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Sleep, stress and aggression: Meta-analyses investigating associations and causality

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ABSTRACT

Prior research suggests that sleep is associated with increased subjective stress and aggression, but important questions remain about the typical magnitude of these relationships, as well as their potential moderators. We therefore conducted the first meta-analysis of this literature. Across 340 associational and experimental studies, significant associations were identified between sleep with both subjective stress ($r = 0.307$, $p < .001$) and aggression ($r = 0.258$, $p < .001$) in individuals from the general population, as well as between sleep with subjective stress ($r = 0.425$, $p < .001$) in individuals with sleep disorders. Experimental sleep restriction also led to increased subjective stress ($g = 0.403$, $p = .017$) and aggression ($g = 0.330$, $p = .042$). These findings suggest that poorer sleep is associated with - and leads to - heightened levels of subjective stress and aggression. These findings, and their implications, are discussed in relation to neurobiological literature, which highlights the complex interplay between metabolic activity in the brain, hormonal changes, and behavior.

1. Introduction

Sleep is a fundamental biological process that is critical for a range of key brain functions including cognition and mental health (Lo et al., 2015; Lovato and Gradisar, 2014; Pires et al., 2016). It is therefore of concern that, in recent years, the average nightly sleep duration per individual has decreased, with sleep disorders such as insomnia continuing to increase in prevalence (Adams et al., 2016; Ford et al., 2015; Knutson et al., 2010; Kronholm et al., 2008). This is largely due to both population aging (as sleep disturbances are more common in older age) and engagement in lifestyle behaviors that negatively impact sleep quality (such as use of internet devices and consumption of energy drinks; Adams et al., 2016). Sleep disturbances are also a common feature of many neurological and psychiatric disorders (Adams et al., 2016; Petit et al., 2004; Spiegelhalder et al., 2013; Videnovic et al., 2014), and chronic sleep disturbances or disorders have been linked to poorer health outcomes such as diabetes and cardiovascular disease (see Sigurdson and Ayas, 2007 for a review).

The present meta-analysis was designed to investigate the relationship between sleep with both subjective stress and aggression. Although these are distinct constructs, both reflect important behavioral outcomes that have been investigated frequently in relation to sleep disturbances, and can negatively affect health and psychosocial wellbeing (Bosch et al., 2013; DeLongis et al., 1988; Jennings et al., 2017; Meyrueix et al., 2015; Paterson et al., 2011; Wu et al., 2015; Quick et al., 1987). For instance, stress can have detrimental effects on everyday cognitive functioning and mood (Bosch et al., 2013; DeLongis et al., 1988; Quick et al., 1987). Similarly, aggression is associated with generalized anxiety and depression symptoms (Meyrueix et al., 2015; Paterson et al., 2011; Wu et al., 2015), as well as increased cardiovascular risk and mortality (see Jennings et al., 2017). This study will therefore investigate the relationship between sleep with both subjective stress and aggression.

Whilst most studies have found that poorer sleep was associated with increased subjective stress and aggression, a few discrepancies have been noted, with some studies finding no significant association for either relationship (Grano et al., 2008; Hisler and Krizan, 2017; Kramer et al., 2019; Pilcher et al., 1997; Veeramachaneni et al., 2019). However, what is not clear from this literature is the typical magnitudes of these effects, which is important to understand the potential practical importance of these relationships. Here, we discuss the neurobiological, behavioral, and experimental literatures that speak to these relationships. We then report the first meta-analysis of the association – and

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causal impact – of sleep with both subjective stress and aggression. Because individuals with diagnosed sleep disorders and individuals from the general population experience quite distinct levels, and possibly types, of sleep disruptions over time which may in turn differentially impact sleep and aggression, these questions will be considered separately for these two populations. Another key focus of this meta-analysis is the potential influence of moderators on the proposed relationships, as this has implications for our understanding of when and why these effects might be expected to emerge in everyday life.

