007; Tononi and Cirelli, 2006). It is clear then that this account precludes a role for REM sleep (for a discussion, see Tononi and Cirelli, 2014) despite evidence for REM sleep involvement in downscaling (e.g., Grosmark et al., 2012; Li et al., 2017; Poe, 2017). Similarly, evidence shows that synaptic connections representing recently encoded events are disproportionately strengthened during sleep (e.g., Aton et al., 2014; Chauvette et al., 2012; Ribeiro et al., 1999), yet sleep-related memory benefits, as per the synaptic homeostasis hypothesis, should be non-specific and proportional to the degree of global downscaling. To better incorporate REM sleep and synaptic strengthening, To better incorporate REM sleep and synaptic strengthening, Born and Feld (2012; see also Niethard and Born, 2019) proposed an update of this theory in which slow oscillations during SWS increase certain synaptic strengths as a tagging mechanism, and theta activity during subsequent REM sleep supports downscaling (see also Genzel et al., 2014). Hence, the synaptic homeostasis hypothesis would predict that restricting SWS will impair subsequent encoding ability, whilst more recent work would suggest that restricting REM sleep may in fact impair encoding ability to a similar degree.

1.2. Why might restricting sleep after encoding impair memory?

1.2.1. Active systems consolidation theory

Several theoretical attempts have also been made to delineate how sleep after encoding benefits recently encoded information. The most well-known is the active systems consolidation theory (Klinzing et al., 2019; Kumaran et al., 2016; McClelland et al., 1995) which emphasises the importance of post-encoding SWS for transforming declarative (or hippocampal-dependent) memories. Declarative memory here refers to

conscious and effortful retrieval of facts (semantic memories) and events (episodic memories; Squire and Zola, 1996). According to this theory, newly encoded information is initially stored in distributed neocortical regions with this distributed representation bound together by the hippocampus. At this time, the new memory representation is liable to disruption and is stored separately from existing memories. In subsequent SWS, however, the neuronal groups which represent the memory repeatedly reactivate through hippocampally driven replay causing the information to be gradually reorganised at a systems level such that it becomes independent from the hippocampus and entirely represented by neocortical regions. Neocortical representations are less liable to disruption and are integrated with pre-existing knowledge networks allowing for integration of new memories with existing knowledge and discovery of similarities between new and old memories (Durrant et al., 2011; Lewis et al., 2018; Tamminen et al., 2010, 2012).

Support for this theory comes primarily from experimental designs in which the retention interval between encoding and retrieval is filled with either wakefulness or sleep. In the AM/PM design, for example, some participants undergo the encoding phase in the morning (AM group) and their memory is tested, usually 12 hours later, after a day of normal waking activities. The other half of participants (PM group) undergo the encoding phase in the evening and are tested on their memory in the morning after a night of normal sleep. In a daytime nap study, participants usually sleep for 90 minutes between encoding and retrieval (to allow for one full sleep cycle) or they perform 90 minutes of quiet wake activities. Sleep deprivation studies require participants to remain awake for at least one night between encoding and retrieval, and performance is compared to a rested condition involving a normal night of sleep. Findings across these designs typically show that when the retention interval consists of sleep, memory strength is improved compared to the wake condition (typically measured by higher accuracy; e.g., Van Schalkwijk et al., 2019, reduced response time; e.g., Sánchez-Mora and Tamayo, 2021, or fewer errors; e.g., Petzka et al., 2021) and evidence of integration into semantic memory emerges (Durrant et al., 2011; Lewis et al., 2018; Tamminen et al., 2010, 2012). Additionally, the underlying neural mechanism of hippocampal replay has been evidenced during NREM sleep in humans and animals, and experimentally biasing the content of such replay (using a technique known as targeted memory reactivation; for a review, see Carbone and Diekelmann, 2024) boosts memory performance for the targeted content (Bendor and Wilson, 2012; Crowley et al., 2019; Hu et al., 2020; Rasch et al., 2007). Hence, it follows from the active systems consolidation theory that sleep restriction compared to normal sleep after encoding should impair memory, particularly for sleep restriction that shortens SWS as opposed to REM sleep, for example.

Critically, although recent findings indicate that hippocampal replay might also contribute to the consolidation of memories encoded independently of the hippocampus which typically reflects nonconscious learning known as non-declarative memory (King et al., 2017; Sawangjit et al., 2018; Schapiro et al., 2019), the scope of the active systems consolidation theory is largely limited to post-encoding SWS transforming declarative memories. Hence, sleep restriction after encoding would be expected to impair declarative memory more than non-declarative memory. Moreover, if the active systems consolidation theory is correct in its assertion that hippocampal involvement underpins the benefit of sleep for memory, then sleep restriction may impair performance more on some memory tasks than others. For example, declarative memory is typically assessed using recall or recognition tasks whereby participants retrieve details of a memory or indicate recognition of presented material, respectively. There is ongoing debate concerning the extent to which recognition tasks, compared to recall tasks, require hippocampal involvement given recognition can be supported by a sense of familiarity and that familiarity can be achieved neocortically (Diekelmann et al., 2009; Norman, 2010; Norman and O'Reilly, 2003; Wixted and Squire, 2010). Hence, sleep restriction impairments, as per the active systems consolidation theory, may be less severe in recognition tasks compared to recall tasks (e.g., Berres and Erdfelder, 2021; Drosopoulos et al., 2005; Morgan et al., 2019).

1.2.2. Dual process theories

It is well-accepted that SWS contributes to declarative memory consolidation, but another family of theories, dual process theories, also argue that post-encoding REM sleep is critical for the consolidation of non-declarative information such as procedural, perceptual, and emotional memories (e.g., Plihal and Born, 1997, 1999, Smith, 1995, 2001). REM sleep is characterised by high frequency and low amplitude brain activity, loss of muscle tone and rapid eye movements. REM sleep occurs predominantly during the second half of nocturnal sleep when minimal SWS occurs, while SWS dominates the first half of nocturnal sleep. This defining feature of human sleep allows for clever methodological designs in which memory performance is assessed following undisturbed periods of either early or late nocturnal sleep to determine the relative contributions of SWS and REM sleep on declarative and non-declarative memory consolidation.

Evidence has now accumulated from the early/late paradigm showing improved declarative memory performance following early (SWS-rich) sleep and improved non-declarative memory performance following late (REM sleep-rich) sleep (Ekstrand et al., 1977; Gais et al., 2000; Plihal and Born, 1997, 1999; Wagner et al., 2001; Yordanova et al., 2008; for reviews, see Born et al., 2006; Diekelmann et al., 2009; Marshall and Born, 2007; Rasch and Born, 2013; Rauchs et al., 2005). Further, overnight improvements in procedural memory correlate with REM sleep duration (Fischer et al., 2002), emotional memory consolidation correlates with theta power during REM sleep and REM sleep duration (Nishida et al., 2009), and brain regions associated with procedural learning have been shown to reactivate during REM sleep (Maquet et al., 2000; Laureys et al., 2001; Peigneux et al., 2003). Hence, it follows from dual process theories that there would be an interaction between the sleep stage that sleep restriction after encoding targeted and the type of memory affected, such that declarative memory would be most impaired following SWS-targeted sleep restriction (similarly to active systems consolidation theory) whereas non-declarative memory would be most impaired following sleep restriction during REM sleep.

1.2.3. The sequential hypothesis

The sequential hypothesis postulates that SWS and REM sleep likely have complementary roles in memory consolidation, regardless of memory type, given the cyclic succession of sleep stages in the human sleep cycle (Giuditta, 2014; Giuditta et al., 1995; see also, Diekelmann and Born, 2010; Klinzing et al., 2019; Lewis et al., 2018; Rasch and Born, 2013). More specifically, it assumes that SWS is responsible for tagging to-be-remembered information as such and weakening irrelevant or competing information, whereas REM sleep governs a storage process in which SWS-tagged information is integrated with pre-existing memories. In support, texture discrimination performance improves more following a nap containing SWS and REM sleep compared to solely SWS (Mednick et al., 2003; see also Stickgold et al., 2000), awakenings targeting the natural succession of the sleep cycle impair word retention more than awakenings preserving the sleep cycle (Ficca et al., 2000), and electrophysiological work in rats shows that neural signatures of memory replay during NREM sleep correlate highly with REM theta power (Grosmark et al., 2012; see also Miyawaki & Diba, 2016). Moreover, Diekelmann and Born (2010) built on this complementary SWS and REM sleep hypothesis by explaining how the desynchronised neuronal environment of REM sleep makes it a good candidate for undisturbed synaptic consolidation following systems consolidations in SWS. Hence, sleep restriction after encoding, according to the sequential hypothesis, should impair memory similarly regardless of the memory type (e.g., declarative vs. non-declarative), the task type (e.g., recall vs. recognition), and the sleep stage targeted (e.g., SWS vs. REM sleep).

1.3. Implications for memory encoding and consolidation across theoretical frameworks

We have so far, for the sake of clarity, discussed theories focussing on pre-encoding and post-encoding sleep separately. This however does not mean that the synaptic homeostasis hypothesis has nothing to say about the role of post-encoding sleep in memory consolidation, and that theories of memory consolidation have nothing to say about the role of pre-encoding sleep in restoring subsequent memory encoding capacity. Each of the discussed theories have implications for understanding the roles of both pre-encoding sleep and post-encoding sleep.

The synaptic homeostasis hypothesis argues that as weak synaptic connections are lost through synaptic downscaling, stronger synaptic connections are protected from forgetting (Tononi and Cirelli, 2014). Additionally, simulation evidence shows that low-level details are downscaled more readily than high-level invariants whilst information that does not align with pre-existing knowledge is downscaled more readily than information that does, which support gist extraction and integration respectively (Nere et al., 2013). Hence, synaptic downscaling acts as another memory consolidation mechanism and consequently the synaptic homeostasis hypothesis would also predict that restricted post-encoding SWS should impair memory consolidation.

Active systems consolidation theory, dual process theories and the sequential hypothesis were developed primarily to account for the role of post-encoding sleep in memory consolidation. Yet, they also speak to the effect of short pre-encoding sleep on impairing memory. The active systems consolidation theory argues that the hippocampus is engaged during initial encoding and during hippocampal replay in subsequent SWS but becomes disengaged after the consolidation process has been completed. It follows then that restricting SWS should impair subsequent encoding because hippocampal capacity would not yet have been freed from previously learned information. This suggestion is supported by evidence showing hippocampal encoding deficits under conditions of sleep loss (Yoo et al., 2007) or suppressed slow wave activity (Van der Werf et al., 2009). Dual process theories and the sequential hypothesis are less well-specified so predictions they might make about the neural mechanisms involved in pre-encoding sleep and encoding capacity restoration are harder to make with a great deal of confidence. It is plausible however that they would allocate a role for both REM sleep and SWS in these processes.

1.4. The current sleep restriction meta-analysis

It is clear from the above theories that insufficient sleep before and after encoding has the potential to limit various neural mechanisms occurring during each of the sleep stages and impair performance across multiple memory types and task types. A meta-analytic approach is useful here for synthesising data across studies, calculating typical effect sizes in this literature, and evaluating evidence in support of different sleep theories by determining which moderating variables affect the memory costs of sleep loss.

The present meta-analysis reviews published and unpublished data from the last 50 years of sleep restriction and long-term memory research to quantify the magnitude of the associated effect size, and to provide the first investigation into which factors moderate the sleep restriction effect. Beyond the theoretically-motivated moderating variables discussed above (timing of sleep restriction in relation to encoding, sleep stage targeted by sleep restriction, memory type, and task type), we included the number of sleep restriction nights and sleep duration per night as moderators given that most studies adopt sleep restriction protocols for one week or less, therefore little is known about the cumulative long-term nature of sleep restriction. The presence of recovery sleep and the number of recovery nights were included to determine whether memory deficits remain in the absence of sleepiness due to restricted sleep (Lim and Dinges, 2008) and whether catch-up sleep compensates for lost sleep (Diekelmann et al., 2009; Schönauer et al., 2015). Experimental sleep restriction is typically either achieved by the experimenter repeatedly waking participants throughout the night or by providing participants with in-bed and out-of-bed instructions which shorten their overall sleep time, therefore the method for achieving sleep restriction was included as a moderator. Despite most studies recruiting young-to-middle-aged adults to control for developmental confounds, we included age to assess whether tolerance to sleep restriction varies across the lifespan. For example, most of the theories discussed might predict that effects would be dramatic for children whose sleep architecture constitutes largely SWS, whereas older adults may adapt to their already fragmented sleep (Bliese et al., 2006; Ohayon et al., 2004; Skeldon et al., 2016; Stenuit and Kerkhofs, 2005). Four additional moderators were included to assess the impact of statistical power and methodological quality and are discussed in detail in Section 2.5. Newbury et al. (2021) identified low statistical power as an issue in total sleep deprivation research, therefore we wanted to quantify statistical power in the sleep restriction literature and determine whether there is an association between the statistical power of a study and the effect size observed by it. We also wanted to investigate whether there is an association between the methodological quality of a study and its associated effect size given that Newbury et al. (2021) found evidence suggesting that studies with more stringent exclusion criteria yield smaller total sleep deprivation effects.

1.5. Combining sleep restriction data with total sleep deprivation data

The current meta-analysis also compares the sleep restriction data extracted here with the total sleep deprivation data reported in the Newbury et al. (2021) meta-analyses to determine whether total sleep deprivation or sleep restriction elicits greater impairments to memory. Given that sleep, albeit limited sleep, still occurs during sleep restriction, it could be that effects of sleep restriction on memory are less severe than the effects of total sleep deprivation found in Newbury et al. (2021). Theoretically this might be predicted in light of growing evidence that napping, perhaps for as little as 6–10 minutes (Lahl et al., 2008), produces memory benefits that are comparable to, or even greater than (Berres and Erdfelder, 2021), entire nights of sleep (although perhaps only for declarative memories; Diekelmann et al., 2009; Mander et al., 2011). Similarly, the active systems consolidation theory and the synaptic homeostasis hypothesis prioritise the importance of SWS for sleep-associated memory benefits (for a discussion, see Born, 2010; Walker, 2009), and SWS is most easily preserved in sleep restriction given that it dominates the early hours of sleep (Brunner et al., 1993; Guilleminault et al., 2003). Additionally, the need for memory reconsolidation, whereby recently retrieved information must undergo new consolidation opportunities (Nader and Einarsson, 2010), might suggest that bursts of total sleep loss would be more damaging for long-term memory than short but consistent sleep reductions. Hence, it could be that some sleep is better than none for long-term memory and that the effect size associated with sleep restriction is significantly smaller than the effect associated with total sleep deprivation.

Alternatively, sleep restriction in real life as well as in many laboratory paradigms spans multiple consecutive days whereas total sleep deprivation typically occurs in isolated instances (Alhola and Polo-Kantola, 2007) and rarely extends beyond one night. If effects of sleep restriction are cumulative, long-term memories might be similarly, or perhaps more severely (see Pilcher and Huffcutt, 1996), impaired following chronic sleep restriction compared to a night of total sleep deprivation. In fact, a seminal study found that 14 nights of 6 or 4 hours of sleep caused comparable impairments to working memory as 1 or 2 nights of total sleep deprivation respectively (Van Dongen et al., 2003). Further, whilst some suggest that an adaptive mechanism would compensate for prolonged sleep restriction in the long-term memory domain (Drake et al., 2001), a previous meta-analysis (Lowe et al., 2017) assessed the impact of 2–6 hours of sleep loss and found that the effect size associated with long-term memory was in fact similar to that

recently reported by Newbury et al. (2021). Of note, Lowe et al.'s (2017) meta-analytic estimate was derived from just 12 declarative memory effect sizes, some of which are better described as working memory tasks (e.g., Brown Peterson task; Stenuit and Kerkhofs, 2008). These findings do however fit with sequential sleep theories discussed in Section 1.2.3 which emphasise complementary roles for SWS and REM sleep because sleep restriction often disrupts the natural progression between sleep stages (Banks and Dinges, 2007). Therefore, although some sleep was achieved in Lowe et al.'s (2017) studies, the interactions between sleep stages were likely disrupted which may have caused memory to be impaired similarly to total sleep deprivation investigated by Newbury et al. (2021).

