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Theoretical Review

A meta-analytic investigation of the effect of sleep deprivation on inhibitory control

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ABSTRACT

Sleep deprivation may have a deleterious effect on inhibitory control; however, this effect is not consistent across studies. To arrive at an overall estimate of the relationship between sleep deprivation and inhibitory control, this report used meta-analysis to summarise the magnitude of the effects of sleep deprivation on inhibitory control as measured by the Go/No-Go and Stop Signal Tasks. These are two widely used tasks in the literature. A systematic search of four databases (APAPsycINFO, Medline, CINAHL and Embase) from their inception to November 2023 identified 24 studies involving 712 healthy individuals. Separate random-effects models were used to estimate the effect size of sleep deprivation on performance in these tasks. The meta-analysis revealed a moderate negative effect of sleep deprivation on inhibitory control in both the Go/No-Go and Stop Signal Tasks. Given the importance of inhibitory control in everyday behaviour, future research should investigate the neural and neurophysiological mechanisms underlying this relationship and explore its impact in clinical populations.

2. Introduction

Considerable research has demonstrated that poor sleep impairs a range of cognitive functioning related to learning and memory [1,2]. There is also evidence suggesting poor sleep may negatively affect inhibitory control [3–9]. Yet, the detrimental effects of sleep deprivation on inhibitory control have not been consistently replicated [10–14], indicating the need for a meta-analysis to quantify the magnitude of this effect across studies.

2.1. The inhibitory control system

Inhibitory control (IC) refers to a collection of neural processes evoked in order to supress or change a prepotent response [15,16], following a cue signalling that the response is no longer appropriate. For instance, when driving a car, an environmental signal (e.g., a red stop signal/traffic sign) requires either the cancellation of a pre-potent motor response (pressing down on the accelerator) or switching your response (moving to the break). Failure to do so can have negative consequences. IC is critical to human behaviours, underlying almost all behavioural and emotional regulation responses [17,18]. It has recently been identified as one of the core cognitive mechanisms that underpin performance under pressure [19].

The IC system is supported by the prefrontal cortex (PFC); specifically, the frontostriatal network is critical for successful inhibition [20–25]. Hampshire and colleagues [22,23] suggest that the frontostriatal network exerts IC through modulating local lateral inhibition processes that occur universally throughout the cortex. Although studies suggest that the frontostriatal network is involved in IC in a broad sense, different task paradigms may engage various brain regions to optimise IC performance [26–28]. For instance, during action suppression/restraint, inhibition is more reliant on the activation of the right inferior frontal cortex, right middle frontal gyrus and parietal regions. However, when suppressing an already initiated response, inhibition is strongly associated with activation in the right inferior frontal cortex, left anterior insula and thalamus [25,29]. The varying activation patterns of neural regions across tasks indicate that distinct components of IC may be differentially affected by sleep deprivation.

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Abbreviations

CI	confidence interval
GNG	Go/No-Go Task
IC	inhibitory control
PFC	prefrontal cortex
RCT	randomised controlled trial
SSRT	stop signal reaction time
SST	Stop Signal Task

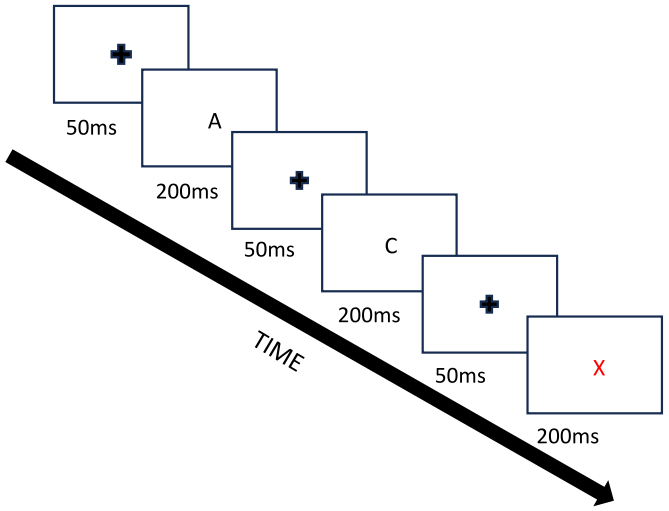

Fig. 1. An overview of the Go/No-Go task. In this example, participants need to respond to a stream of letter stimuli (Go) except for the letter X (No-Go). This figure serves as an example of the Go/No-Go task but does not represent the exact task designs used in the studies included in the meta-analysis.

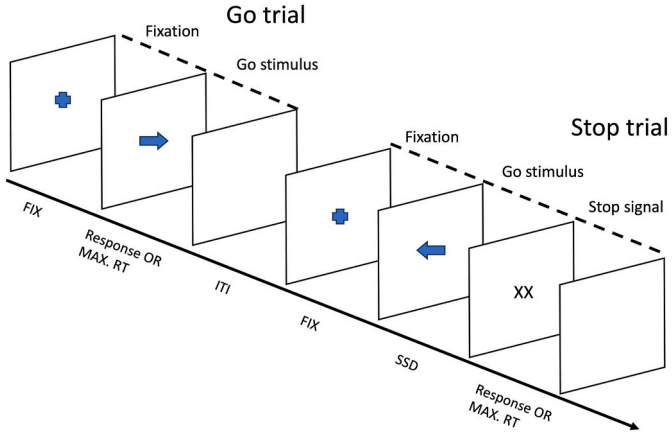

Fig. 2. An overview of the sequence of events in a Stop-Signal Task. On a majority of trials, participants respond to the direction of blue arrows by pressing corresponding arrow key. On a minority of the trials, the arrow is replaced by "XX" after a variable stop-signal delay and participants are instructed to stop their response. FIX = fixation duration; ITI = inter-trial interval; MAX. RT = maximum reaction time; SSD = stop-signal delay. This figure serves as an example of the Stop-Signal Task but does not represent the exact task designs used in the studies included in the meta-analysis. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

2.1.1. Go/no-go and Stop-Signal Tasks are commonly used to study inhibitory control

IC is most widely studied using the Go/No-Go (GNG) [30] and Stop-Signal Tasks (SST) [31]. These tasks assess the ability to suppress a prepotent response when prompted by a no-go and stop signal cue respectively. Other tasks which do have some inhibitory control component such as the Stroop or Flanker are thought to predominantly capture attentional processes rather than inhibitory control [32–35].