This paper makes a distinction between 'individuals with sleep disorders' and 'individuals from the general population'. The former refers to studies where samples were purposefully selected to include individuals with a diagnosed sleep disorder (e.g., individuals diagnosed with Insomnia). The term 'individuals from the general population' refers to studies where samples were not selected based on the diagnosis of a sleep disorder (e.g., national samples, university students, etc.).

1.1. Sleep and stress

Stress is often separated into three broad components, each with their own range of nuances - stressors, physiological stress, and psychological stress (Kemeny, 2003). Lazarus (1999) proposed that the stress process is initiated by an individual's appraisal of an event or time period as an imposition. As such, the focus of this meta-analysis will be on the psychological stress response, or one's subjective stress. Although stress can help with the detection of threat or motivate us to expend effort, perceived stress can also have a negative impact on everyday cognitive functioning, general health, and mood (Boals and Banks, 2012; DeLongis et al., 1988; Quick et al., 1987). Therefore, it is important to investigate whether, and if so how, potentially modifiable lifestyle factors, such as sleep, can influence one's perceived stress.

1.1.1. Neurobiological literature

Given that the neuroanatomical regions affected by sleep deprivation overlap with those that underpin stress responses, neurobiological models of sleep suggest that more or higher quality sleep should be associated with lower stress. For instance, a recent meta-analysis found that sleep deprivation led to reduced activity in the intraparietal sulcus and superior parietal lobule, and further impacted regions connected to the superior parietal lobule such as the insula, inferior frontal gyrus, and the superior parietal cortex – all key neural regions activated in the psychological and physiological stress response (Javaheripour et al., 2019). The neuroendocrine literature also provides evidence that cortisol, commonly referred to as 'the stress hormone', increases following sleep deprivation, suggesting that lower levels of sleep should be associated with greater stress. Cortisol follows a strong diurnal rhythm, whereby levels are naturally elevated in the morning and gradually decrease throughout the day until night-time (Stone et al., 2001). Importantly, both responding to stressors and undergoing sleep deprivation are known to increase cortisol levels (Goodman et al., 2017; Leproult and Van Cauter, 2010; Nejtek, 2002; Redwine et al., 2000; Wright et al., 2015). Indeed, sleep deprivation can itself be considered a stressor since a full sleep appears to suppress certain neuroendocrine stress systems, whereas sleep deprivation tends to maintain activity in these stress systems (see Meerlo et al., 2008 for a review). Taken together then, sleep may directly impact stress levels by impacting the key neuroanatomical and neuroendocrine pathways involved in the stress response.

1.1.2. Behavioral literature

There is a clear neurobiological basis for predicting that an association between sleep and subjective stress should exist in individuals from the general population. Although a few studies have failed to identify any association, (Hisler and Krizan, 2017; Kramer et al., 2019; Veeramachaneni et al., 2019), most have identified a significant inverse association, whereby poorer sleep is associated with higher levels of subjective stress (e.g., Åkerstedt et al., 2015; Alfasi and Soffer-Dudek, 2018; Bani-Issa et al., 2020; Barber et al., 2010; Bidulescu et al., 2010; Cardoso et al., 2021; Cuadros et al., 2012; Doolin et al., 2018; Hall et al., 2007). A similar pattern has emerged in the literature focused on individuals with sleep disorders, with most studies finding poorer quality sleep is linked to greater stress (Basishvili et al., 2012; Chester and Dzierzewski, 2020; Giese et al., 2013; Lothian et al., 2016; Puzino et al., 2020; Vand et al., 2021; Veeramachaneni et al., 2019), and a smaller number finding no evidence of any association (Hall et al., 2000, 2007). However, what both of these literatures fail to clearly show is the typical magnitude of these effects, with the absolute magnitude of the associations reported across individual studies varying considerably. A key aim of this meta-analysis was therefore to establish, not only whether the predicted aggregate negative associations between sleep and subjective stress in both individuals from the general population and individuals with sleep disorders are statistically significant, but their overall magnitude (i.e., whether the typical effects in these literatures are small, moderate or large).