2. Method

2.1. Transparency and openness

We adhered to the MARS guidelines for meta-analytic reporting (Appelbaum et al., 2018). All meta-analytic data, analysis code, and research materials are available at https://osf.io/j8sqx/. This meta-analysis was not preregistered.

2.2. Literature search

In an attempt to include a comprehensive list of sleep restriction studies and to mitigate publication bias, we used four search strategies. First, on 8th March 2023, we used the Boolean search term "Sleep AND (deprivation OR restriction OR loss) AND (learning OR memory OR conditioning)" to search the electronic databases EBSCOhost (included PsycARTICLES, PsycINFO, and PsycTESTS) and PubMED. This search yielded 1817 empirical articles published between 01/01/1970 – 08/03/2023 in peer-reviewed journals in English using human participants.

Second, using the same terms as above, we widened our search criteria in EBSCOhost and PubMED to include unpublished dissertations and theses; we searched the bioRxiv and PsyArXiv repositories for preprints; and we searched the ProQuest and OpenGrey databases for unpublished dissertations and theses, conference materials, and for research grants and fellowship awards. Third, we manually searched the reference lists of six seminal review papers and meta-analyses (Lowe et al., 2017; Maquet, 2001; Pilcher and Huffcutt, 1996; Rasch and Born, 2013; Stickgold et al., 2001; Walker and Stickgold, 2004). Fourth, we contacted all authors with reports that passed our full-text eligibility assessment to ask for any additional published or unpublished data that fit our inclusion criteria (80.6 % response rate). These additional searches yielded 829 reports. In sum, we searched (a) peer-reviewed published articles, (b) in-press articles, (c) preprints, (d) unpublished dissertations and theses, (e) conference materials, and (f) research grants and fellowship awards. Fig. 1 displays a PRISMA flowchart (Moher et al., 2009) showing that after exclusions were removed, 125 effect sizes were included extracted from 32 peer-reviewed published articles, 2 preprints, and 5 unpublished dissertations and theses ($k = 39$).

2.3. Inclusion/exclusion criteria

To identify relevant reports and effect sizes, two authors scanned abstracts and full-texts according to the following criteria:

(a) Participants were from healthy populations.
(b) The primary independent variable was a manipulation of sleep restriction in which the experimental condition slept less than a control condition for a minimum of one night, and the control condition experienced a normal night of uninterrupted sleep.

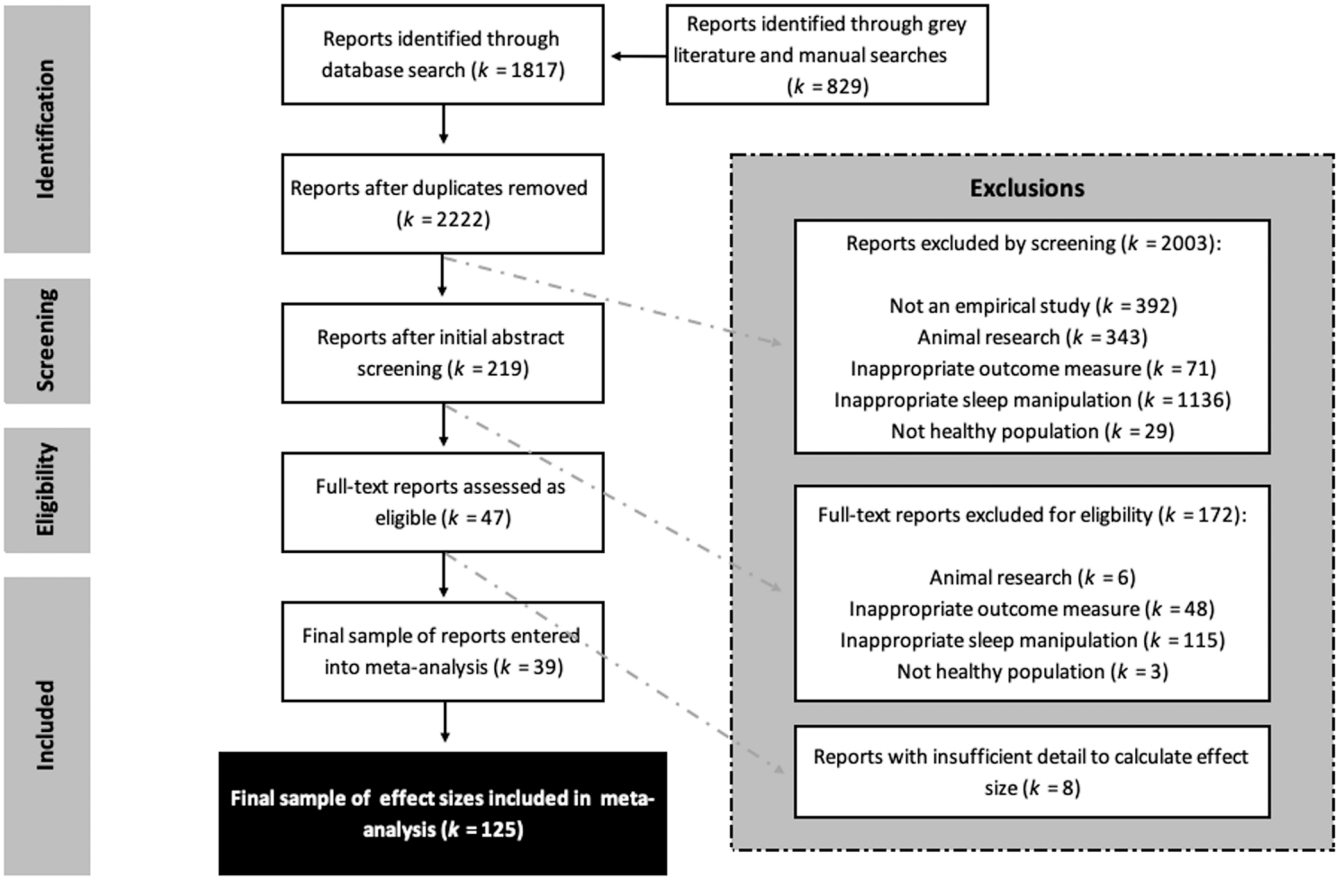

Fig. 1. Flowchart displaying literature search process according to PRISMA guidelines (Moher et al., 2009).

Studies were excluded if there was concern over whether total sleep deprivation may have occurred in some participants. Studies using total sleep deprivation protocols were excluded because the authors have previously conducted a systematic review and meta-analyses on this protocol (Newbury et al., 2021).

(c) The primary dependent variable was at least one measure of long-term memory where the task was described in sufficient detail to ascertain which cognitive function it assessed.

(d) The cognitive task consisted of an encoding phase and retrieval phase(s) that were temporally separate. For studies investigating sleep restriction after encoding, learning and retrieval were separated by either a period of sleep restriction or a period of unrestricted sleep. For studies investigating sleep restriction before encoding, learning and retrieval were separated in time by a retention interval. The reason for requiring the encoding and retrieval phases to be separated in time is that analysis of short-term and working memory tasks has been covered in detail elsewhere (e.g., Lim and Dinges, 2010).

(e) Where studies assessed the effects of other interventions (e.g. motion; Kaplan et al., 2017) in ameliorating or promoting sleep restriction effects, studies were included only if data could be obtained from the control sleep restriction and control sleep groups that were not subject to the intervention.

(f) The report must have included sufficient statistical details to calculate effect sizes (e.g., means and *SD or SEM*). When statistical details were not reported in the text, we contacted corresponding authors to request relevant data. Where this yielded no results and where possible, we extracted relevant data from figures using WebPlotDigitizer (Rohatgi, 2019). The accuracy of figure estimates was confirmed by instances where corresponding authors later provided relevant data.

(g) Where studies used multiple outcome measures to assess performance in a single task (e.g., accuracy and reaction time), we chose only one outcome measure according to the following a priori hierarchy from most to least preferred: signal detection measures (e.g., d-prime), accuracy as measured by retention performance (i.e., performance change from training to test), accuracy at test only, reaction time measured by retention performance, reaction time at test only, error measured by retention performance, error at test only.

(h) Where studies included multiple conditions with varying sleep durations (e.g., Roehrs et al., 2003), we compared the condition with the longest sleep duration to each condition with a sleep duration below 6.5 hours but above 0 hours. We did this to avoid an overlap between the definition of restricted and normal sleep within a study and because sleep durations above 6.5 hours seemed to be the consensus for when sleep was no longer considered to be restricted.

2.4. Coding of study characteristics

Two authors coded the sample and experimental design characteristics of each effect size. Specifically, we coded sample size, gender ratio, mean age, age range, and country of origin. We also coded the experimental design as between-subjects or within-subject, the memory paradigm as declarative or non-declarative, the memory task as free recall, cued recall, recognition memory, motor skill, priming, or texture discrimination, the method of sleep restriction as whether participants were given time-in-bed instructions for a reduced sleep duration or whether they were repeatedly awakened by the experimenter, the sleep stage that sleep restriction targeted as REM sleep, SWS, or indiscriminate, the timing of sleep restriction as before or after encoding, the number of sleep restriction hours, the number of sleep restriction days, whether recovery sleep occurred or not, and the number of recovery nights. Table 1 includes the sample and experimental design properties for each effect size.

2.5. Methodological quality

The reporting and methodological rigour of studies varies considerably across the sleep restriction literature. For example, some studies give a comprehensive overview of participant characteristics (e.g., Genzel et al., 2009) whereas others report no such details (e.g., Cartwright et al., 1975). Some studies ensure strict compliance with the sleep restriction protocol including pre-experimental sleep and human observation of the sleep control and sleep restriction nights (Cousins et al., 2019), whereas others confirm compliance via self-report sleep logs and give no instructions for pre-experimental sleep (e.g., Kim et al., 2015). Some studies use the statistically more powerful within-subject designs and counterbalance conditions (e.g., Kaida et al., 2015) whereas others use between-subjects designs with unequal sample sizes (Voderholzer et al., 2011). Therefore, we assessed reporting and methodological quality for each effect size and included this in our moderator analyses to determine whether the average meta-analytic effect size varies as a function of reporting or methodological quality.

To assess quality, we adapted a 22-item checklist that we developed for a previous sleep deprivation meta-analysis (Newbury et al., 2021; see https://osf.io/j8sqx/) and each item was scored with a zero or one for each effect size according to whether the criterion was satisfied. Given that the items form a multidimensional scale, and each item does not necessarily deserve equal weight (Valentine, 2009), the items were clustered according to the Downs and Black (1998) instrument which assesses five types of bias: reporting, internal validity - bias, internal validity - confounding, power, and external validity. The external validity cluster was dropped from the current meta-analysis because it was designed for clinical intervention studies with nontypical populations. We also dropped the power cluster, which assesses whether a priori power analyses were used, because only one effect size satisfied this criterion in the current meta-analysis. The reporting cluster assesses whether sufficient information regarding participant characteristics was reported (e.g., “Did the study exclude participants taking medication that is known to affect sleep?”). The bias cluster assesses whether the experimental procedure was biased towards a single experimental condition (e.g., “Was the control sleep condition given in-bed and out-of-bed instructions?”). The confounding cluster assesses whether biases were present in the selection and allocation of participants (e.g., “For between-subjects studies, was there random allocation to the sleep restriction and sleep control groups?”).

The methodological quality cluster score for each effect sizes was transformed into a risk of bias rank according to Stone et al. (2020) by dividing each score by the maximum score for that cluster such that lower values represent lower ranked studies and higher values represent higher ranked studies relative to the highest scoring study. In line with Cochran Collaboration recommendations (Higgins et al., 2011), the three clusters were then included in moderator analyses. Table 1 displays the normalised methodological quality cluster scores for each effect size, and the item-level ratings can be found at https://osf.io/j8sqx/.

2.6. Effect size calculation

Effect sizes were coded such that positive effect sizes represent a detrimental effect of sleep restriction on performance compared to a normal sleep control condition. The standardised mean difference (Cohen’s *d*) was calculated for each item included in our meta-analysis but given that effect sizes in Cohen’s *d* can be overestimated for small sample sizes (Lakens, 2013), each effect size was transformed according to the Hedges’ *g* correction. The study means, standard deviations, statistical data where relevant, and effect sizes for each included item can be found at https://osf.io/j8sqx/. The majority of effect sizes ($k = 113$) were calculated directly from means and standard deviations (or standard errors) reported in the texts or provided by authors. A further ten effect sizes were calculated from the means and standard deviations/errors estimated from figures published in the texts. Two effect

Table 1
Key sample and experimental design characteristics for each included effect size. See https://osf.io/j8sqx/ for the categorical recovery sleep moderator, the memory type moderator, location, and % female.

Reference	N	Design	Age	Task	SR days	SR hrs	Recov. nights	SR timing	SR stage	SR meth.	*g*	Power	Reporting	Bias	Confounding
Alberca-Reina et al.; After; Congruence; Test 1	40	B	22	Rec	1	4	0	After	Ind	TIB	−0.15	14.40	0.50	0.86	0.83
Alberca-Reina et al.; After; Congruence; Test 2	40	B	22	Rec	1	4	1	After	Ind	TIB	−0.55	14.40	0.50	0.86	0.83
Alberca-Reina et al.; After; Incongruence; Test 1	40	B	22	Rec	1	4	0	After	Ind	TIB	−0.47	14.40	0.50	0.86	0.83
Alberca-Reina et al.; After; Incongruence; Test 2	40	B	22	Rec	1	4	1	After	Ind	TIB	−0.49	14.40	0.50	0.86	0.83
Alberca-Reina et al.; Associative Memory	40	B	21.8	Rec	1	4	1	Before	Ind	TIB	−0.01	14.40	0.50	0.86	0.83
Alberca-Reina et al.; Before; Congruence; Test 1	40	B	22	Rec	1	4	1	Before	Ind	TIB	0.02	14.40	0.50	0.86	0.83
Alberca-Reina et al.; Before; Congruence; Test 2	40	B	22	Rec	1	4	2	Before	Ind	TIB	−0.45	14.40	0.50	0.86	0.83
Alberca-Reina et al.; Before; Incongruence; Test 1	40	B	22	Rec	1	4	1	Before	Ind	TIB	−0.17	14.40	0.50	0.86	0.83
Alberca-Reina et al.; Before; Incongruence; Test 2	40	B	22	Rec	1	4	2	Before	Ind	TIB	0.17	14.40	0.50	0.86	0.83
Baena et al.	27	B	21.8	Rec	1	4	1	Before	Ind	TIB	0.18	11.11	0.50	0.86	0.83
Biggs et al.; Verbal Free Recall Task	14	W	10.6	FR	1	5	0	After	Ind	TIB	−0.37	16.99	0.83	0.86	1.00
Cartwright et al.; Control v Moderate REM; Free Recall Task	20	B		FR	1		0	After	REM	Wake	−0.62	9.36	0.00	0.86	0.67
Cartwright et al.; Control v REM Absent; Free Recall Task	20	B		FR	1		0	After	REM	Wake	0.04	9.36	0.00	0.86	0.67
Casey et al.; Episodic Task; Factual; REM Restriction	18	W	23.3	FR	1		0	After	REM	Wake	1.41	21.09	0.83	0.86	1.00
Casey et al.; Episodic Task; Factual; SWS Restriction	18	W	23.3	FR	1		0	After	SWS	Wake	2.13	21.09	0.83	0.86	1.00
Casey et al.; Episodic Task; Spatial; REM Restriction	18	W	23.3	FR	1		0	After	REM	Wake	0.88	21.09	0.83	0.86	1.00
Casey et al.; Episodic Task; Spatial; SWS Restriction	18	W	23.3	FR	1		0	After	SWS	Wake	1.78	21.09	0.83	0.86	1.00
Casey et al.; Episodic Task; Temporal; REM Restriction	18	W	23.3	FR	1		0	After	REM	Wake	0.39	21.09	0.83	0.86	1.00
Casey et al.; Episodic Task; Temporal; SWS Restriction	18	W	23.3	FR	1		0	After	SWS	Wake	1.02	21.09	0.83	0.86	1.00
Casey et al.; Spatial Task; Object A; REM Restriction	18	W	23.3	FR	1		0	After	REM	Wake	0.25	21.09	0.83	0.86	1.00
Casey et al.; Spatial Task; Object A; SWS Restriction	18	W	23.3	FR	1		0	After	SWS	Wake	2.33	21.09	0.83	0.86	1.00
Casey et al.; Spatial Task; Object B; REM Restriction	18	W	23.3	FR	1		0	After	REM	Wake	−0.55	21.09	0.83	0.86	1.00
Casey et al.; Spatial Task; Object B; SWS Restriction	18	W	23.3	FR	1		0	After	SWS	Wake	−0.31	21.09	0.83	0.86	1.00
Casey et al.; Visual Priming; REM Restriction	18	W	23.3	Prim	1		0	After	REM	Wake	0.09	21.09	0.83	0.86	1.00
Casey et al.; Visual Priming; SWS Restriction	18	W	23.3	Prim	1		0	After	SWS	Wake	0.06	21.09	0.83	0.86	1.00