A summary of the GNG is presented in Fig. 1. In this task, participants are instructed to press a button as quickly as possible to letters "A, B, C, D" (Go trials) and refrain from pressing the button when presented with the letter "X" (NoGo trials). The NoGo trials require participants to inhibit their automatic response. IC performance on the task is assessed by the number of mistaken responses a participant makes on NoGo trials (commission errors). Fig. 2 presents an overview of the SST. During this task, participants are typically asked to perform a quick response, such as pressing a button, when they observe a visual cue such as a green arrow (Go Signal). Unexpectedly, on some trials, a stop signal such as a red arrow or an auditory cue, appears shortly after the go signal, instructing participants to withhold their response. The primary measure of interest is the stop-signal reaction time (SSRT), which estimates the time required to inhibit an ongoing (already started) response. While other indices, such as omission errors, offer useful information about attention and task engagement [36,37], commission errors and SSRT more accurately represent failures in inhibitory control. Therefore, we have limited our focus to these indices in the current meta-analysis.

Evidence suggest the GNG and SST capture different aspects of IC [26–28]. Specifically, GNG appears to capture processes related to "action restraint", that is, restraining a strong response tendency after observing a signal to not initiate the response. SST, however, captures processes related to "action cancellation", that is, cancelling an ongoing response when a signal to stop occurs.

2.2. The effects of sleep deprivation on the inhibitory control system and related processes

It has been proposed sleep deprivation may impair IC processes [38–40]. As noted earlier, the IC system is supported by the structures that comprise the PFC. Of the many functions of sleep, it is widely believed to restore neuronal function worn down by wakefulness and to regulate connectivity of prefrontal brain networks [41–43]. The brain is actively working throughout wakefulness, and over time, neurons become fatigued [44]. Certain brain regions, such as the PFC, are particularly susceptible to the effects of prolonged wakefulness or insufficient sleep, leading to difficulties in focusing on tasks [45], memory and concentration issues [1] and troubles supressing inappropriate responses [38–40]. Sleep provides an opportunity to restore neurons to their optimal state so that it can continue to function normally [41,46,47]. This restoration process includes repairing cell damage, synthesising proteins and allowing neurotransmitters – chemicals that transmit signals in the brain – to "rest" and regain sensitivity [44, 48]. Sleep deprivation may disrupt these crucial processes, thereby reducing the efficiency or effectiveness of IC processes.

The effects of sleep deprivation on IC processes have been widely studied in laboratory settings using total sleep deprivation or partial sleep restriction protocols. In total sleep deprivation protocols, participants are kept awake continuously for a period ranging from 24 to 72 h [49]. In partial sleep restriction protocols, participants' sleep time is restricted over consecutive nights (typically 2–4 h restriction per night) [47]. These sleep deprivation protocols have been widely used to study the effects of sleep deprivation on brain/executive functioning [49–51]. The results of these studies were summarised in a review of the functional magnetic resonance imaging literature by Ma and colleagues [51]. This review found total sleep deprivation and partial sleep restriction protocols were associated with reduced brain activation in the PFC; a key structure supporting the IC system. At the behavioural level, total

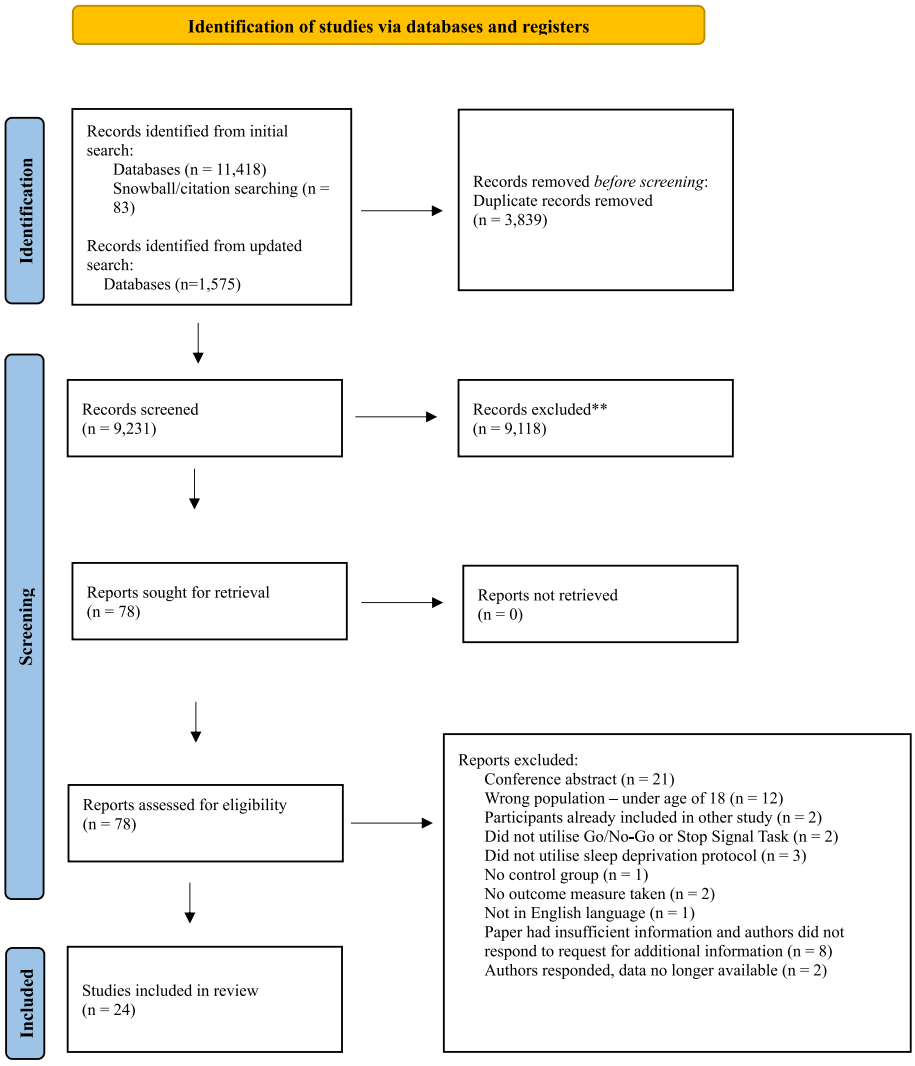

Fig. 3. PRISMA flowchart.