1.1.3. Experimental literature

Importantly, the studies referred to so far have been observational, whereby sleep and subjective stress were measured, and the association between the two simply assessed. However, a stronger approach - that allows for causal inferences to be made - is to experimentally manipulate sleep duration and then assess the impact of this manipulation on a later stress response. Several studies to date have now used experimental methods to investigate the causal role of sleep deprivation on subjective stress levels in individuals from the general population. Here again, although a recent study identified no difference in ratings of subjective stress between sleep restricted and rested conditions (Abbott et al., 2020), most studies have found evidence consistent with a causal pathway, whereby individuals experimentally assigned to a sleep restriction condition demonstrated higher levels of subjective stress than those assigned to a full sleep condition (Franzen et al., 2011; Larsen et al., 2020; Minkel et al., 2012; Schwarz et al., 2018). However, because this prior literature fails to provide clear evidence about the typical magnitude of this effect, with reported effect sizes varying considerably across individual studies, this study will also use meta-analytic methodology, not only to test whether the aggregate impact of sleep deprivation on subjective stress is statistically significant, but the absolute magnitude of this aggregate effect.

1.2. Sleep and aggression

Aggression refers to a spectrum of behaviours that intend to harm another individual who is motivated to avoid being harmed (see (Bettencourt et al., 2006). The term encompasses four main components. *Anger* refers to the affective/emotional dimension; *hostility* refers to the cognitive dimension; and *verbal* and *physical* aggression refer to the two distinct behavioural dimensions (Bettencourt et al., 2006; Buss and Perry, 1992; García-León et al., 2002). Given the considerable harms caused by aggression in modern society, from road rage to domestic violence, to bullying in workplaces and schools, it is not only theoretically but also practically important to understand whether modifiable lifestyle factors – such as sleep – influence aggressive behaviours.

1.2.1. Neurobiological literature

The neural basis of aggression has been characterized as a coupling between the ventromedial prefrontal cortex and the amygdala (Rosell and Siever, 2015). Previous research has demonstrated that, as with stress, neural areas affected by sleep deprivation overlap with those that underpin aggression. For instance, sleep deprivation reduces the cerebral metabolic rate for glucose in several cortical and subcortical structures, including the prefrontal and posterior parietal cortices – key neural regions known to moderate aggressive behavior (Bosch et al., 2013; Shao et al., 2014; Thomas et al., 2000; Yoo et al., 2007). However,

the neurotransmitters and hormones that underpin aggression are influenced by many different variables, making their impact on aggression more complex and indirect (Abu-Samak et al., 2018; Maggio et al., 2013; Oh et al., 2012). Specifically, sleep deprivation has been shown to decrease testosterone levels, and increase levels of cortisol (Leproult and Van Cauter, 2010; Redwine et al., 2000; Wright et al., 2015) and serotonin (Elmenhorst et al., 2012; Lopez-Rodriguez et al., 2003; Senthilvelan et al., 2006). However, cortisol, testosterone, and serotonin appear to have relationships with aggression that are moderated by factors such as the type of aggression being assessed, sex, psychopathy, and other pathological personality factors (Denson et al., 2012; Goetz et al., 2014; Krämer et al., 2011; Kuepper et al., 2010; Passamonti et al., 2012; Popma et al., 2007; Rubia et al., 2005; van Wingen et al., 2010; see Rosell and Siever, 2015 for a review). Therefore, it is possible that sleep impacts aggression via the neuroanatomical and neuroendocrine pathways involved in modulating aggression.