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Table 1 (*continued*)

Reference	N	Design	Age	Task	SR days	SR hrs	Recov. nights	SR timing	SR stage	SR meth.	*g*	Power	Reporting	Bias	Confounding
Cedernaes et al.; Finger-Tapping Task	16	W	22.9	FR	3	4.25	0	Both	Ind	TIB	−3.40	19.04	0.67	0.86	1.00
Cedernaes et al.; Spatial Memory Task	16	W	22.9	CR	3	4.25	0	Both	Ind	TIB	−0.07	19.04	0.67	0.86	1.00
Chatburn et al.; Verbal (2017)	22	W	24.9	Rec	4	4	0	Before	Ind	TIB	0.12	25.19	0.83	0.71	1.00
Chatburn et al.; Visual (2017)	22	W	24.9	Rec	4	4	0	Before	Ind	TIB	0.22	25.19	0.83	0.71	1.00
Cousins et al.; 30 Minute Test	59	B	16.1	FR	4	5	0	Before	Ind	TIB	0.52	19.26	0.83	0.86	0.83
Cousins et al.; Continuous; Factual Knowledge Task; Evening	58	B	17	CR	3	6.5	1	Both	Ind	TIB	0.12	19.01	0.83	0.86	0.83
Cousins et al.; Continuous; Factual Knowledge Task; Morning	58	B	17	CR	3	6.5	1	Both	Ind	TIB	0.11	19.01	0.83	0.86	0.83
Cousins et al.; Continuous; Picture Encoding Task	58	B	17	Rec	5	6.5	2	Before	Ind	TIB	0.73	19.01	0.83	0.86	0.83
Cousins et al.; Day 3 Test	59	B	16.1	FR	5	5	2	Both	Ind	TIB	0.40	19.26	0.83	0.86	0.83
Cousins et al.; Day 42 Test	36	B	16.5	FR	5	5	41	Both	Ind	TIB	1.01	12.96	0.83	0.86	0.83
Cousins et al.; Image Recognition Task	59	B	16.1	Rec	5	5	3	Before	Ind	TIB	0.86	19.26	0.83	0.86	0.83
Dodson; Cued Recall Task; Related Words	17	W	28.9	CR	7	6	1	Before	Ind	TIB	−0.18	20.07	0.83	0.57	1.00
Dodson; Cued Recall Task; Unrelated Words	17	W	28.9	CR	7	6	1	Before	Ind	TIB	0.34	20.07	0.83	0.57	1.00
Dodson; Finger Tapping Sequence Task	17	W	28.9	FR	7	6	1	Before	Ind	TIB	−0.14	20.07	0.83	0.57	1.00
Drake et al.; Probed-Recall Memory Task; 8 v 4 hours	12	W	27.5	CR	2	4	0	Before	Ind	TIB	0.33	14.95	0.83	0.86	1.00
Drake et al.; Probed-Recall Memory Task; 8 v 6 hours	12	W	27.5	CR	4	6	0	Before	Ind	TIB	0.06	14.95	0.83	0.86	1.00
Gais et al.; REM Restriction; Visual Discrimination Task	14	B	27	TD	1	3	0	After	REM	TIB	1.63	7.81	0.50	0.71	0.67
Gais et al.; SWS Restriction; Visual Discrimination Task	13	B	27	TD	1	3	0	After	SWS	TIB	2.39	7.61	0.50	0.71	0.67
Genzel et al.; REM Restriction; Finger Tapping Task	12	W	25	FR	1		2	After	REM	Wake	1.83	14.95	1.00	0.86	1.00
Genzel et al.; REM Restriction; Word Pair Recall Task	12	W	25	CR	1		2	After	REM	Wake	1.96	14.95	1.00	0.86	1.00
Genzel et al.; SWS Restriction; Finger Tapping Task	12	W	25	FR	1		2	After	SWS	Wake	0.68	14.95	1.00	0.86	1.00
Genzel et al.; SWS Restriction; Word Pair Recall Task	12	W	25	CR	1		2	After	SWS	Wake	0.86	14.95	1.00	0.86	1.00
Hennecke et al.; Word Pair Task; Day 1	36	B	28.5	CR	1	5	0	Before	Ind	TIB	−0.21	13.15	0.50	0.86	0.67
Hennecke et al.; Word Pair Task; Day 2	36	B	28.5	CR	2	5	0	Both	Ind	TIB	−0.12	13.15	0.50	0.86	0.67
Hennecke et al.; Word Pair Task; Day 3	36	B	28.5	CR	3	5	0	Both	Ind	TIB	0.59	13.15	0.50	0.86	0.67
Hennecke et al.; Word Pair Task; Day 4	36	B	28.5	CR	4	5	0	Both	Ind	TIB	0.75	13.15	0.50	0.86	0.67
Hennecke et al.; Word Pair Task; Day 5	36	B	28.5	CR	5	5	0	Both	Ind	TIB	0.36	13.15	0.50	0.86	0.67
Hennecke et al.; Word Pair Task; Recovery Day	36	B	28.5	CR	5	5	1	Before	Ind	TIB	−0.32	13.15	0.50	0.86	0.67
Huang et al.; Massed Learning; Test 1	56	B	17	CR	4	5	0	After	Ind	TIB	0.53	18.42	0.83	0.86	0.83
Huang et al.; Massed Learning; Test 2	56	B	17	CR	7	5	1	After	Ind	TIB	0.57	18.42	0.83	0.86	0.83

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Table 1 (*continued*)

Reference	N	Design	Age	Task	SR days	SR hrs	Recov. nights	SR timing	SR stage	SR meth.	*g*	Power	Reporting	Bias	Confounding
Huang et al.; Spaced Learning; Test 1	56	B	17	CR	4	5	0	After	Ind	TIB	0.30	18.42	0.83	0.86	0.83
Huang et al.; Spaced Learning; Test 2	56	B	17	CR	7	5	1	After	Ind	TIB	0.14	18.42	0.83	0.86	0.83
Kaida et al.; Recognition Task; Negative; Test 1	18	W	22	Rec	1		0	Before	REM	Wake	−0.17	21.09	1.00	0.86	1.00
Kaida et al.; Recognition Task; Negative; Test 2	18	W	22	Rec	1		0	Before	REM	Wake	0.15	21.09	1.00	0.86	1.00
Kaida et al.; Recognition Task; Neutral; Test 1	18	W	22	Rec	1		0	Before	REM	Wake	−0.10	21.09	1.00	0.86	1.00
Kaida et al.; Recognition Task; Neutral; Test 2	18	W	22	Rec	1		0	Before	REM	Wake	0.10	21.09	1.00	0.86	1.00
Kaida et al.; Recognition Task; Positive; Test 1	18	W	22	Rec	1		0	Before	REM	Wake	0.04	21.09	1.00	0.86	1.00
Kaida et al.; Recognition Task; Positive; Test 2	18	W	22	Rec	1		0	Before	REM	Wake	0.03	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Negative; Test 1	18	W	22	CR	1		0	Before	REM	Wake	0.05	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Negative; Test 2	18	W	22	CR	1		0	Before	REM	Wake	0.11	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Neutral; Test 1	18	W	22	CR	1		0	Before	REM	Wake	0.00	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Neutral; Test 2	18	W	22	CR	1		0	Before	REM	Wake	0.00	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Positive; Test 1	18	W	22	CR	1		0	Before	REM	Wake	0.07	21.09	1.00	0.86	1.00
Kaida et al.; Source Memory Task; Positive; Test 2	18	W	22	CR	1		0	Before	REM	Wake	0.08	21.09	1.00	0.86	1.00
Kaplan et al.; Stationary; Procedural Memory Task	30	B	20.6	MS	2	4	0	After	Ind	TIB	0.15	11.84	0.83	0.86	0.83
Kaplan et al.; Stationary; Visual Discrimination Task	27	B	20.5	TD	2	4	0	After	Ind	TIB	0.19	11.04	0.83	0.86	0.83
Karni et al.; REM Restriction;	6	W	19.5	TD	1.5		0	After	REM	Wake	8.66	8.94	0.50	0.71	0.50
Karni et al.; SWS Restriction	6	W	19.5	TD	1.5		0	After	SWS	Wake	1.68	8.94	0.50	0.71	0.50
Kim et al.; Moderate SD; California Verbal Learning Test; Delayed Recall	28	B	27.2	FR	14		0	Before	Ind	TIB	0.35	11.37	0.67	0.29	0.50
Kim; Free Recall Task	12	W	18.9	FR	4	6	0	Before	Ind	TIB	3.13	14.95	0.50	0.57	0.67
Kim; Recognition Task	12	W	18.9	Rec	4	6	0	Before	Ind	TIB	0.83	14.95	0.50	0.57	0.67
Kopasz et al.; Auditory Verbal Learning Task	22	W	15.5	CR	1	4	1	After	Ind	TIB	0.10	25.19	0.83	0.86	1.00
Kopasz et al.; Paired-Associate Word List Task	22	W	15.5	CR	1	4	1	After	Ind	TIB	1.25	25.19	0.83	0.86	1.00
Kopasz et al.; Verbal Memory Task	22	W	15.5	CR	1	4	1	After	Ind	TIB	−0.25	25.19	0.83	0.86	1.00
Kopasz et al.; Visual Memory Task	22	W	15.5	CR	1	4	1	After	Ind	TIB	−0.22	25.19	0.83	0.86	1.00
Leong et al.; Target Word Recognition	59	B	16.1	Rec	5	5	0	After	Ind	TIB	0.20	19.26	0.83	0.86	0.83
Lo et al.; Experiment 1	40	B	22.1	CR	7	5	0	Before	Ind	TIB	−0.39	14.40	0.67	0.57	0.83
Lo et al.; Experiment 2	54	B	16.7	CR	7	5	0	Before	Ind	TIB	−0.17	17.91	0.83	0.86	0.83
Lo et al.; Highlighted; Test 1	45	B	16.6	FR	7	5	0	After	Ind	TIB	0.41	15.54	0.67	0.86	0.67
Lo et al.; Highlighted; Test 2	45	B	16.6	FR	7	5	35	After	Ind	TIB	0.20	15.54	0.67	0.86	0.67
Lo et al.; Non-highlighted; Test 1	45	B	16.6	FR	7	5	0	After	Ind	TIB	0.09	15.54	0.67	0.86	0.67

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Table 1 (*continued*)

Reference	N	Design	Age	Task	SR days	SR hrs	Recov. nights	SR timing	SR stage	SR meth.	*g*	Power	Reporting	Bias	Confounding
Lo et al.; Non-highlighted; Test 2	45	B	16.6	FR	7	5	35	After	Ind	TIB	0.23	15.54	0.67	0.86	0.67
Lorenzetti; Visual Object Learning Test	9	W	20	Rec	7		0	Before	Ind	TIB	−6.06	9.92	0.83	0.71	0.67
Morgenthaler et al.; Negative	29	B	23	Rec	1		0	After	REM	Wake	0.19	11.61	0.67	0.86	0.83
Morgenthaler et al.; Neutral	29	B	23	Rec	1		0	After	REM	Wake	0.39	11.61	0.67	0.86	0.83
Randazzo et al.; California Verbal Learning Task	16	B	11.9	FR	1	5	0	Before	Ind	TIB	0.41	8.37	0.67	0.86	0.83
Reid et al.; Word Pair Task	51	B	24	CR	1		0	After	Ind	Wake	1.07	17.17	1.00	1.00	0.83
Roehrs et al.; Word Pair Recall Task; 8 v 4 hours	12	W	27.5	CR	1	4	0	Before	Ind	TIB	0.31	14.95	0.83	0.86	1.00
Roehrs et al.; Word Pair Recall Task; 8 v 6 hours	12	W	27.5	CR	1	6	0	Before	Ind	TIB	0.00	14.95	0.83	0.86	1.00
Saxvig et al.; California Verbal Learning Test	24	B	23.3	FR	1		0	After	REM	Wake	0.42	10.36	0.67	0.86	0.67
Saxvig et al.; Complex Figure Test	24	B	23.3	FR	1		0	Before	REM	Wake	−0.14	10.36	0.67	0.86	0.67
Saxvig et al.; Word Stem Test	19	B	23.3	Prim	1		0	Before	REM	Wake	0.10	8.82	0.67	0.86	0.67
Smith et al.; Experiment 1; N1–3 v Control	20	B	21.5	MS	1		6	After	Ind	TIB	0.49	9.36	0.33	0.86	0.83
Smith et al.; Experiment 1; N2–3 v Control	20	B	21.5	MS	1		6	After	Ind	TIB	0.30	9.36	0.33	0.86	0.83
Smith et al.; Experiment 1; N3–3 v Control	20	B	21.5	MS	1		6	After	Ind	TIB	0.09	9.36	0.33	0.86	0.83
Smith et al.; Experiment 1; N4–3 v Control	20	B	21.5	MS	1		6	After	Ind	TIB	−0.57	9.36	0.33	0.86	0.83
Smith et al.; Experiment 2; LH-TSD v Control	17	B	21.5	MS	1	3.5	6	After	SWS	TIB	0.21	8.61	0.33	0.86	0.83
Smith et al.; Experiment 2; NREMA v Control	17	B	21.5	MS	1		6	After	SWS	Wake	0.23	8.61	0.33	0.86	0.83
Smith et al.; Experiment 2; REMD v Control	16	B	21.5	MS	1		6	After	REM	Wake	0.52	8.37	0.33	0.86	0.83
Sopp et al.; Explicit Memory	40	B	24	Rec	1	3.5	0	After	REM	TIB	0.78	14.38	0.83	0.86	0.83
Sopp et al.; Implicit Memory	41	B	24	Prim	1	3.5	0	After	REM	TIB	0.06	14.65	0.83	0.86	0.83
Stenstrom; Experiment 1; Incidental Factual	16	B	25.1	CR	1		1	After	REM	Wake	0.25	8.37	0.50	0.57	0.83
Stenstrom; Experiment 1; Incidental Spatial	16	B	25.1	CR	1		1	After	REM	Wake	1.11	8.37	0.50	0.57	0.83
Stenstrom; Experiment 1; Target Factual	16	B	25.1	CR	1		1	After	REM	Wake	−0.88	8.37	0.50	0.57	0.83
Stenstrom; Experiment 1; Target Spatial	16	B	25.1	CR	1		1	After	REM	Wake	−0.50	8.37	0.50	0.57	0.83
Stenstrom; Experiment 1; Target Temporal	16	B	25.1	CR	1		1	After	REM	Wake	0.43	8.37	0.50	0.57	0.83
Stenuit et al.; Old; Buschke Test; Total Recall; 20-Minute Test	10	W	60	FR	3	4	0	Before	Ind	TIB	0.47	12.92	0.83	0.86	0.67
Stenuit et al.; Young; Buschke Test; Total Recall; 20-Minute Test	10	W	23.2	FR	3	4	0	Before	Ind	TIB	0.41	12.92	0.83	0.86	0.67
Tantawy et al.; Experiment 1; REM Restriction	20	B	18	CR	1	4	0	After	REM	TIB	1.20	9.36	0.33	0.71	0.83
Tantawy et al.; Experiment 1; SWS Restriction	20	B	18	CR	1	4	0	After	SWS	TIB	1.50	9.36	0.33	0.71	0.83
Tucker; Number Sequence Task	24	B	20.9	FR	1	3.5	0	After	Ind	TIB	0.07	10.32	0.50	0.86	0.83