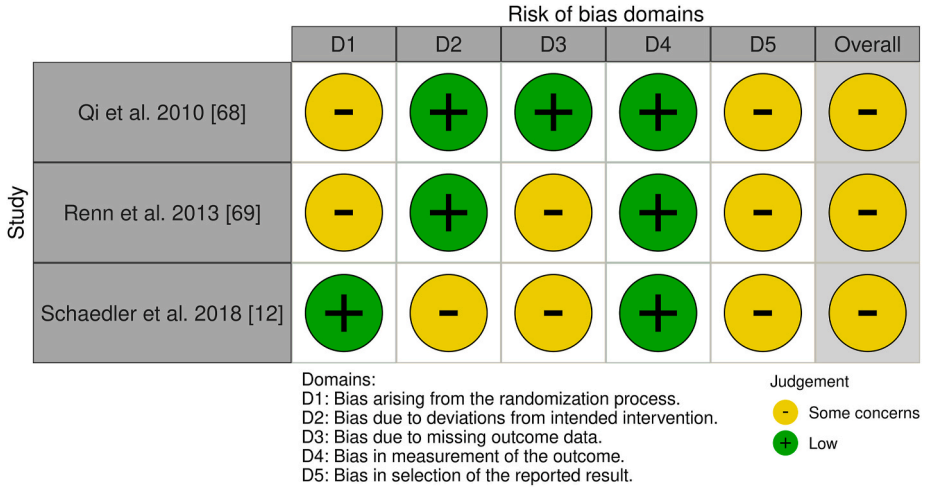

Fig. 4. Summary of the risk of bias plot of included randomised controlled trials.

sleep deprivation or partial sleep restriction protocols appear to have mixed effects on GNG and SST performance. For example, several studies have demonstrated that GNG and SST performance are negatively affected by either protocol compared to uninterrupted sleep [3–9]. These findings, however, have not been universally replicated. A number of studies have found total sleep deprivation or partial sleep restriction protocols have no effect on GNG and SST performance [10–14].

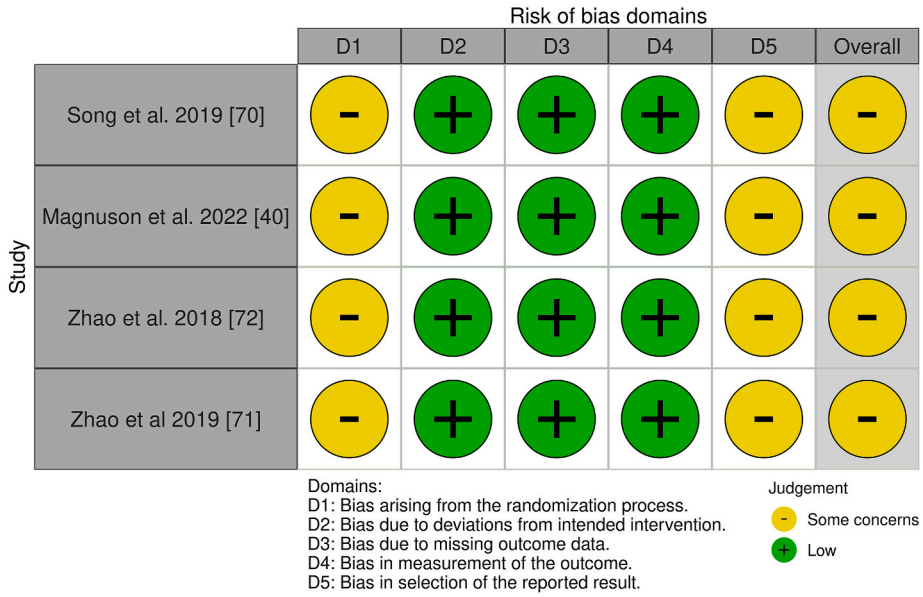

Fig. 5. Summary of the risk of bias plot of included randomised cross-over trials.

2.3. Prior meta-analysis

To our knowledge, no meta-analysis has been conducted on the effects of sleep deprivation on IC. Guarana and colleagues [52] conducted a meta-analysis on the correlational relationships between sleep and IC. Specifically, they found significant correlations between IC and sleep quality (between-individual: 0.26, confidence interval (CI): 0.21 to 0.31; and within-individual: 0.36, CI: 0.24 to 0.45) and sleep duration (between-individual: 0.41, CI: 0.07 to 0.21; and within-individual: 0.20, CI: 0.09 to 0.31). Building upon previous work, the current meta-analysis investigated and quantified the specific impacts of sleep deprivation on IC, thus providing a more in-depth understanding of this relationship and contributing valuable insights to the existing literature.

2.4. Aim of the current meta-analysis

The aim of this report was to examine the relationship between sleep deprivation and performance on IC tasks. Meta-analysis was used to summarise the results of studies that used either total sleep deprivation or partial sleep restriction protocols to study the effects of sleep deprivation on GNG and SST performance in healthy adult samples. Only GNG or SST were included as measures of IC, as other measures of IC such as the Flanker and Stroop tasks, also assess attentional processes making them less specific to IC. The main analysis tested whether deficits in IC were observed on these two tasks after total sleep deprivation or partial sleep restriction protocols, compared to a control condition comprising non-interrupted sleep.

3. Methods

The meta-analyses were performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [53,54] (see Fig. 3). The protocol for the meta-analytic investigation of the effect of sleep deprivation on IC in general population was pre-registered on May 2022 [CRD42022330418] using the International Prospective Register of Systematic Reviews (PROSPERO).

3.1. Databases and search strategy

Four databases (APAPsycINFO, Medline Complete, CINAHL Complete and Embase) were searched systematically from inception to June 13, 2022. An updated search was conducted on November 30, 2023. The search strategy was developed using the PICO (Population, Intervention, Comparison, Outcome) framework [55], the keywords related to sleep (i.e., sleep*, "sleep wake*") were paired with keywords related to IC (i.e., inhibit*, control*, impulse*, SSRT, SST, GNG). The keywords were combined by appropriate truncation and Boolean operators in the title and abstract fields to run the search. Citation searching was done in Scopus for citations and references of key articles. See Supplementary Material 1 for details of the search strategy. The titles and abstracts were independently screened by two researchers (SYC & MRY). Full-text versions of the remaining articles were screened for inclusion. There was a 99% agreement rate between the two researchers, differences were resolved by discussion and taken to a senior co-author (PKS) when required.

3.2. Inclusion criteria considerations: age and health factors

Past research has indicated age, as well as medical and/or psychiatric diagnoses may serve as confounding variables in the relationship between sleep and IC. For instance, IC exhibits age-related changes, characterised by developmental improvements during childhood and subsequent declines in late adulthood [56,57]. Furthermore, individuals diagnosed with medical and/or psychiatric disorder typically exhibit abnormalities in sleep and/or IC [58–62]. As such, considering these age-related and medical/psychiatric condition differences in sleep and IC, the current meta-analysis exclusively focuses on healthy adult samples.

3.3. Inclusion criteria

Studies were included in this review if they: 1) were published in peer-reviewed journal and in English; 2) examined the impact of sleep deprivation on IC using either the GNG or SST; 3) participants were healthy adults, 4) sleep deprivation was induced using either a total sleep deprivation or partial sleep restriction protocol, 5) and the effects of sleep deprivation on the IC task was compared to a control condition or group comprising uninterrupted sleep.

3.4. Data extraction

The following data were extracted from each study: 1) publication

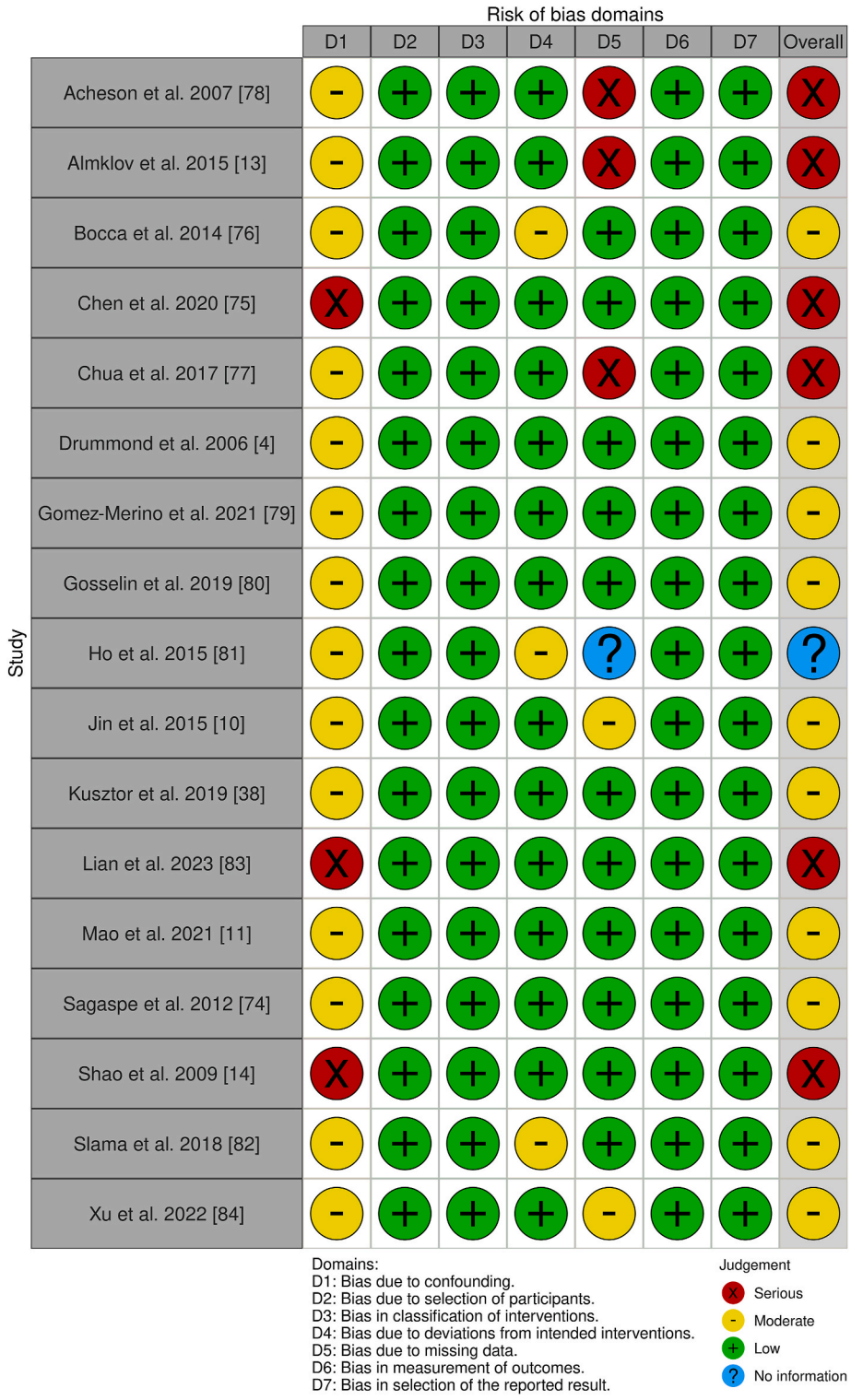

Fig. 6. Summary of the risk of bias plot of included non-randomised controlled trials.

details; 2) study design; 3) aim; 4) sample characteristics; 5) inclusion/exclusion criteria of samples; 6) type of sleep deprivation; 7) sleep measures; 8) IC measure; 9) outcome measures. Two researchers (SYC & MRY) independently extracted the data, and any differences were resolved through discussion or consultation with the senior author (PKS).