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Table 1 (*continued*)

Reference	N	Design	Age	Task	SR days	SR hrs	Recov. nights	SR timing	SR stage	SR meth.	*g*	Power	Reporting	Bias	Confounding
Tucker; Paired Associates	24	B	20.9	CR	1	3.5	0	After	Ind	TIB	0.21	10.32	0.50	0.86	0.83
Voderholzer et al.; Mirror Tracing Task; Test 1; 9 v 5 hours	29	B	15	MS	4	5	2	After	Ind	TIB	0.14	11.42	0.67	0.71	0.67
Voderholzer et al.; Mirror Tracing Task; Test 1; 9 v 6 hours	33	B	15	MS	4	6	2	After	Ind	TIB	0.15	12.62	0.67	0.71	0.67
Voderholzer et al.; Mirror Tracing Task; Test 2; 9 v 5 hours	29	B	15	MS	4	5	30	After	Ind	TIB	0.55	11.42	0.67	0.71	0.67
Voderholzer et al.; Mirror Tracing Task; Test 2; 9 v 6 hours	33	B	15	MS	4	6	30	After	Ind	TIB	0.35	12.62	0.67	0.71	0.67
Voderholzer et al.; Word Pair Task; Test 1; 9 v 5 hours	29	B	15	CR	4	5	2	After	Ind	TIB	−0.38	11.42	0.67	0.71	0.67
Voderholzer et al.; Word Pair Task; Test 1; 9 v 6 hours	33	B	15	CR	4	6	2	After	Ind	TIB	−0.30	12.62	0.67	0.71	0.67
Voderholzer et al.; Word Pair Task; Test 2; 9 v 5 hours	29	B	15	CR	4	5	30	After	Ind	TIB	−0.52	11.42	0.67	0.71	0.67
Voderholzer et al.; Word Pair Task; Test 2; 9 v 6 hours	33	B	15	CR	4	6	30	After	Ind	TIB	−0.28	12.62	0.67	0.71	0.67

Note. SR = sleep restriction; recov. = recovery; W = within-subject; B = between-subjects; FR = free recall; CR = cued recall; Rec = recognition; MS = motor skill; TD = texture discrimination; Prim = priming; Ind = indiscriminate; TIB = time-in-bed instructions; Wake = awakenings; Power refers to statistical power; Reporting, bias, and confounding refer to the three clusters of the methodological quality checklist.

sizes were calculated from the Mann Whitney *U* statistic. Effect sizes were calculated according to the formulae reported in the supplementary materials.

2.7. Data analysis

2.7.1. Meta-analytic procedure

A three-level random effects model was fitted to our data, using the *metafor* package in R (Viechtbauer, 2010), to determine whether there was a meta-analytic effect of sleep restriction compared to normal sleep on long-term memory. There were two key reasons for choosing a multilevel random effects model over a fixed effects model. First, given that sleep restriction studies employ different lengths of sleep restriction, different methods for achieving sleep restriction, different timings of sleep restriction and a variety of memory paradigms, we expected variability in the true effect size underlying each study. Random-effects models account for this by assuming systematic variability between effect sizes over and above random sampling error. A fixed effects model would assume that all studies share the same underlying true effect size. Consequently, random effects models produce more conservative meta-analytic estimates owing to larger standard errors (Borenstein et al., 2009).

Second, each report contributed an average of 3.21 effect sizes to the meta-analysis due to data from multiple test sessions, within-group experimental conditions (e.g., massed vs. spaced learning; Huang et al., 2016), and multiple tasks (e.g., spatial memory task, episodic memory task, and priming; Casey et al., 2016). Including dependent effect sizes from the same study would violate the data independence assumption of typical random effects models (Borenstein et al., 2009). Multilevel random effects models, however, account for such dependencies by modelling within-study and between-study variances (Van den Noortgate et al., 2014). Hence, effect sizes in a three-level random effects model can vary between participants (sampling error), outcomes (multiple effect sizes from the same study), and studies (effect sizes from different studies). To further account for this hierarchical structure, we used robust variance estimation to correct for dependencies amongst effect sizes (Hedges et al., 2010; Pustejovsky and Tipton, 2022) using the *clubSandwich* package in R (Pustejovsky, 2020). All analysis code can be found at https://osf.io/j8sqx/.

2.7.2. Heterogeneity

The *Q* test (Cochran, 1954) is a method for determining whether there is significant variability between all effect sizes beyond that which can be explained by random sampling error. However, we are more interested in whether there is significant variability in effect sizes within and between studies than would be expected based on sampling error alone. To determine whether there is significant within-study heterogeneity and between-study heterogeneity, we ran two one-sided log-likelihood-ratio tests. Here, the fit of the overall multilevel model where within- and between-study variances are freely estimated is compared to the fit of a model where only within-study variance is freely estimated and to the fit of a model where only between-study variance is freely estimated.

We then used the I^2 statistic, as calculated by Cheung (2014), to quantify how the total variance is distributed across the three levels of the meta-analytic model (Higgins and Thompson, 2002). If less than 75 % of overall variance is attributed to sampling error, it is necessary to examine whether moderating variables may explain some of the within- and between-study variance in effect sizes.

Finally, we report τ^2, as recommended by Borenstein et al. (2009), which provides absolute values that quantify the dispersion of true effect sizes as opposed to observed effect sizes. We also report prediction intervals which indicate the range in which 95 % of future effect sizes will fall.

2.7.3. Publication bias

We used four methods for investigating whether our meta-analytic effect size estimate is confounded by publication bias. We first examined a contour-enhanced funnel plot displaying effect sizes according to their standard error with contour lines representing boundaries of statistical significance (e.g., $p < .01$, $p < .05$, $p < .10$; Peters et al., 2008). Visual inspection of contour-enhanced funnel plots aids interpretation of

funnel plot asymmetry by revealing where "missing" effect sizes lie. If effect sizes are missing from regions of statistical non-significance, this indicates that biases exist in favour of reporting statistically significant results in the literature. If effect sizes are missing from regions of statistical significance, asymmetry is likely driven by other biases, such as the size of true effects varying as a result of qualitative differences between differently sized studies and poor methodological quality inflating effect sizes in smaller studies (see Sterne et al., 2011).

We next conducted a multi-level Egger's regression test (Egger et al., 1997) to determine whether the funnel plot was significantly asymmetrical according to a linear regression function. Third, we conducted the trim-and-fill method (Duval and Tweedie, 2000) which suppresses (trims) extreme effect sizes to calculate a bias-corrected overall meta-analytic estimate and imputes missing effect sizes (fill) to correct for subsequently reduced variance.

However, there are limitations to the Egger's test and the trim-and-fill method: a) they are means for assessing funnel plot asymmetry rather than publication bias per se, b) Egger's test can produce false positives when used with standardised mean differences (Hedges' *g*) given that these effect sizes are intrinsically related to their standard errors, and this is particularly true in the presence of small within-subject sample sizes or large between-study heterogeneity (Pustejovsky and Rodgers, 2019), and c) the performance of the trim-and-fill method decreases with increasing between-study heterogeneity (Peters et al., 2007; Terrin et al., 2003). Therefore, we also conducted the three-parameter likelihood selection model (3-PSM; Iyengar and Greenhouse, 1988) which assesses publication bias without relying on funnel plot asymmetry, and the statistical power of which does not decrease with increasing between-study heterogeneity. Note however that the 3-PSM does not account for dependence between effect sizes which can inflate the Type I error rate (Rodgers and Pustejovsky, 2020). The 3-PSM corrects for the likelihood of publication according to specified p value boundaries and then produces an adjusted average effect size, a heterogeneity parameter, and a weight parameter which gives the likelihood of a non-significant effect being reported relative to a significant effect. Here we used a one-tailed model with a step function cut-off of $p = .025$ in line with a selection process where predicted and statistically significant studies are more likely to be reported.

2.7.4. Outlier and influential case analysis

We explored whether outliers and influential cases impacted the size of the meta-analytic estimate. First, effect sizes with studentized residuals $> \pm 3$ were classified as statistical outliers and excluded. Next, we used case deletion diagnostics to determine the influence of deleting each effect size on all parameter estimates (Cook's distance) and on single parameter estimates (DFBETAs; Viechtbauer and Cheung, 2010). Effect sizes with a problematic Cook's distance or DFBETAs were excluded. A problematic Cook's distance was defined as 4/no. of observations (Van der Meer et al., 2010), and problematic DFBETAs were defined as 2/no. of observations (Belsley et al., 1980).

2.7.5. Moderator analyses

Moderator analyses were conducted using the Q statistic because Monte Carlo simulations indicate that Type 1 error rates and statistical power are acceptable and stable with this method (Aguinis & Pierce, 1998). We separately analysed the effect of ten theoretically-motivated moderator variables on the size of the sleep restriction effect: 1) sleep duration in hours for the sleep restriction condition, 2) number of sleep restriction nights, 3) method of sleep restriction (timing vs. awakenings), 4) sleep stage that restriction targeted (SWS, REM sleep, or indiscriminate), 5) timing of sleep restriction (before encoding vs. after encoding), 6) recovery sleep (yes vs. no), 7) number of recovery sleep nights, 8) memory type (declarative vs. non-declarative), 9) task type (cued recall, free recall, recognition, or motor skill), and 10) age (continuous moderator using the mean age for each effect size but where mean age was not reported, we used the midpoint of the age range, $k = 34$). We also performed four methodologically-motivated moderator analyses: 1) normalised score for the reporting cluster of the methodological quality checklist, 2) normalised score for the bias cluster, 3) normalised score for the confounding cluster, and 4) statistical power to detect the average meta-analytic effect size.

Note that 50 effect sizes were not included in the sleep restriction duration moderator analysis because sleep duration in the sleep restriction condition was not reported, either due to sleep duration being tailored per participant (e.g., sleep for 90 minutes less than the pre-experimental week average; Lorenzetti, 2020) or because sleep restriction was achieved via repeated awakenings (e.g., Morgenthaler et al., 2014). Ten effect sizes were removed from the timing of sleep restriction moderator analysis due to sleep restriction occurring both before and after encoding. Note that we did not perform two separate meta-analyses on sleep restriction before encoding versus after encoding as in Newbury et al. (2021) because the number of available effect sizes in the sleep restriction literature is smaller and we did not want to limit statistical power for remaining moderator analyses. Nine effect sizes were removed from the task type moderator analysis (texture discrimination, $k = 5$; priming, $k = 4$) because their inclusion would severely impact the proportion of effect sizes in each subcategory of the moderator variable which reduces statistical power (Hempel et al., 2013), and we did not feel the task types were homogenous enough to be combined into a single category.

3. Results

This meta-analysis provides a comprehensive summary of the sleep restriction and long-term memory literature across the last 50 years (1970– 2023). We collected 39 reports which contributed 125 effect sizes to the meta-analysis. All reports used healthy populations and restricted participants' sleep to 3–6.5 hours over 1–14 nights either prior to (43 effect sizes) or after encoding (72 effect sizes). Four reports (10 effect sizes) restricted participants sleep both before and after encoding. Sleep restriction for 45 effect sizes was achieved by repeatedly waking participants in order to disrupt specific sleep stages (SWS: $k = 10$; REM sleep: $k = 34$; Indiscriminate: $k = 1$) whilst restriction for 80 effect sizes was achieved via time-in-bed instructions. For 50 effect sizes, participants were allowed recovery sleep before retrieval. The duration of sleep experienced in control groups ranged from 7 – 11 hours (median = 7 hours, mean = 8 hours, 17 minutes) which aligns with the minimum and maximum recommendations given by the National Sleep Foundation (Hirshkowitz et al., 2015) for the age groups represented. 28 effect sizes did not report sleep duration for the control group. This dataset represents 1234 participants (% female mean [*SD*] = 49 % [23 %], range: 0–100 %) and spans childhood to older age (mean [*SD*] = 21.79 [5.38], range: 10–65 years). Although note that one effect size represents adults over 55 and the remaining effect sizes represent 10–40-year-olds. Of the 116 effect sizes reporting location, 59 were conducted in Europe, 24 in North America, 30 in Asia, 2 in Africa, and 1 in Australasia. Studies employed within-subject (sample size mean [*SD*] = 16.12 [4.00], range: 6–22) and between-subjects designs (sample size mean [*SD*] = 34.03 [13.85], range: 14–59) similarly often, however there was considerably low statistical power across both experimental designs (mean [*SD*] = 15.41 % [4.88 %]; range = 7.61 % - 25.19 %). We return to the implications of this problem in detail in Section 4.3. Cued recall, free recall, and recognition paradigms were similarly represented across declarative memory studies, however there is a clear gap in the literature for non-declarative memory studies given that only 20 % of the current effect sizes investigated this memory type (motor skill, discrimination, and priming). See Table 1 for study and experimental design properties for each effect size.

3.1. Overall meta-analytic effect size

The overall meta-analytic estimate for the mean difference in

memory performance between sleep restriction and control conditions ($g = 0.29$, $SE = 0.08$) was significantly different from 0 (95 % CI = [0.13, 0.44], $p < .001$). This indicates a detrimental effect of sleep restriction on memory with a small effect size according to Cohen's guidelines for categorising effect sizes as small (0.20), medium (0.50), and large (0.8). Note that the overall meta-analytic estimate did not differ after robust variance estimation had been applied ($g = 0.29$, $SE = 0.08$; 95 % CI = [0.13, 0.45], $t(33.93) = 3.64$, $p < .001$). See Fig. 2 for a forest plot displaying Hedges' g and 95 % confidence intervals for all included effects sizes. Heterogeneity analysis revealed significant variability across all effect sizes beyond that which is explained by random error ($Q(124) = 398.52$, $p < .001$). There was significant heterogeneity at an outcome level (within-study, $p < .001$) and at a study level (between-study, $p < .001$). I^2 revealed that 25.95 % of the variance in observed effect sizes could be attributed to random sampling error, 38.76 % to within-study variance, and 35.28 % to between-study variance. We next quantified the dispersion of true effect sizes as $\tau^2 = 0.14$ for within-study variance and $\tau^2 = 0.12$ for between-study variance. Further, prediction intervals show that 95 % of future effect sizes should fall between −0.73 and 1.30. Given the considerable heterogeneity, the large prediction intervals which cross 0, the variance of true effect sizes, and that only 25.95 % of variance could be attributed to sampling error (Hunter and Schmidt, 2004), it is necessary to compare effects of sleep restriction across potential moderating variables.

3.2. Publication bias

The distribution of effect sizes according to standard error is displayed in the funnel plot in Fig. 3. Two large effect sizes with large standard errors are excluded from the funnel plot ($g = -6.06$, $SE = 1.65$ and $g = 8.66$, $SE = 2.52$) because they made visual inspection difficult but see Figure S1 for the funnel plot with outliers included. Given that many studies are plotted in areas of non-significance, it seems as though the sleep restriction literature does not bias against publishing statistically non-significant data. However, visual inspection also shows that when standard errors are highest, and therefore the precision of effect sizes is lowest, large detrimental (positive Hedges' g) effects of sleep restriction are reported most often. This could be an indication of publication bias or it could be driven by other biases including chance, heterogeneity and methodological quality (Sterne et al., 2011). For example, statistical power is often low in sleep restriction research which renders these studies prone to false positives occurring by chance. Poor methodological quality (e.g., not excluding participants with sleep disorders) may lead to inflated effect sizes in smaller studies. Additionally, there may be methodological differences between studies of different sizes which lead to heterogeneity in the size of the underlying true effect. As an example, sleep restriction effects may be larger in participants that are monitored continuously throughout the night rather than being given time-in-bed instructions at home, and these studies likely have smaller sample sizes because they are more resource-intensive.