3.5. *Effect size calculations*

The results from each study were summarised using Hedge's *g* and its variance. Hedges' *g* considers the influence of small sample size on the effect size value and its variances when summarising the differences between two groups in standard deviation units [63]. This effect size was computed such that negative values indicate performance on the IC tasks was poorer following sleep deprivation, compared to the control

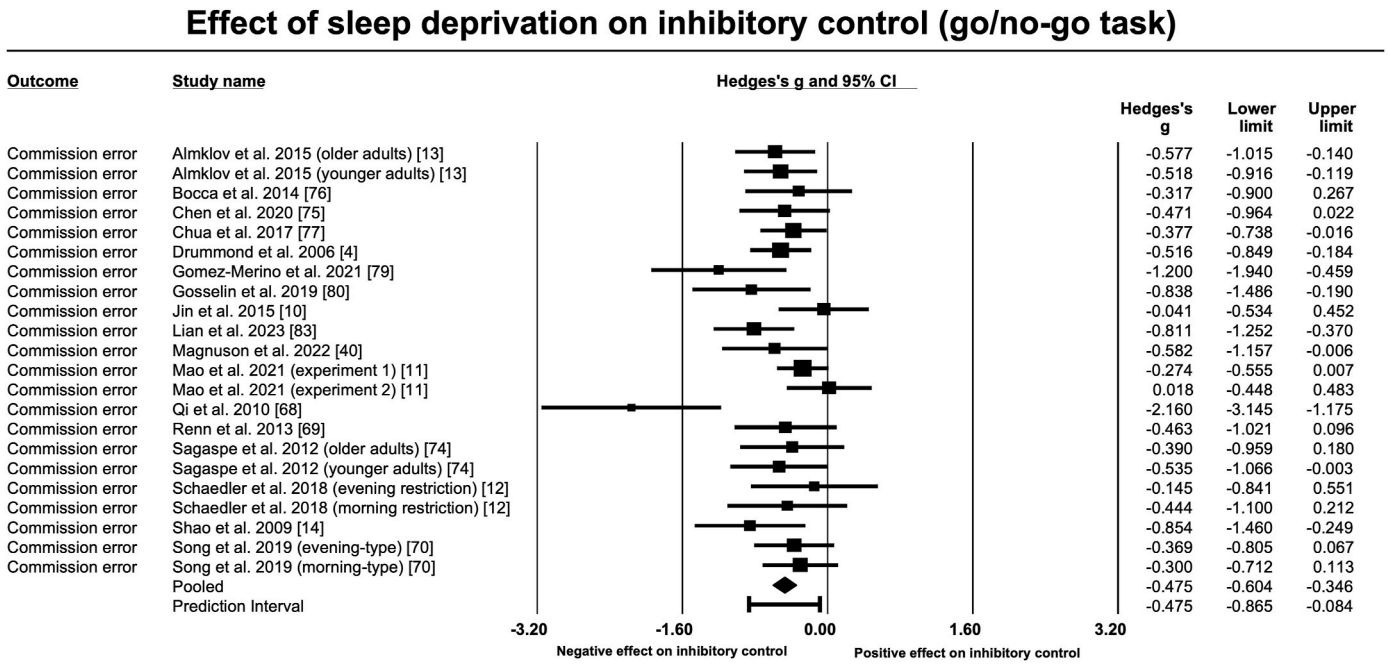

Fig. 7. Forest plot of effect sizes of the effect of sleep deprivation on inhibitory control (Go/No-Go Task).

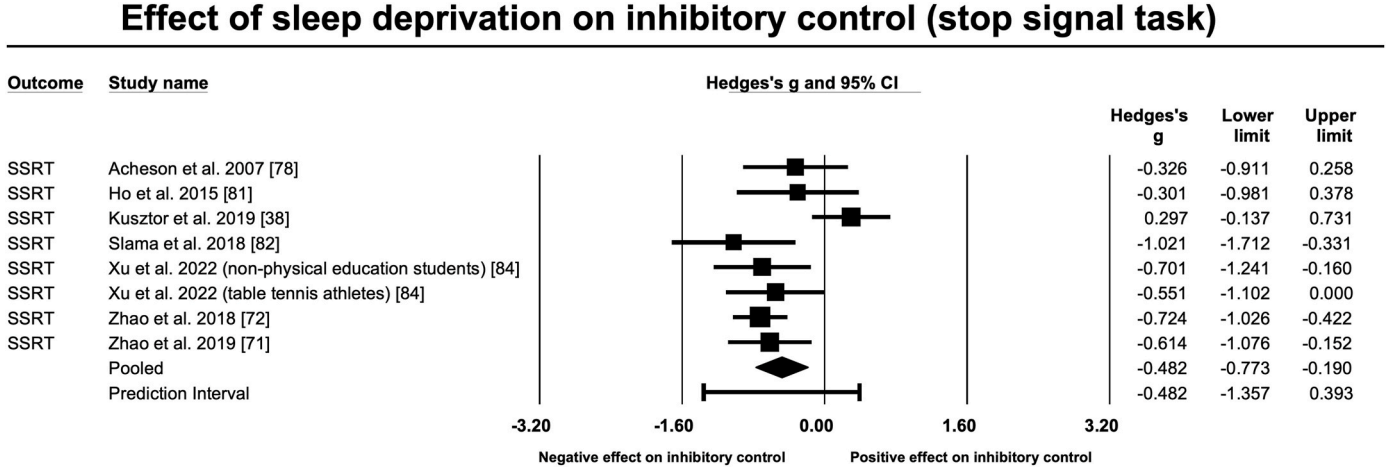

Fig. 8. Forest plot of effect sizes of the effect of sleep deprivation on inhibitory control (Stop Signal Task).

Table 1
Summary of weighted average effect sizes and publication bias assessments.

Which meta-analyses?	Total number of effect sizes (*k*)	Observed effect size (Hedge's *g*)	Eggers Test of Asymmetry (*p*-value)	Duval and Tweedie's Trim and Fill method (Hedges' *g*)
Go/No-Go Task	22	−0.475[a]	0.02	−0.475
Stop Signal Task	8	−0.482[a]	–	−0.355

[a] $p < .001$.

condition/group. Details on data extracted from each study and computed effect size is presented in Supplementary Material 2.

3.6. Meta-analytic procedures

Individual study effect sizes were averaged using random effects meta-analysis. Separate meta-analyses were undertaken to investigate the effects of sleep deprivation on IC with respect to GNG and SST. All statistical analyses were conducted using Comprehensive Meta-Analysis Version 4 [64]. For all meta-analyses, alpha was set at 0.05 (two-tailed). The I^2 statistic was used to assess heterogeneity or systematic influences between study level effect sizes. I^2 values were interpreted according to the guidelines outlined by Thompson and Higgins [65] such that values of 25%, 50% and 75% correspond to low, moderate, and high levels of heterogeneity respectively. Moderator analyses were used to investigate whether studies were from the same laboratories (yes or no) and whether differences in study designs (cross-over or parallel) accounted for variability in effect sizes. The leave-one-out analysis was conducted to examine the stability of the pooled estimates and to identify any influential samples in the main analyses.