We next examined Egger's regression test which further revealed that the funnel plot is significantly asymmetric ($z = 3.90$, $p < .001$). Given the distribution of effect sizes in Fig. 3 and that Egger's test was significant, we sought to determine whether trim-and-fill analysis would impute missing effect sizes and how this might influence the meta-analytic estimate. This method yielded a smaller, but still statistically significant, adjusted overall meta-analytic effect size of $g = 0.26$ ($SE = 0.05$, 95 % CI = [0.15, 0.36], $Z = 4.92$, $p < .001$). However, we caution against interpreting this estimate because 0 missing effect sizes were imputed and therefore the variance of the effect size is left uncorrected.

Given the limitations of Egger's regression test and the trim-and-fill method in terms of being measures of funnel plot asymmetry rather than publication bias as well as their performance against false positives and between-study heterogeneity, we next performed a 3-PSM. This model found no evidence of publication bias in the sleep restriction literature because there was no significant improvement in model fit when publication bias was modelled versus when it was not ($\chi^2(1) = 1.46$, $p = .228$).

Overall, it seems that reliable evidence for publication bias is not detectable in the sleep restriction literature. However, there is statistical evidence for funnel plot asymmetry indicating that other biases may exist.

3.3. Outlier and influential case analysis

Six effect sizes were identified as statistical outliers and were excluded. Five effect sizes were identified as having a problematic Cook's distance, but two matched those previously identified as statistical outliers. Five effect sizes were identified as having problematic DFBETAs, but two had previously been identified as statistical outliers and three had previously been identified as having problematic Cook's distances. In sum, nine effect sizes were identified as outliers or influential cases and were excluded: (1) Casey et al.; Spatial Task; Object A; SWS Restriction; 2016, (2) Casey et al.; Episodic Task; Factual; SWS Restriction; 2016, (3) Cedernaes et al.; Finger-Tapping Task; 2016, (4) Karni et al.; REM Restriction; 1994, (5) Karni et al.; SWS Restriction; 1994, (6) Kim; Free Recall Task; 2015, (7) Lorenzetti; Visual Object Learning Test; 2020; (8) Gais et al.; SWS Restriction; Visual Discrimination Task; 2000, (9) Reid et al.; Word Pair Task; 2023. Removal of outlier and influential cases caused the original meta-analytic effect size estimate of $g = 0.29$ to decrease to $g = 0.23$ ($SE = 0.06$) but this is still significantly above 0 (95 % CI = [0.12, 0.35], $p < .001$) and within Cohen's guidelines for a small effect size.

3.4. Moderator analyses

Although outliers and influential cases have larger influences on smaller subsets of data as is the case in moderator analyses, some argue against outlier and influential case removal given that such cases may simply arise by chance rather than incorrect data (Hunter and Schmidt, 2004). Viechtbauer and Cheung (2010) suggested that inspection of outliers and influential cases should form a sensitivity analysis whereby it is possible to determine which conclusions hinge on the inclusion of a small number of rare studies. For this reason, we chose to report moderator analyses both with and without the nine outliers and influential cases identified above.

In light of the average meta-analytic effect size estimate being $g = 0.29$, a post hoc power analysis was conducted in G*Power (where $\alpha = 0.05$; two-tailed; Faul et al., 2009) to determine achieved statistical power of each included effect size to detect this meta-analytic estimate (mean [SD] = 15.41 % [4.88 %]; range = 7.61 % - 25.19 %), which was then included as a moderator. We also calculated achieved statistical power to detect the 95 % lower and upper confidence intervals of the average meta-analytic effect size ($g = 0.13$ and $g = 0.44$). The distribution of statistical power to detect the average meta-analytic effect size estimate is displayed in Fig. 4.

3.4.1. Before removing outlier and influential cases

When outliers and influential cases were retained in the dataset, the sleep stage that restriction targeted significantly moderated the overall meta-analytic effect size ($Q(2, 122) = 11.72$, $p = .003$). Specifically, the size of the effect was larger following SWS-targeted sleep restriction compared to REM-targeted ($\beta = -0.45$, $p = .016$) or indiscriminate ($\beta = -0.68$, $p < .001$) sleep restriction (Table 2).

3.4.2. After removing outlier and influential cases

When outliers and influential cases were removed from the dataset, the overall meta-analytic effect was not significantly moderated by sleep stage or any other potential moderators ($ps > .135$). This is unsurprising upon closer inspection of the outliers and influential cases which show that four of the five largest positive (detrimental) effects of SWS-targeted

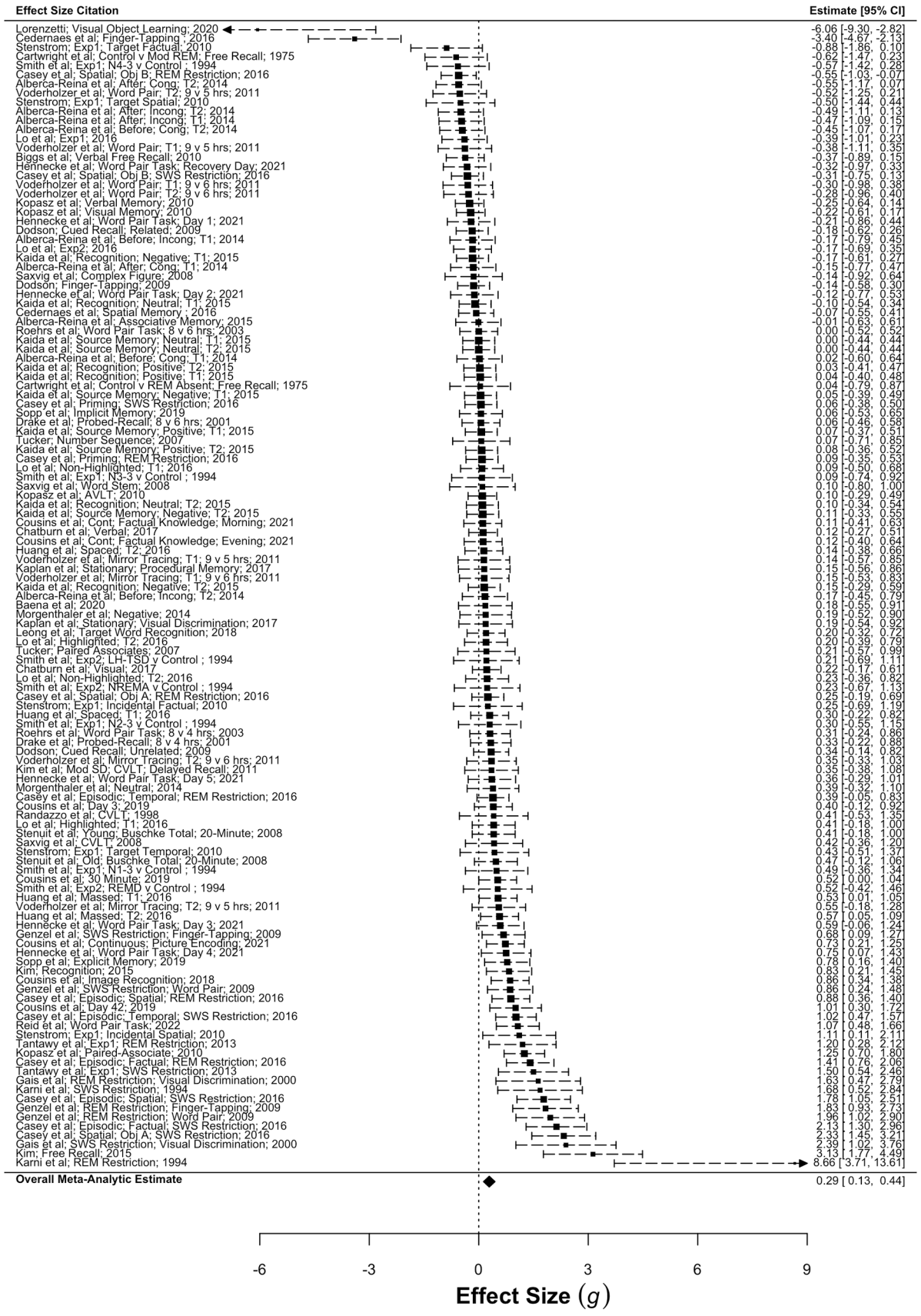

Fig. 2. Forest plot displaying Hedges' g and 95 % confidence intervals for all included effects sizes and for the overall meta-analytic estimate. Positive Hedges' g values indicate a sleep restriction impairment.

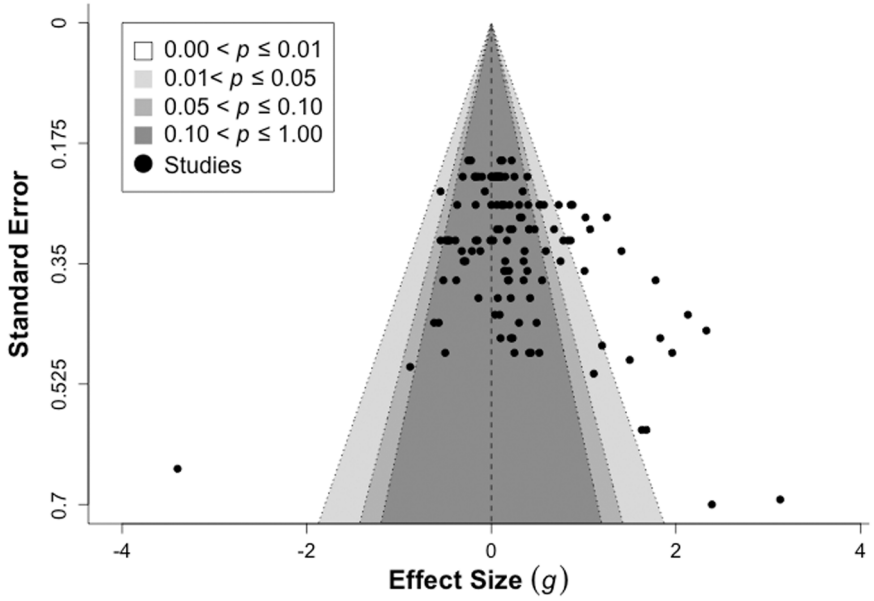

Fig. 3. Contour-enhanced funnel plot displaying the magnitude of Hedges' g for each included effect size on the X-axis according to the standard error of Hedges' g on the Y-axis. Two outliers are excluded. Positive Hedges' g values indicate a sleep restriction impairment.

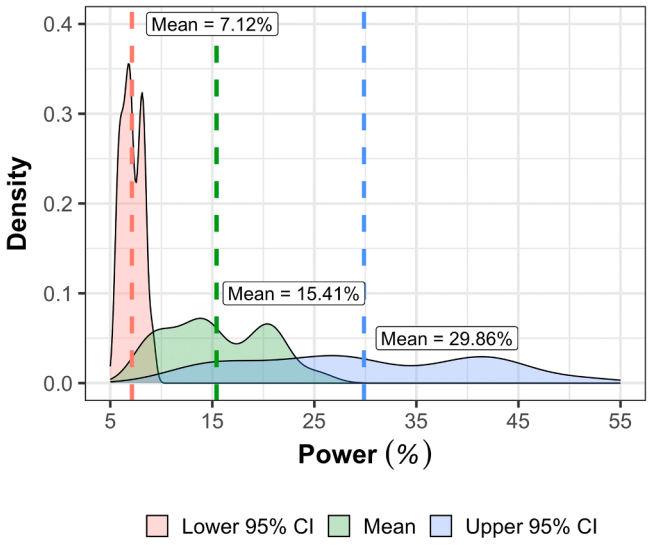

Fig. 4. Distribution of statistical power to detect the average meta-analytic effect size estimate (green; middle), the lower bound of the 95 % confidence interval (red; left), and the upper bound of the 95 % confidence interval (blue; right).

restriction on memory (Hedges' *g*s = 1.68–2.39) and the two largest negative (beneficial) effects of indiscriminate sleep restriction (Hedges' *g*s = −6.06 and −3.40) are removed when outlier and influential cases are removed.

Table 2 reports the *Q* statistic for each test of moderators and the corresponding *p* value. For continuous moderators, Table 2 also displays the change in Hedges' *g* (β) for each one unit increase of the moderator alongside the 95 % confidence intervals. For categorical moderators, Table 2 displays the value of Hedges' *g* alongside the 95 % confidence intervals and corresponding *p* value for each subcategory of the moderator. Figs. 5 and 6 display the distributions of effect size for each categorical and continuous moderator variable.

3.5. Moderator analyses with combined sleep loss data

We chose to combine data from our 125 sleep restriction effect sizes with the 55 total sleep deprivation before encoding effect sizes and 130 total sleep deprivation after encoding effect sizes reported in Newbury et al. (2021), resulting in 310 effect sizes. We did this for two reasons. The primary reason was to include the type of sleep loss (total sleep deprivation vs. sleep restriction) as a moderator to determine whether short sleep has as bad consequences for memory formation as not sleeping at all. Secondly, we ran further analyses on the eight moderators that were shared between the three meta-analyses: recovery sleep (yes vs. no), memory type (declarative vs. non-declarative), task type (recall vs. recognition; note that to match the coding in Newbury et al. 2021, cued recall and free recall were combined into a recall variable and motor skill was dropped), timing of sleep loss (before vs. after encoding), age, normalised score for the reporting cluster of the methodological quality checklist, normalised score for the bias cluster, normalised score for the confounding cluster. To the best of our knowledge, these analyses are the highest-powered of their kind in the sleep loss literature. For clarity and succinctness, all moderator analyses except the type of sleep loss (sleep restriction vs., total sleep deprivation) analysis are reported in the supplementary materials.

We recalculated outlier and influential cases as per the aforementioned criteria. Nine effect sizes were identified as statistical outliers, and 11 more were identified as having a problematic Cook's distance. In sum, 20 effect sizes were identified as outliers or influential cases and were excluded. We report moderator analyses both with and without these 20 outliers and influential cases.

The type of sleep loss did not significantly moderate the overall meta-analytic effect size before outlier and influential cases were removed ($Q(1, 308) = 2.63$, $p = .105$) or after they were removed ($Q(1, 288) = 2.16$, $p = .141$) with the average effect size being similar between sleep restriction and total sleep deprivation before (restriction, $g = 0.28$, 95 % CI = [0.15, 0.41]; deprivation, $g = 0.41$, 95 % CI = [0.31, 0.50]) and after (restriction, $g = 0.25$, 95 % CI = [0.15, 0.35]; deprivation, $g = 0.34$, 95 % CI = [0.27, 0.42]) outlier and influential case removal.

In our main analysis of sleep restriction, we chose to combine data from studies that looked at pre-encoding and post-encoding sleep effects rather than analyse the two separately. This was partly motivated by the lack of a significant moderating effect for timing of restriction in relation

Table 2
Statistics displaying the results of each moderator analysis both when outliers and influential cases are removed from the dataset and when they are retained.

Moderator	*k*	*Q*	β	*g*	*95 % CI*	*p*	*k*	*Q*	β	*g*	*95 % CI*	*p*
	Full dataset with outliers and influential cases retained						*Outliers and influential cases removed*					
N of restriction hours	74	0.39	−0.05		[−0.22, 0.12]	.533	71	0.37	−0.04		[−0.18, 0.09]	.546
N of restriction days	125	0.32	−0.02		[−0.08, 0.04]	.572	116	0.01	0.003		[−0.04, 0.05]	.907
N of recovery nights	125	0.25	0.004		[−0.01, 0.02]	.616	116	0.43	0.004		[−0.01, 0.02]	.514
Power	125	0.91	−0.02		[−0.05, 0.02]	.340	116	0.30	−0.007		[−0.03, 0.02]	.585
Quality - Reporting	125	0.13	0.14		[−0.62, 0.90]	.721	116	1.22	0.32		[−0.25, 0.88]	.269
Quality - Bias	125	0.18	−0.29		[−1.61, 1.03]	.666	116	0.004	0.03		[−1.01, 1.07]	.953
Quality - Confounding	125	1.69	−0.80		[−2.00, 0.40]	.193	116	0.04	−0.10		[−1.01, 0.81]	.833
Age	123	0.09	0.003		[−0.02, 0.03]	.766	114	0.13	0.003		[−0.01, 0.02]	.717
Restriction Method	125	1.82				.177	116	0.37				.542
Timing	80			0.23	[0.05, 0.41]	.012*	76			0.21	[0.08, 0.35]	.002**
Awakenings	45			0.46	[0.17, 0.76]	.002**	40			0.30	[0.07, 0.52]	.012*
Restriction Stage	125	11.72				.003**	116	4.01				.135
Indiscriminate	74			0.17	[−0.02, 0.33]	.030*	70			0.17	[0.04, 0.30]	.013*
SWS	13			0.85	[0.49, 1.21]	<.001***	9			0.49	[0.16, 0.82]	<.001***
REM sleep	38			0.40	[0.15, 0.65]	.002**	37			0.36	[0.15, 0.56]	.001**
Restriction Timing	115	0.71				.400	107	0.16				.693
Before encoding	43			0.24	[0.02, 0.46]	.034*	41			0.21	[0.03, 0.38]	.023*
After encoding	72			0.36	[0.16, 0.56]	<.001***	66			0.25	[0.09, 0.42]	.002**
Recovery	125	0.23				.631	116	0.03				.869
Yes	50			0.25	[0.02, 0.48]	.034*	50			0.22	[0.05, 0.39]	.010*
No	75			0.31	[0.13, 0.50]	.001**	66			0.24	[0.10, 0.38]	.001**
Memory Type	125	0.12				.734	116	0.05				.817
Declarative	100			0.30	[0.13, 0.46]	<.001***	95			0.23	[0.11, 0.35]	<.001***
Non-declarative	25			0.25	[−0.05, 0.54]	.097	21			0.26	[0.02, 0.50]	.034*
Task Type	116			0.87		.832	110	1.27				.737
Cued recall	46			0.24	[−0.03, 0.45]	.029*	45			0.18	[0.01, 0.35]	.037*
Free recall	32			0.26	[−0.01, 0.53]	.060	28			0.27	[0.04, 0.49]	.019*
Recognition	26			0.21	[−0.06, 0.47]	.135	25			0.24	[0.02, 0.45]	.030*
Motor Skill	12			0.45	[−0.02, 0.92]	.059	12			0.39	[0.01, 0.77]	.047*

Note. N = number; k = number of effect sizes, Q = moderator test statistic where H_0 is that the moderator does not influence the meta-analytic effect size; g = Hedges' g for each moderating subcategory of data (categorical moderators); β = change in Hedges' g for one unit increase of the moderator (continuous moderators); 95 % CI = 95 % confidence intervals; *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

to learning, and partly by a desire to avoid reducing statistical power by dividing the data into two separate analyses. However, an advantage of our combined sleep loss analysis is that we can re-visit this issue of sleep loss timing in a substantially larger dataset. To identify those moderators that have different effects during pre- and post-encoding sleep loss conditions, we analysed the interaction between each moderator and the timing of sleep loss. The results are presented in Table 3. Three moderators interacted significantly with timing of sleep loss ($ps < .038$): age, memory type (declarative vs. non-declarative), and sleep loss type (restriction vs. deprivation), although only the last of these survived removal of outliers ($p = .017$).

To understand the implications of these interactions, we then analysed the pre- and post-encoding sleep loss conditions separately. Age ($Q(1, 122) = 4.05$, $p = .044$) and memory type ($Q(1, 200) = 5.73$, $p = .017$) were significant moderators only when sleep loss occurred after encoding, although neither survived removal of outliers. Following post-encoding sleep loss, older participants showed larger memory impairments than younger participants ($\beta = 0.04$) and non-declarative memory was associated with larger impairments than declarative memory (non-declarative, $g = 0.48$, 95 % CI = [0.30, 0.65]; declarative, $g = 0.24$, 95 % CI = [0.14, 0.34]). Sleep loss type (restriction vs. deprivation) was a significant moderator only when sleep loss occurred before encoding but regardless of whether outlier and influential cases were retained ($Q(1, 96) = 12.06$, $p < .001$) or removed ($Q(1, 87) = 11.31$, $p < .001$). Sleep deprivation (outliers retained, $g = 0.63$, 95 % CI = [0.48, 0.77]; outliers removed, $g = 0.48$, 95 % CI = [0.36, 0.59]) was associated with larger impairments to memory than sleep restriction (outliers retained, $g = 0.20$, 95 % CI = [0.01, 0.39]; outliers removed, $g = 0.20$, 95 % CI = [0.01, 0.39]).

4. Discussion

Sufficient sleep has long been thought to be critical for optimal encoding and consolidation of memory (McClelland et al., 1995; Tononi and Cirelli, 2003). Yet, many features of modern society pervasively hamper sleep opportunities (Pallesen et al., 2021; Roenneberg et al., 2007; Skeldon et al., 2017). Sleep scientists have been trying to unravel the costs of short sleep on learning and memory for decades, but comprehensive summaries are so far limited to cognitive domains most susceptible to sleepiness such as attention and decision-making or effects of total sleep deprivation (Banks and Dinges, 2007; Harrison and Horne, 2000; Lim and Dinges, 2008, 2010; Lowe et al., 2017; Newbury et al., 2021; Pilcher and Huffcutt, 1996). Here, for the first time, we provide a comprehensive summary of the sleep restriction and long-term memory literature, we quantify the magnitude of the associated effect size alongside potential moderating variables, and we compare it with the total sleep deprivation effect sizes reported in Newbury et al. (2021). Using multilevel random effects modelling on 125 effect sizes, representing 1234 participants across 5 continents over the last 50 years, we find that restricting participants' sleep to 3–6.5 hours compared to 7–11 hours of unrestricted sleep for 1–14 nights impairs memory formation with a small effect size according to Cohen's guidelines (Hedges' $g = 0.29$ with outliers; $g = 0.23$ without outliers).

4.1. Investigating predictions made by the sleep theories

Here, we investigated the effect of potential moderating variables in an attempt to evaluate the predictions made by existing theories of sleep and memory. The sleep stage that restriction targeted significantly moderated the effect size (only when outliers and influential cases were retained) such that the impact on memory formation was more detrimental for sleep restriction that reduced SWS ($g = 0.85$) compared to REM sleep ($g = 0.40$) or indiscriminate sleep restriction ($g = 0.17$). This finding is of potential theoretical interest. The most dominant sleep theories in the literature – the active systems consolidation theory (e.g., Klinzing et al., 2019) and the synaptic homeostasis hypothesis (Tononi

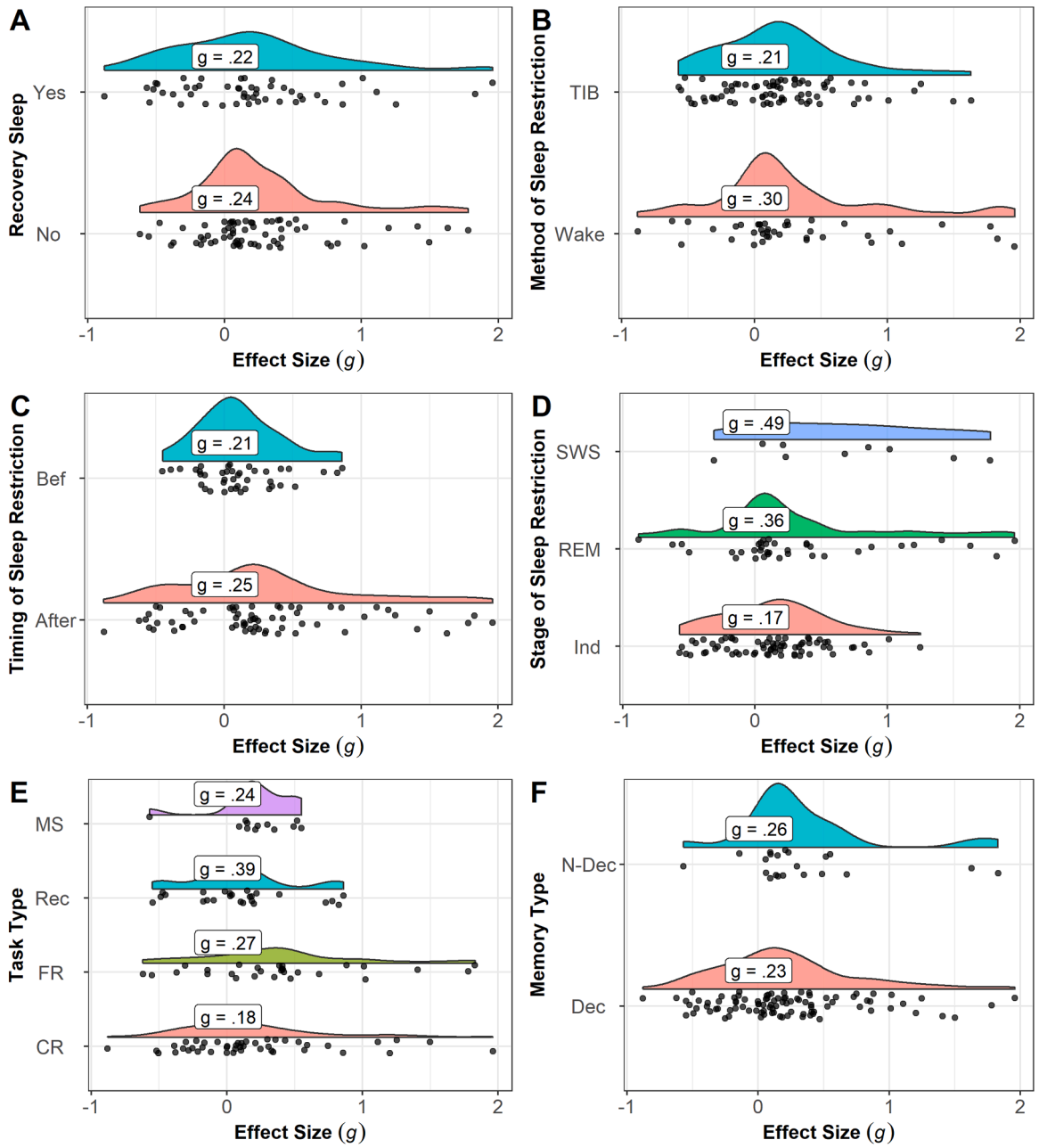

Fig. 5. Raincloud plots showing the distribution of Hedges' g for each categorical moderator after outlier and influential case removal: **A)** recovery sleep, **B)** method of sleep restriction, **C)** timing of sleep restriction, **D)** sleep stage, **E)** task type, and **F)** memory type. Positive Hedges' g values indicate a sleep restriction impairment. TIB = time-in-bed instructions; Wake = awakenings; Bef = before; Ind = indiscriminate; Rec = recognition; MS = motor skill; FR = free recall; CR = cued recall; N-Dec = non-declarative; Dec = declarative.

and Cirelli, 2003) – both prioritise the role of SWS over REM sleep. Specifically, these theories predict that losing SWS, rather than losing REM sleep, would impair encoding capacity (synaptic homeostasis hypothesis) and consolidation ability (both theories). Hence, in light of these theories it is unsurprising that SWS-targeted sleep restriction is the most detrimental for memory. The moderating effect of sleep stage did not persist after outliers and influential cases were removed, however. Whilst outliers and influential cases should not be dismissed (Hunter and Schmidt, 2004), conclusions that hinge on the inclusion of a small number of cases should be considered tentatively (Viechtbauer and Cheung, 2010). Future work should assess the robustness of this moderating effect given its potential theoretical value.

The timing of sleep restriction relative to encoding also did not significantly moderate the overall effect. The cost to memory was similar when participants had short sleep before encoding and after encoding. This finding is of theoretical interest. Much empirical work in the literature is concerned with effects of post-encoding sleep on memory, and theoretical work also concentrates on delineating a role for sleep-dependent memory consolidation after encoding as opposed to processes that occur during sleep prior to encoding. Despite this, the current data would suggest that the benefit of sleep in preparing the brain for encoding, as per the synaptic homeostasis hypothesis (Tononi and Cirelli, 2003), is as important as the benefit of sleep for consolidation purposes. This finding should be treated with caution, however, given that there are methodological differences between the sleep restriction before encoding and the sleep restriction after encoding effect sizes here. For example, nearly all non-declarative memory effect sizes here are from studies that restricted sleep after encoding.

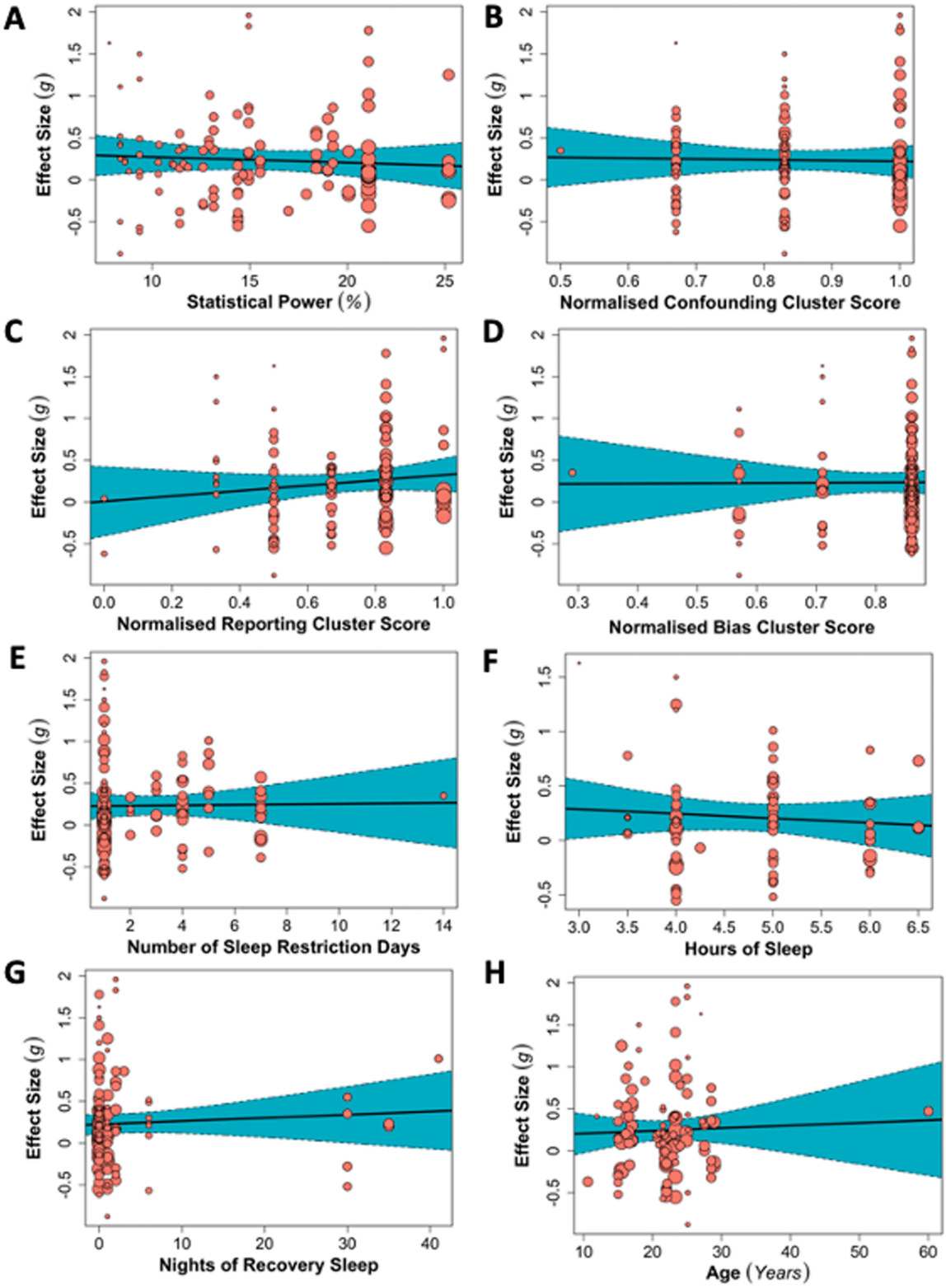

Fig. 6. Meta-analysis regression plots, after outlier and influential case removal, displaying Hedges' g according to **A)** statistical power, **B)** confounding cluster score, **C)** reporting cluster score, **D)** bias cluster score, **E)** sleep restriction days, **F)** sleep restriction duration, **G)** nights of recovery sleep, and **H)** age. Positive Hedges' g values indicate a sleep restriction impairment. The regression line (solid line) and its 95 % confidence intervals (dashed lines) were calculated without outliers. The size of each point is proportional to the weight that each effect size received in the analysis such that bigger points represent larger weightings.