3.7. Publication bias assessment

Publication bias was separately examined for the meta-analyses. Publication bias was assessed using funnel plot, Egger's Test [66] for

funnel plot asymmetry, and Trim and Fill analyses [67].

3.8. Risk of bias assessment

The methodological quality of the studies was also assessed. Seven of the studies [12,40,68–72] included in this report used a randomised controlled design (i.e., random allocation to groups or counterbalancing of conditions). For these studies, risk of bias was assessed using the Cochrane Collaboration risk of bias (RoB) 2.0 tool [73]. Seventeen studies included in this report used a non-randomised design (e.g., counterbalancing of conditions, lack of random allocation to groups or counterbalancing of conditions, no mentions of allocation strategy used) [4,10,11,13,14,38,74–84]. Risk of bias for these studies was assessed using the Risk of Bias in Non-randomised Studies of Intervention (ROBINS-I) tool [85]. Two researchers (SYC and MP) independently assessed the methodological quality of the included studies. Uncertainty in appraisal decisions were resolved within the research team.

4. Results

4.1. Study identification and selection

The initial search (conducted in June 2022) identified a total of 22 studies that met our inclusion criteria and were included in the meta-analysis. An updated search was conducted on November 30, 2023 with two additional studies identified, for a total of 24 studies. A summary of the study selection process is summarised in Fig. 3.

4.2. Overview of study and participant characteristics

Participant demographics and study characteristics for eligible papers are presented in Table S5 (see Supplementary Material 3). Twenty-four studies (three randomised controlled trials (RCT), fourrandomised crossover studies, 17 non-RCTs studies) met our selection criteria and were included. As some studies contributed two independent samples [11–13,70,74,84], a total of 30 samples were included in the meta-analysis. The included studies involved 712 healthy participants with their mean age ranging from 18 to 68 years.

4.3. Sleep deprivation protocols details

Twenty-three included studies (28 independent samples) conducted experimental sleep deprivation manipulation in laboratories [4,10,11,13,14,38,40,68–72,74–84], one study manipulated sleep at home (two independent samples) [12]. Habitual sleep without disruption was employed as the control condition in a laboratory setting for 19 studies [4,10,11,13,14,38,40,68,69,71,72,74,75,77–80,83,84], and in a home setting for five studies [12,70,76,81,82].

Seventeen studies controlled for sleep duration prior to conducting the experiment, five used objective measures when monitoring sleep/wake patterns (e.g., actigraphy) [12,69,74,76,77], six studies used subjective measures (e.g., sleep diaries, logs, agenda) [10,40,68,70,80,84] and six studies used a combination of objective and subjective measures [4,11,13,78,79,82]. Seven studies did not mention utilising either objective or subjective measures to validate prior sleep/wake patterns [14,38,71,72,75,81,83], Of these seven studies, two asked participants to maintain a regular sleep pattern of at least 8 h per night for one week preceding the experiment [14,75]. Two studies reported that participants generally maintained a normal sleep/wake schedule of seven to 9 h each night [71,72], one study instructed participants to maintain a regular sleep/wake schedule the day preceding experiment [38] and two studies did not report previous sleep patterns nor provide any instruction regarding sleep preceding the experiment [81,83].

There were 22 total sleep deprivation studies [4,10,11,13,14,38,40,68–72,74–78,80–84] and three partial sleep restriction studies [11,12,79]. Across the 22 total sleep deprivation protocols, participants were required to stay awake for at least 24 h (ranging from 24 h to 43 h). The three partial sleep restriction protocols had participants restrict sleep ranging from three to 6 h, with the protocol lasting from two to six days.

4.4. Risk of bias

The methodological quality of the included RCTs, randomised cross-over studies and non-RCTs are summarised in Figs. 4–6. All three included RCTs were judged to have some concerns in at least one domain [12,68,69]. The four randomised cross-over trials had some concerns in at least one domain [40,70–72]. The common sources of risk of bias in the RCTs and randomised cross-over studies were: 1) unclear randomisation process; 2) no proper handling of missing data; and 3) unclear selection of reported results. The ROBINS-I assessments of 17 included non-RCTs had moderate (n = 10) [4,10,11,38,74,76,79,80,82,84] and serious (n = 6) [13,14,75,77,78,83] risk of bias. One study [81] had no information on which to base a judgement about risk of bias. Their common sources of risk of bias were: 1) lack of consideration of potential confounders and 2) no proper handling of missing data. The inter-reviewer reliability for RoB 2.0 (RCTs and randomised cross-over studies) and ROBINS-I assessments were moderate (k = 0.602), almost perfect agreement (k = 0.829) and substantial (k = 0.745) respectively.

4.5. Quantitative synthesis of results (meta-analyses)

Results from the meta-analyses examining the effect of sleep deprivation on IC outcomes are presented in three sections. The first and second sections present the meta-analyses examining the effect of sleep deprivation protocol on the GNG and SST respectively. The third section presents results from the assessment of publication bias.

4.5.1. Effects of sleep deprivation on go/no-go task parameters

The random-effects meta-analysis contained 22 effect sizes from 17 studies (N = 501) to investigate the effects of sleep deprivation on the GNG (commission error). Fourteen studies utilised a total sleep deprivation protocol [4,10,11,13,14,40,68–70,74–77,80,83] whereas participants in three studies were subjected to a partial sleep restriction protocol [11,12,79]. The pooled effect was statistically significant and showed a medium negative effect size (Hedge's $g = -0.475$ [95 % CI: 0.604 to −0.346], Z = −7.216, $p < .001$, $I^2 = 34$ %: see Fig. 7). This indicates that on average, sleep deprivation significantly increased commission errors indicating worse IC.

4.5.2. Effects of sleep deprivation on Stop Signal Task parameters

The random-effects meta-analysis contained eight effect sizes from seven studies (N = 211) to investigate the effects of sleep deprivation on the SST. All seven studies assessed participants with a total sleep deprivation protocol [38,71,72,78,81,82,84]. The pooled effect was statistically significant and showed a medium negative effect size (Hedge's $g = -0.482$ [95 % CI: 0.773 to −0.190], Z = −3.238, $p < .001$, $I^2 = 63$ %: see Fig. 8). This indicates that on average, sleep deprivation significantly increased SSRT suggestive of deficits in IC.

4.5.3. Evaluation of publication bias

For each meta-analysis, effect sizes were averaged using a random effects model. These weighted average effect sizes are presented in Table 1 under "Observed Effect Size". For these effect sizes, significant negative values were observed for all meta-analyses, indicating sleep deprivation having a significant negative effect on IC.

Publication bias was assessed using the funnel plot, Egger's Test of Asymmetry [66], Duval and Tweedie's Trim and Fill method [67]. Aside from the funnel plots being presented in the Supplementary Material 4, results from each of these methods are presented in Table 1. Eggers Test of Asymmetry indicated that publication bias was likely for one meta-analysis (GNG), and it was not performed on the second meta-analysis (SST) as it did not have a minimum of 10 studies included

in the meta-analysis.

According to the imputed average effect size using Duval and Tweedie's trim and fill method, publication bias was unlikely for both meta-analyses. This result assumed that missing studies reported positive effect sizes (indicative of sleep deprivation having a positive effect on IC).

Leave-one-out analyses indicated limited variation in the effect sizes for both meta-analyses (for the GNG meta-analysis; Hedges' g ranged from −0.604 to −0.346, $p < .001$) and (for the SST meta-analysis; Hedges' g ranged from −0.773 to −0.190, $p < .001$). See Supplementary Material 5 for more information.

4.5.4. Moderator analyses

It is important to note that there were quite a number of studies from the same research groups [4,10–14,68,70–72,74,75,83,84] in both meta-analyses. While the authors stated that they were independent samples, several studies were conducted by the same laboratories and hence this shared context may introduce bias into the meta-analysis. To address this, two moderation analyses were performed to assess whether these factors influenced our findings. For the GNG meta-analysis, moderation by whether studies were from the same laboratories (yes or no) was non-significant (Q = 0.403, $p = .525$), indicating no significant difference between study groups from the same laboratories (Hedge's $g = -0.451$, $p < .001$) and study groups from different laboratories (Hedge's $g = -0.540$, $p < .001$). For the SST meta-analysis, moderation by whether studies were from the same laboratories (yes or no) was non-significant (Q = 1.527, $p = .217$), indicating no significant difference between study groups from the same laboratories (Hedge's $g = -0.298$, $p = .291$) and study groups from different laboratories (Hedge's $g = -0.672$, $p < .001$). See Supplementary Material 6 for the moderation analyses conducted.

Two moderation analyses were performed to assess whether there are differences in effects of sleep deprivation on inhibitory control across cross-over and parallel study designs. Results indicated that type of study design did not influence the results for either meta-analysis (for the GNG meta-analysis; Q = 0.580, $p = .446$, for the SST meta-analysis; Q = 0.303, $p = .582$). See Supplementary Material 7 for more information.

5. Discussion

A meta-analysis of the effect of sleep deprivation on IC was conducted in healthy adult samples. Twenty-four peer-reviewed papers were included, resulting in 30 effect sizes across two meta-analyses. The meta-analyses indicated an overall moderate negative effect of sleep deprivation on IC reported for both the GNG and SST. The implications of these findings are discussed below along with a discussion of limitations and potential avenues for future research.

5.1. Explanation of findings of two meta-analyses

Two meta-analyses reported significant, negative, moderate effects of the impact of sleep deprivation on IC, observed in both GNG and SST performance. Results indicated that sleep-deprived individuals exhibited significantly higher commission errors (or false alarm rates) on the GNG and longer SSRT on the SST compared to either control conditions or their own well-rested performance. Specifically, sleep deprivation protocols (total sleep deprivation or partial sleep restriction) may induce difficulties in withholding a response to a No-Go trial (i.e., action restraint) and impede on individuals' ability to stop ongoing responses upon receiving a stop signal (i.e., action cancellation). These findings suggest a relationship between sleep deprivation and impaired inhibitory control processes.

Within the meta-analyses, two studies [11,38] reported non-significant findings regarding the effects of sleep deprivation on IC. This may be due to differences in study methodologies. For instance, Mao and colleagues [11] utilised a partial sleep restriction protocol involving only 6 h of time-in-bed for two nights. It is possible that this sleep restriction protocol was insufficient to induce sleep deprivation effects within participants, as it may not have been restrictive enough or provided too few nights of partial sleep restriction. It is important to consider that some studies have demonstrated that varying task parameters, such as interstimulus intervals and ratios of go/no-go trials, may affect task performance on the GNG task [86–88]. The studies included in this meta-analysis employed a range of interstimulus intervals (from 300 ms to 2500 ms) and different go/no-go trial ratios (most used a ratio of 3:1 to 4:1, three studies used a 1:1 ratio) which could be due to functional magnetic resonance imaging or electroencephalogram protocol requirements [87]. Consequently, certain tasks may impose greater inhibitory pressures across studies. However, across all studies, well-rested and sleep-deprived participants completed the tasks under similar timing and stimulus parameters (although the exact color or shape of a stimulus may have been different). Therefore, these differences are unlikely to have significantly influenced the effect sizes reported for individual studies. Therefore, our meta-analyses indicated overall, there is a moderate negative effect of sleep deprivation on IC.

Given that sleep deprivation appears to negatively affect IC, there are some real-world implications worth noting. There has been increasing evidence linking sleep deprivation with poor driving and heightened risk of road traffic accidents [89–91], raising serious public health concerns. Additionally, there is a growing body of research indicating that individuals with poor sleep quality were more likely to engage in health risk behaviours such as increased alcohol consumption [92,93], binge-eating [94,95], and drug-use/drug-seeking tendencies [96,97] as compared to individuals with good sleep quality. Based on our current findings, we could speculate that the negative effects of sleep deprivation on IC may play a significant role in these relationships. These dynamics underscore the importance of conducting research on elucidating these complex relationships.