Table 3
Statistics displaying the results of analyses investigating an interaction between the timing of sleep loss and each moderator in the combined sleep restriction and total sleep deprivation data. Where a significant interaction is found, the results of moderator analyses is reported separately for sleep loss before encoding and sleep loss after encoding. Data are reported when outliers and influential cases are removed from the dataset and when they are retained.

Moderator	***k***	***Q***	**β**	***g***	***95 % CI***	***p***	***k***	***Q***	**β**	***g***	***95 % CI***	***p***
Sleep Loss Timing x	*Full dataset with outliers and influential cases retained*						*Outliers and influential cases removed*					
Quality - Reporting	300	1.09				.296	281	0.39				.530
Quality - Bias	300	0.05				.833	281	0.28				.600
Quality - Confounding	300	1.34				.248	281	3.56				.059
Age	194	4.38				.036*	180	2.69				.101
Recovery	300	3.18				.075	281	1.81				.179
Memory Type	300	4.30				.038*	281	3.04				.081
Task Type	245	1.04				.309	232	1.72				.190
Sleep Loss	300	9.22				.002**	281	5.69				.017*
Sleep loss before encoding data												
Age	70	0.60	−0.01		[−0.02, 0.01]	.437	64	0.34	−0.004		[−0.02, 0.01]	.558
Memory Type	98	1.90				.168	89	2.09				.148
Declarative	94			0.49	[0.36, 0.62]	<.001***	85			0.38	[0.27, 0.48]	<.001***
Non-declarative	4			0.19	[−0.23, 0.62]	.373	4			0.08	[−0.31, 0.48]	.683
Sleep Loss	98	12.06				.001***	89	11.31				.001***
Deprivation	55			0.63	[0.48, 0.77]	<.001***	48			0.48	[0.36, 0.59]	<.001***
Restriction	43			0.20	[0.01, 0.39]	.039*	41			0.20	[0.01, 0.39]	.039*
Sleep loss after encoding data												
Age	124	4.05	0.04		[0.001, 0.08]	.044*	116	2.50	0.03		[−0.007, 0.07]	.114
Memory Type	202	5.73				.017*	192	1.77				.184
Declarative	159			0.24	[0.14, 0.34]	<.001***	154			0.25	[0.16, 0.34]	<.001***
Non-declarative	43			0.48	[0.30, 0.65]	<.001***	38			0.37	[0.21, 0.53]	<.001***
Sleep Loss	202	0.61				.434	192	0.14				.705
Deprivation	130			0.27	[0.16, 0.39]	<.001***	126			0.27	[0.17, 0.37]	<.001***
Restriction	72			0.36	[0.18, 0.53]	<.001***	67			0.30	[0.15, 0.46]	<.001***

Note. k = number of effect sizes, Q = moderator test statistic where H_0 is that the moderator does not influence the meta-analytic effect size; g = Hedges' g for each moderating subcategory of data (categorical moderators); β = change in Hedges' g for one unit increase of the moderator (continuous moderators); 95 % CI = 95 % confidence intervals; *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$

In our second analysis, the current dataset was combined with the 185 effect sizes investigating total sleep deprivation in Newbury et al. (2021) which allowed us to statistically assess whether or not the impact of sleep restriction differs from that caused by losing a full night of sleep (note again that the type of sleep loss moderator is the only moderator discussed here from the combined sleep loss dataset, and the remaining are reported in the supplementary materials and Section 4.6 for clarity and succinctness). Crucially, this combined sleep loss moderator analysis revealed that the type of sleep loss did not significantly moderate the magnitude of the detriment to memory (although type of sleep loss may matter when looking at pre-encoding and post-encoding sleep loss separately, see Section 4.6 for a discussion). While we acknowledge that the lack of statistical significance does not necessarily mean that there is evidence for the absence of an effect, and indeed there is a small numerical difference between the conditions ($g = 0.41$ vs. $g = 0.28$ with outliers; $g = 0.34$ vs. $g = 0.25$ without outliers), it does appear that losing just a couple of hours of sleep may have broadly similar consequences for memory as not sleeping at all. This is perhaps further supported by the fact that the number of sleep restriction days and sleep restriction duration did not significantly moderate the effect size in the sleep restriction dataset, suggesting that bursts of short sleep were as bad as prolonged and greater sleep loss. Moreover, these findings align with evidence for no association between the extent of memory benefits and duration of sleep (0.35 – 9.34 hours; Berres and Erdfelder, 2021), as well as the size of the sleep restriction effect for long-term memory reported in previous work ($g = 0.21$; Lowe et al., 2017).

On a theoretical level, the prevailing sleep theories would likely predict total sleep deprivation to impair learning and memory more than sleep restriction. As already discussed in Sections 1.1.1 and 1.2.1, the active systems consolidation theory (e.g., Klinzing et al., 2019) and the synaptic homeostasis hypothesis (e.g., Tononi and Cirelli, 2003) together assume that SWS prepares the brain for new learning and transforms new memories. During total sleep deprivation there is of course no opportunity for SWS, but this is not the case during sleep restriction. Much sleep restriction in the real world (and in the current meta-analysis) is driven by sleep being truncated in the mornings which is particularly problematic for those with a late chronotype (preference for going to bed late in the evening and to wake late in the morning; Goldin et al., 2020; Roenneberg et al., 2007). Brunner et al. (1993) studied this type of sleep restriction (four nights of sleep between 23:00 – 03:00) using EEG, and found that NREM-1, NREM-2 and REM sleep durations were considerably shorter, but SWS duration was preserved (see also Belenky et al., 2003). Others have found that relative durations of SWS increase during periods of restricted sleep, perhaps compensating, to a degree, for reduced total sleep time (Guilleminault et al., 2003). Moreover, 38 (30 %) of the current effect sizes deliberately targeted REM sleep and preserved SWS. It follows then that the effect of sleep restriction on memory, as per the prevailing sleep theories, should be significantly smaller than total sleep deprivation given that considerable SWS still occurs, yet that was not the case in the current data.

Dual process theories (e.g., Smith, 1995) do not fit the current pattern of data well either. According to these theories, SWS and REM sleep are responsible for the consolidation of declarative and non-declarative memories respectively. Accordingly, SWS-targeted sleep restriction (and indiscriminate sleep restriction given Brunner et al., 1993 data) should elicit a clear impairment to declarative memories, and REM sleep restriction should impair non-declarative memories. Such an effect would be represented in the current data by an interaction between sleep stage and memory type moderators but given that memory type did not have a significant moderating effect itself, with declarative and non-declarative memories being similarly affected by sleep restriction, we did not perform this analysis. Furthermore, non-declarative memories are not well-represented in the current meta-analysis with only 25 effect sizes (20 %) assessing this type of memory. It could therefore be that this moderator analysis was rendered insensitive due to the imbalance in number of effect sizes between condition being compared (Hempel et al., 2013). It would be beneficial for future work to populate the literature with studies investigating sleep restriction and non-declarative memory performance to determine whether memory type does moderate the sleep restriction effect and

whether memory type interacts with sleep stage as per the dual process theories.

Similar effects of sleep restriction and total sleep deprivation are perhaps predicted by sequential sleep theories (e.g., Diekelmann and Born, 2010; Giuditta, 2014; Giuditta et al., 1995) however. According to these theories, it is not the occurrence of sleep stages per se which benefits memory, but rather it is interactions between SWS and REM sleep that are critical given they have complementary roles. Specifically, it follows from these theories that if the transition from SWS to REM sleep is disrupted then to-be-remembered information would not be integrated with pre-existing memories (Giuditta, 2014; Giuditta et al., 1995) and synaptic consolidation would not take place following systems consolidation (Diekelmann and Born, 2010). For more than one third of the effect sizes here, sleep restriction was achieved by repeatedly waking participants and therefore the natural progression of the sleep cycle was continuously disrupted. Moreover, when sleep restriction is achieved by reducing time-in-bed, which is most common in real life (Banks and Dinges, 2007) and in the current meta-analysis (80 effect sizes), interactions between sleep stages are still disturbed. First, with decreased time-in-bed, there are fewer iterations of the sleep cycle and thus fewer transitions between sleep stages. Second, late nocturnal sleep which is often lost under such sleep restriction (Roenneberg et al., 2007) is characterised by many more transitions between NREM and REM states than early nocturnal sleep which is preserved (Carskadon and Dement, 2005). In light of this, it is perhaps unsurprising that sleep restriction impairs memory formation similarly to no sleep given that interactions between sleep stages, which are critical under these theories, are considerably altered. In a similar vein, it is unsurprising that the method for achieving sleep restriction did not moderate the sleep restriction effect given that both waking participants and reducing time-in-bed disturb interactions between sleep stages. Notably, the suggestion that transitions between sleep stages are critical fits with growing evidence that populations with fragmented sleep, such as the elderly and psychiatric patients, show limited sleep-related memory benefits (Colvonen et al., 2019; Diekelmann et al., 2009; Mary et al., 2013; Mander et al., 2017; Pace-Schott et al., 2015; Varga et al., 2016).

If short sleep impairs memory formation to a similar extent as total sleep loss, there are practical implications which need to be taken seriously, particularly given that comparable impairments are now documented across most cognitive domains (Berres and Erdfelder, 2021; Lim and Dinges, 2010; Lowe et al., 2017). Consider, for example, adolescents who typically prefer to sleep and wake later than their early school start times allow due to the shift in chronotype associated with this phase of development (Bowers and Moyer, 2017; Roenneberg et al., 2004; 2007). Insufficient sleep is rife in this population with some estimates suggesting that 58 % – 73 % of high-school students sleep for less than the minimum recommendation for their age (Wheaton et al., 2018; see also Peltzer and Pengpid, 2016). This problem is exacerbated during exam season when these estimates rise to 94 % (Wang et al., 2016) and sleep becomes more fragmented (Dewald et al., 2014). These proportions are stark in the context of learning and memory because this suggests a large proportion of adolescents suffer severe costs to their academic performance when it matters most. The need to promote sleep hygiene amongst adolescents is becoming increasingly recognised (Dietrich et al., 2016), but the current data suggest more is needed. If an adolescent who stays up a few hours late (as most students do to cram for exams; Walker, 2006) suffers similar costs to their memory as their peer who walks into the exam hall having not slept at all, urgent interventions are needed to better help adolescents understand these consequences. This suggestion that sleep should not be sacrificed for cramming is supported by evidence showing that recall is impaired following sleep restriction even in the presence of cramming (Huang et al., 2016) and that sleep is more beneficial for long-term retention than cramming (Cousins et al., 2019). In a similar vein, promoting sleep health in the employment sector should not be limited to professions prone to losing entire nights of sleep due to night shift work for example, given that almost all professions can put pressure on sleep and wake times.

4.2. Recovery sleep

The prevailing sleep theories do not make varying predictions regarding the impact of recovery sleep on sleep restriction effects, but we report these findings for completeness. The occurrence of recovery sleep and the number of recovery sleep nights were not found to be significant moderating variables here. In other words, long-term memory was significantly impaired by sleep restriction regardless of whether there was an opportunity for catch-up sleep before testing or not. This is in contrast to the total sleep deprivation after encoding meta-analysis in Newbury et al. (2021) where the occurrence of recovery sleep before testing reduced the impairment on memory significantly, by more than 50 % (recovery sleep; $g = 0.18$, no recovery sleep; $g = 0.41$). It may well be, therefore, that impairments to memory following sleep restriction are less easily ameliorated by recovery sleep than impairments caused by total sleep deprivation. In fact, some evidence suggests that just two nights of recovery sleep is enough to reverse behavioural and psychological effects of acute sleep debt (as is caused by short bursts of total sleep deprivation; Dinges et al., 1997), whereas reversing the effects of three weeks of chronic sleep debt (as is caused by prolonged sleep restriction) requires more than one week of recovery sleep (Ochab et al., 2021).

We did statistically investigate this in the combined sleep loss dataset but no significant interaction between recovery sleep and sleep loss type was found. It is important to note, however, that statistical power for interaction terms in moderator analyses is particularly poor. In a similar vein, the lack of moderating effect for recovery sleep in the sleep restriction analyses may well have been driven by poor statistical power. However, the current sleep restriction meta-analysis and the total sleep deprivation after encoding meta-analysis in Newbury et al. (2021) are comparable across a number of factors known to affect statistical power of moderating effects including number of effect sizes and balance in the number of effect sizes between moderator levels (Hempel et al., 2013). Clearly, future work should investigate this systematically, but the implication here is that lengthening individual nights of sleep (before an exam, for example) is unlikely to successfully reverse the effects of multiple nights of short sleep.

4.3. Statistical power

Careful consideration of statistical power is important in meta-analytic research because whilst a meta-analysis might consist of hundreds of effect sizes, if they are all based on severely underpowered studies, uncertainty around the average effect size grows. In the sleep restriction dataset here, average statistical power to detect the meta-analytic estimate of $g = 0.29$ was very low (mean [*SD*] = 15.41 % [4.88 %]; range = 7.61 % - 25.19 %). These figures are somewhat higher than those typically found in psychology and cognitive neuroscience (Szucs and Ioannidis, 2017), but the widely accepted convention is still much higher at 80 % (di Stephano, 2003). Low statistical power is a non-trivial issue for meta-analytic estimates because it can cause them to be underestimated or overestimated, and it is difficult to determine which is the case. On the one hand, underpowered studies are less likely to detect true effects which may deflate meta-analytic estimates (Fraley and Vazire, 2014). On the other hand, a literature dominated by underpowered studies is likely to suffer from a greater proportion of false positives which may inflate the overall meta-analytic effect (Fraley and Vazire, 2014; Wilson et al., 2020). Statistical power to detect the meta-analytic estimate was included as a moderator here, but the results did not shed light on whether better powered studies elicit smaller or larger sleep restriction effects. Statistical power did not significantly moderate the overall meta-analytic estimate, and this is unsurprising given that statistical power in this analysis ranged from just 7.61 % – 25.19 %. Hence, these data give no indication as to the size of sleep

restriction effects that could be expected if the studies investigating them are statistically powered anywhere near the 80 % convention. We echo Newbury et al. (2021) in that it is now essential for sleep science to move towards replication science and use better-powered studies to more accurately estimate the true effect size.

4.4. Publication bias

On top of low statistical power, there is concern that the resource-intensive nature of sleep research may exacerbate issues of publication bias in this field. Publication bias occurs when there is a preference for publishing statistically significant findings that are in the direction of hypotheses rather than non-significant findings or findings that contradict the hypotheses (Rosenthal, 1979). This causes inflated meta-analytic estimates and problems with replicability if published effects are predominantly false positives. To tackle this, we systematically investigated the extent to which publication bias characterises the last 50 years of sleep restriction research using four methods: visually inspecting contour-enhanced funnel plots, Egger's regression test, the trim-and-fill method, and the three-parameter selection model. Whilst these approaches each have their limitations (see Methods section), together their results reveal no conclusive evidence for publication bias. Hence, selective reporting of hypothesised and significant findings does not seem to compromise the validity of the sleep restriction impairment on learning and memory.