Our findings support the notion that sleep deprivation broadly impact cognitive task performance reliant on the PFC region [49,51]. Previous research has underscored the involvement of various brain regions, including the PFC, in executing both the GNG and SST [26–28]. While we initially hypothesised that sleep loss might affect performance differently in the two IC tasks [26–28], the current meta-analysis reveals that the effect sizes for both tasks are similar, suggesting that sleep loss impacts performance on these tasks to the same extent. It is speculated that sleep deprivation affects the PFC, leading to impaired IC performance [14,40,43,71]. However, little is known about the causal pathways within the PFC activity that leads to impairments in IC. As this is the first meta-analyses to reliably establish the behavioural effects of sleep deprivation on IC, investigating the neural and neurophysiological effects of this association, such as through functional magnetic resonance imaging and electroencephalogram studies, presents a promising avenue for future investigation.

5.2. Limitations and future directions

While the current meta-analyses highlight an overall robust finding regarding the negative effects of sleep deprivation on IC, there are several limitations of the meta-analyses and current literature to be noted. Firstly, only seven studies employed a randomised study design whereas majority of the studies (17 studies) utilised a non-randomised design within their study protocols. Failure of implementing random allocation to groups or counterbalancing of conditions could lead to larger estimates of effects of sleep deprivation on IC as compared to studies utilising a randomised study design [98]. Thus, future research should consider implementing randomised study designs to obtain more accurate effect sizes of the impact of sleep deprivation on IC.

Though our meta-analyses demonstrated a reliable impact of sleep deprivation on IC within healthy adult samples, it remains unclear whether this relationship holds true in clinical populations. Factors commonly present in clinical populations, such as lower quality of life,

7. Practice points

1. Sleep deprivation has a robust, negative impact on one's ability to suppress automatic responding as measured by two widely used inhibitory control tasks (Go/No-Go and Stop Signal Task).
2. Given that sleep deprivation can impair the ability to suppress impulsive behaviours, it may lead to a greater risk of traffic accidents and behaviours such as excessive drinking, binge-eating, and drug use.
3. Considering that sleep is a modifiable factor, improving sleep may result in better inhibitory control performance.

8. Research agenda

1. It is suggested that sleep deprivation negatively affects inhibitory control performance via the prefrontal cortex. Yet, these causal pathways within the prefrontal cortex remain understudied, future studies should investigate the underlying neural and neurophysiological effects (e.g., functional magnetic resonance imaging, electroencephalography).
2. While our meta-analyses demonstrated sleep deprivation reliably impacts inhibitory control in healthy adults, its effect on clinical populations remains unclear. Research is needed to examine whether this relationship holds true within clinical populations (e.g., attention deficit hyperactivity disorder, alcohol use disorder, insomnia).
3. More sleep deprivation studies that include the Stop Signal Task are needed given it measures a critical component of inhibitory control and will enable a more comprehensive assessment of the mechanisms underlying the relationship between sleep deprivation and impaired inhibitory control.
4. There are currently more studies in the literature utilising total sleep deprivation protocols compared to partial sleep restriction protocols. The predominant use of the former represents a significant issue due to its lack of ecological validity when compared to partial sleep restriction protocols. More studies should utilise partial sleep restriction protocols, as they offer a more ecologically valid approach to understanding naturalistic sleep disturbances in the real-world settings.
5. Future research should examine how sleep, circadian rhythms and inhibitory control interact and consider individuals' sleep-wake timing and time-of-day variations in studying the effects of sleep deprivation on inhibitory control.

overall poorer sleep quality, and greater physical and mental health burden may intensify the link between sleep deprivation on IC [99,100]. It would be important for future work to further examine this relationship within clinical populations known to exhibit abnormalities in sleep and/or IC [58–62].

Furthermore, the majority of included studies utilised the GNG (17 studies) compared to the SST (seven studies). Given the disproportionate focus on the former task in the current literature, this may indicate a lack of comprehensive understanding regarding the relationship between sleep deprivation and IC in relation to action cancellation. While both tasks showed similar effect sizes here, future research should nonetheless consider employing the SST alongside the GNG to provide a comprehensive understanding of the impact of sleep deprivation on IC.

Lastly, given the important relationship between sleep and circadian rhythms processes [101,102], simultaneous investigation of sleep, circadian rhythms, and IC is imperative for future studies. Eighteen out of 24 studies [4,10–13,38,40,68–72,74,76–79,81] highlighted the significance of controlling for individuals' preferred sleep-wake timing (i.e., a proxy for circadian phase, [103]) in evaluating individuals' IC performance. Specifically, these studies controlled for participants' sleep-wake schedule, ensured testing occurred at the same time-of-day for both experimental conditions and when IC performance was not at its lowest [104]. As some studies did not mention or did not control for usual sleep-wake times or time-of-day [14,75,80,82–84], this may confound the observed effects of sleep deprivation on IC [105,106]. Therefore, in addition to sleep deprivation effects, it is plausible that sleep-wake timing and time-of-day also influence IC performance. Future research should explore how sleep, circadian rhythms, and IC interact, and consider individuals' sleep-wake timing and time-of-day variations in studying the effects of sleep deprivation.

6. Conclusion

The current meta-analyses revealed a robust, significant negative effect of sleep deprivation on IC in healthy adult samples. Specifically, sleep deprivation impairs individuals' ability to withhold prepotent responses (i.e., GNG) and cancel ongoing responses when prompted to stop (i.e., SST). Given the fundamental role of IC in regulating everyday behaviour and the observed behavioural effects of sleep deprivation on IC, it is crucial to consider future directions. This includes delving into the neural and neurophysiological effects of this association and examining how sleep deprivation may impact IC in clinical populations (e.g., insomnia, attention-deficit hyperactivity disorder, and alcohol use disorder). Finally, a deeper exploration of the interplay between sleep, circadian rhythms and IC is warranted to gain insight into how these factors collectively influence IC processes. Importantly, the meta-analyses contribute to existing literature by establishing a moderate behavioural effect of sleep deprivation on the two theoretical aspects of IC: action restraint and action cancellation. These findings have crucial real-world implications for areas such as work productivity and motor-vehicular safety [107,108], underscoring the importance for further investigation into this association, not only for individual well-being but also for broader societal concerns.

Conflicts of interest

The authors do not have any conflicts of interest to report.

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Appendix A. Supplementary data

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