While the absence of evidence for publication bias is positive, the sleep science community must continue to combat practices that could lead to bias. Pre-registration and registered reports are key tools for encouraging non-significant findings being published, but the uptake of open science practices has been slow in sleep research (Németh et al., 2024; Spitschan et al., 2020). Although high-powered replications (e.g., Denis et al., 2022) and registered reports (e.g., Morgan et al., 2019; March et al., 2023; Mak et al., 2023) are now beginning to appear in the broader sleep and memory literature, none of the sleep restriction studies in this meta-analysis were pre-registered and only one performed an a priori power analysis to justify sample size.

4.5. Methodological quality

The reliability and validity of conclusions drawn from meta-analytic and systematic reviews depends on the scientific rigour of included studies. For this reason, best practice guidelines increasingly recommend that methodological quality is assessed and reported (e.g., AMSTAR, Shea et al., 2017; PRISMA, Moher et al., 2009, see also Johnson and Hennessy, 2019). To assess methodological quality here, we adapted the 22-item checklist used in Newbury et al. (2021), and the items were clustered and transformed into a risk of bias rank (see Methods section) according to Valentine (2009) and Stone et al. (2020). Assessing methodological quality in this way not only allows for an overview of scientific rigor in the field, but it also allows for methodological quality scores to be included in moderator analyses to determine the size of effects yielded from higher quality studies (as per Cochran Collaboration recommendations; Higgins et al., 2011).

On average, quality scores were highest for the confounding cluster which assesses biases in the selection and allocation of participants, followed by the bias cluster which assesses biases in experimental procedures, and finally the reporting cluster which assesses information provided regarding participant characteristics. Methodological rigour varied considerably within each cluster. For example, Reid et al. (2023) satisfied all 6 items of the reporting cluster by ensuring that participants had no neurological, psychiatric or sleep disorders, had not recently taken medication known to affect sleep or travelled across time zones, and were matched according to chronotype, whereas Cartwright et al. (1975) did not satisfy any. Despite this, scores were not well-represented across the range for each cluster which likely rendered the moderator analyses insensitive. Only 3 effect sizes score below 0.67 on the confounding cluster, scores on the bias cluster range from 0.29 to 1 but over three quarters of the effect sizes scored 0.86, and only 11 effect sizes score below 0.5 on the reporting cluster with over a third scoring 0.83. Consequently, the null effects found for the methodological quality moderator analyses must be treated with caution.

There are additional participant characteristics which are relevant to studies of sleep restriction which we did not include in the reporting cluster here. For example, stimulant consumption, such as alcohol, caffeine, and nicotine, might attenuate sleep restriction effects. Similarly, effects of sleep restriction may differ in individuals habituated to nightshift work. These characteristics were not included in the current methodological quality checklist, but they would be unlikely to add to its diagnosticity given that studies accounting for these characteristics also accounted for other characteristics that we did include, such as excluding participants who take medication known to affect sleep and matching sleep restriction and control groups for chronotype. For the interested reader, though, approximately half of the current effect sizes used exclusion criteria related to alcohol (44 %) and caffeine (59 %) consumption. Only 4 effect sizes considered nicotine use and whilst 33 (26 %) effect sizes excluded participants engaged in nightshift work, these effect sizes came from a group of 5 individual studies. We would urge future sleep restriction research to better control for (or report) characteristics such as nicotine use and nightshift work given their potential confounding influence on sleep restriction effects.

4.6. Sleep loss before and after encoding

In our analysis of sleep restriction, we combined studies investigating effects of sleep restriction before and after encoding into a single meta-analysis. This contrasts with Newbury et al. (2021) where studies investigating total sleep deprivation before encoding and after encoding were separated into distinct meta-analyses. The key reason for combining effect sizes here was to avoid uncertainty around moderating effects due to low statistical power given that only a relatively small number of studies (43 effect sizes) investigated sleep restriction before encoding. Additionally, there were several studies where sleep was restricted both before and after encoding. The lack of significant moderating effects for sleep loss timing in both the restriction analysis and the combined restriction and deprivation analysis appears to vindicate this position. However, given that the neural processes underpinning the role of sleep in memory formation are thought to differ before and after encoding and are largely addressed by different theories, combining effect sizes in this way might mask potentially informative moderating effects. For this reason, we ran a further, more targeted moderator analysis on our combined sleep loss data (restriction and total sleep deprivation) as this larger dataset benefits from higher statistical power. Specifically, we investigated whether each of our original moderators showed a significant interaction with the timing of sleep loss (before vs. after encoding) to establish whether analyses looking at each moderator at each timing of sleep loss condition separately was justified.

Before outliers and influential cases were removed, both age and memory type (declarative vs. non-declarative) interacted significantly with the timing of sleep loss. Sleep loss after encoding impaired memory consolidation more in older participants and in non-declarative memory studies. Neither moderated the effect size in sleep loss before encoding. Critically, only four effect sizes assessed non-declarative memory following sleep loss before encoding compared to 94 declarative memory studies. Only 70 effect sizes reported age, and whilst age ranged from 11 – 60 years, 60 % of effect sizes involved 20–25-year-olds. Additionally, neither the moderating effect for timing of sleep loss and age or memory type survived after outliers were removed. We are therefore reluctant to draw strong conclusions about memory type or age in this analysis, but future work should address this gap in the literature given that much of the current literature focuses on sleep loss effects in students and for declarative memories. Instead, older

individuals and non-declarative memories could be more vulnerable to such effects.

The timing of sleep loss also interacted significantly with the type of sleep loss (restriction vs. deprivation), and this was the case regardless of whether outliers were removed or retained. Specifically, sleep restriction and total sleep deprivation after encoding affected memory similarly, whereas sleep deprivation before encoding impaired memory significantly more than sleep restriction before encoding. There are limitations to this finding because only 98 effect sizes investigated sleep loss before encoding compared to 202 effect sizes investigating sleep loss after encoding. It is possible that this moderating effect could change with a more balanced set of studies (Hempel et al., 2013). Current theories do not make explicit predictions about the impact of sleep deprivation versus sleep restriction. Our data however suggest that the impact of interfering with sleep before encoding on subsequent memory may be dose dependent (for similar findings in working memory, see Belenky et al., 2003; Short et al., 2018; Van Dongen et al., 2003), whereas interfering with sleep after encoding to any extent will impair memory. In the context of the synaptic homeostasis hypothesis (Cirelli and Tononi, 2021; Tononi and Cirelli, 2003, 2006, 2012), perhaps the degree of synaptic downscaling, and therefore the degree of encoding capacity restoration, is proportional to the amount of sleep (likely the amount of SWS) experienced. In the context of consolidation theories (Klinzing et al., 2019; Kumaran et al., 2016; McClelland et al., 1995), perhaps losing just some sleep is enough to disrupt the hippocampal-neocortical interactions that memory consolidation relies on, especially if such interactions depend on intact sequences of sleep stages. If future work replicates these effects, it will be critical for sleep scientists to map out the precise relationship between the amount of sleep loss and the degree of memory deficit, including whether this relationship is linear or nonlinear.

4.7. Limitations

There are limitations to the current meta-analysis which should be acknowledged. Whilst we attempted to search comprehensively for grey literature to mitigate against publication bias, only seven reports that fit our screening criteria were identified (5 unpublished dissertations and 2 preprints). Clearly, unpublished literature may simply not exist when it comes to sleep research and the lack of statistical evidence for publication bias here would support this. This also makes practical sense because sleep studies are time and resource intense, therefore there is considerable incentive for publication irrespective of the results. Despite this, the possibility remains that unpublished sleep restriction literature does exist and would bring the validity of the current findings into question.

The inclusion and exclusion criteria we employed here may have biased our findings and undermined their generalisability. Whilst our data are taken from five continents, we included only reports written in English which runs the risk of introducing bias (mono-language bias; Johnson, 2021). For example, it is unsurprising then that 51 % of effect sizes reporting location were conducted in Europe and 72 % were conducted in western cultures more generally. If vulnerability to effects of sleep restriction differs cross-culturally, the generalisability of our findings would be severely limited by including mostly western samples. Although there is no evidence, to the best of our knowledge, to suggest that effects of sleep restriction vary between cultures, this is an important consideration given that many inter-individual differences do impact vulnerability to cognitive effects of sleep loss (Maire et al., 2014; Rupp et al., 2012; Van Dongen et al., 2004) and nightly sleep durations are known to differ between cultures (Cheung et al., 2021). It would be beneficial for future research to systematically compare effects of sleep restriction between western and non-western cultures to determine whether findings are comparable and can be generalised across cultures.

Our inclusion criteria were further limited to healthy, typical populations, excluding those with sleep disorders and psychiatric conditions. This was deliberate in light of growing evidence that the mechanisms underlying sleep memory benefits differ in these populations (Cellini, 2017; Manoach and Stickgold, 2019). Hence, including these populations might invalidate conclusions that can be drawn with regards to how sleep restriction typically affects memory. However, short sleep durations are most commonly found in these populations (Cohrs, 2008; Reynolds and Banks, 2010), and therefore, a better understanding of the consequences of sleep restriction for populations that it most affects is now warranted. Additionally, we aimed to include sleep restriction data representing the entire lifespan, but only one effect size in our final dataset had a mean age above 30 years old. 31 (25 %) effect sizes investigated under 18-year-olds and the remaining investigated 18- to 40-year-olds, therefore it is impossible to infer how the sleep restriction effect might look in older age. Elderly individuals tend to have more fragmented and shorter sleep than younger individuals and reduced SWS durations (Carskadon et al., 1982; Feinberg and Campbell, 2010; Ohayon et al., 2004). Hence, it could be that the current findings do not generalise to older populations who may be more tolerant to sleep restriction effects because they habituate to disturbed sleep across healthy ageing (Bliese et al., 2006; Skeldon et al., 2016), or perhaps effects are heightened in the elderly given their prior history of short sleep. Future work should address this.

To the best of our knowledge, the moderator analyses reported here are the highest-powered of their kind in the sleep literature, yet power limitations still exist. On the one hand, the lack of significant moderating variables (after outlier and influential case removal) might reflect the robustness of the sleep restriction effect in that its size is not impacted by differences in methodology. On the other hand, moderator analyses are highly sensitive to statistical power because they depend on smaller subsets of data and are impacted by number of effect sizes, number of participants per effect size, residual heterogeneity, and imbalance in the number of effect sizes between moderator levels (Hempel et al., 2013). This is relevant here because, for example, there were four times as many declarative memory studies as there were non-declarative memory studies, only 13 effect sizes (10 %) used SWS-targeted sleep restriction, and only 12 effect sizes (10 %) investigated motor skill. Similarly, our continuous moderators such as statistical power, age, sleep restriction days, sleep restriction hours, and methodological quality clusters are not well-represented across their entire range which causes the validity of these analyses to be questioned given that we cannot show how effect sizes change beyond the limited ranges that the current data represent, and future work should investigate this. For example, 75 (60 %) of the effect sizes here restricted sleep for just a single night and only one effect size restricted sleep for more than 7 nights. Similarly, only 74 effect sizes reported the duration of sleep restriction and more than one third of these restricted sleep to 5 hours per night. Further, sleep loss is predicted to disproportionately impair encoding and consolidation of emotional memories over neutral memories (Crowley et al., 2019; Lipinska et al., 2019; Tempesta et al., 2018; although no moderating effect of emotionality was found following total sleep deprivation in Newbury et al., 2021), yet the number of effect sizes manipulating emotionality here was insufficient to assess this (emotional, $k = 11$; neutral, $k = 5$).

It is important also to mention that three out of nine effect sizes identified as outliers or influential cases were from studies assessing texture discrimination ability whereby participants must identify a property of a target item embedded amongst distractors. Only five texture discrimination effect sizes were entered into the current meta-analysis before three of them were removed as outliers. This raises the question as to whether they occurred by chance or whether performance on texture discrimination tasks is particularly susceptible to disruption by the type of sleep restriction seen here. In fact, Mednick et al. (2003) demonstrated that performance on a texture discrimination task improved 10 times more when an intervening nap consisted of both SWS and REM sleep as opposed to only SWS. Mednick et al. (2003) proposed that SWS is needed to stabilise performance on texture discrimination

tasks whilst subsequent REM further facilitates it. We have seen that sleep restriction often disrupts the cyclic succession of SWS and REM sleep stages, and if such dependence is critical for texture discrimination tasks, it could be that the large effect sizes in the current meta-analysis represent meaningful variability. Research must now populate the sleep restriction literature with texture discrimination tasks so as to further investigate this.

The current meta-analysis combines sleep restriction data with the total sleep deprivation data in Newbury et al. (2021) to determine whether the effect of short sleep is as detrimental to learning and memory as complete sleep loss in an attempt to adjudicate between predictions that follow from prominent sleep theories, namely the active systems consolidation theory, the synaptic homeostasis hypothesis, dual process theories, and the sequential hypothesis. We acknowledge however that the current meta-analysis is based on behavioural data only. Future meta-analyses using neuroimaging data (e.g., assessing hippocampal activity and neural replay) and determining the moderating effect of sleep parameters (e.g., length of specific sleep stages, slow wave activity, sleep spindles) will be invaluable for further comparing the relative predictions made by each of these theories (see Kumral et al., 2023, for a recent example).

Finally, it must be acknowledged that there may be differences between total sleep deprivation protocols and sleep restriction protocols which limit the extent to which they can be directly compared in a moderator analysis. However, it seems to us that the sleep restriction data reported here are relatively comparable with the experimental and methodological designs that constitute Newbury et al.'s (2021) total sleep deprivation effect sizes. For both types of sleep loss protocol, gender was split equally, and the average age was 22–23 years old. Note however that the age range was greater here with the youngest age being 10-years-old versus 18-years-old in Newbury et al. (2021) who restricted their meta-analysis to adult studies. Moreover, total sleep deprivation studies tended to use between-subjects designs more often (75 %) than sleep restriction studies (60 %). In terms of task type, roughly 90 % of sleep restriction and total sleep deprivation effect sizes investigated declarative memory although these were evenly split between recall and recognition tasks in Newbury et al. (2021) whereas three quarters of the sleep restriction effects here were recall tasks. Finally, in terms of experimental procedure, 60 % of the effect sizes here restricted sleep for just one night which is similar to the number of studies that deprived participants of total sleep for just one night in Newbury et al. (2021), and a similar number of effect sizes employed recovery sleep here (60 %) and in total sleep deprivation effect sizes (47 %). It would be beneficial for future meta-analyses to further investigate which factors might vary between sleep loss protocols and cause the underlying true effect sizes to vary.

4.8. Conclusion

The data presented here represent the largest systematic investigation and quantification of the effect of sleep restriction on long-term memory. The findings show that sleeping for 6.5 hours or less impairs the brain's ability to strengthen and transform new memories, and to restore learning capacity for the next day. When these data were combined with 185 total sleep deprivation effects, short sleep after memory encoding was found to impair memory formation to a similar extent as not sleeping at all. Depriving learners of sleep prior to memory encoding had a larger impact than merely curtailing their sleep, although the latter did also result in significant memory impairment. These findings are of considerable theoretical importance because they are not explicitly predicted by current sleep theories and may fit best with sequential sleep theories which emphasise complementary roles of SWS and REM sleep in learning and memory (e.g., Diekelmann and Born, 2010; Giuditta, 2014; Giuditta et al., 1995). Future work should attempt to better understand the consequences of sleep restriction for the elderly, psychiatric patients, and individuals with sleep disorders given that these population struggle with short sleep as much as students but are often side-lined. For sleep hygiene interventions to successfully educate the public about the cognitive consequences of short sleep, future meta-analyses will be critical (once statistical power has improved) for delineating which moderating factors ameliorate and worsen the impairments.

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Declarations of Interest

None

Author note

All meta-analytic data, analysis scripts and research materials are available at https://osf.io/j8sqx/

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2024.105929.

Data Availability

The link to our data/code is presented in text

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