1.2.2. Behavioral literature

Consistent with predictions from neurobiological literature, although some studies have failed to identify any association between sleep and aggression (Grano et al., 2008; Hisler and Krizan, 2017; Pilcher et al., 1997), most have identified an inverse association, whereby poorer sleep is associated with increased aggression (Deguchi et al., 2017; Farnill and Robertson, 1990; Kirwan et al., 2019; Lund et al., 2010; Tsuchiyama et al., 2013). However, again, because the size of these effects has varied considerably across individual studies, of interest here is not only whether the aggregate effect is significant, but also its typical magnitude. Because all studies completed to date focused on this question have sampled individuals from the general population (i.e., there has been no consideration of the association between sleep and aggression in individuals with sleep disorders), these analyses were only able to consider individuals from the general population.

1.2.3. Experimental literature

Similar to the corresponding behavioural literature, while some studies have failed to identify any effect of sleep manipulation on aggression (Haack and Mullington, 2005; MacDonald et al., 2019; Scott and Judge, 2006; Vohs et al., 2011), most have shown that sleep deprivation or restriction results in higher levels of aggression compared to individuals who experienced regular sleep (Hart et al., 1987; Krizan and Hisler, 2019; Larsen et al., 2020; Paterson et al., 2011; Taub, 1977). Others have revealed more nuanced relationships, whereby variables such as gender or type of aggression indexed moderated the relationship between sleep and aggression (Cote et al., 2013; Minkel et al., 2012). These inconsistencies again speak to the importance of using meta-analytic methodology to provide a direct test of the presence and magnitude of the effect of sleep deprivation on aggression, as well as potential moderators of these effects.

1.3. The present study

Despite more than three decades of research on this topic and many hundreds of research studies, current understanding of the associations between sleep with subjective stress and aggression is limited because of the mixed findings reported across individual studies, as well as large variation in the magnitude of any identified relationships. The present study was therefore designed to provide the first statistical integration of prior literature that assessed these relationships. In line with neurobiological models, we predicted that poorer sleep would be associated with higher levels of subjective stress in individuals from the general population and individuals with sleep disorders. We also predicted that poorer sleep would be associated with higher levels of aggression in individuals from the general population. Finally, we predicted that manipulated sleep restriction would significantly increase levels of both subjective stress and aggression in individuals from the general population.

1.4. Key moderators

Although many variables have been proposed that may influence the relationship between sleep with subjective stress and/or aggression, in this meta-analysis we focused on three potential moderators: age, body mass index (BMI), and participant type (for information regarding this latter variable, please see the Supplementary Document). These variables were selected, as not only are there strong theoretical grounds for predicting that each of these variables might influence how sleep affects subjective stress and aggression, but these are also characteristics routinely reported in contributing studies, in the form of recruitment or demographic information.

With respect to chronological age, a recent meta-analysis of age and sleep as indexed via actigraphy found that with increasing age, sleep duration was shorter and sleep efficiency reduced (Bowman et al., 2020). A separate meta-analysis investigating the age and stress relationship found that as age increases, so too does cognitive irritation – in this study, defined as a component of work stress. Interestingly, the results of this latter meta-analysis also found that the relationship between age and specific aspects of work stress varied depending on the occupation (Rauschenbach et al., 2013). A recent review further suggests that the physiological process underpinning stress may change as a function of normal adult ageing, potentially predisposing older adults to more negative health outcomes (Gaffey et al., 2016). Furthermore, a recent longitudinal study demonstrated increased reactivity to daily stress with increased age (Sliwinski et al., 2009).

While the literature in relation to aggression also suggests a relationship with age, this is in the reverse direction to that seen with stress. Compared to younger adults, older adults report and demonstrate lower levels of anger expression (Phillips et al., 2006; Kunzmann and Thomas, 2014). Older age has also been linked to decreased hostility (Silton et al., 2013), less aggression in specific contexts such as sport spectatorship (Icekson et al., 2021), and lower aggression in general (Vigil-Colet et al., 2015). Given these findings, we intended to explore if age accounted for significant variance in the causal and correlational relationships between sleep with stress and aggression.

Obesity also has strong links with stress (Tomiyama, 2019; Vgontzas et al., 2014, 2008; Yang et al., 2014). Stress can induce overeating and can trigger physiological changes in pathways that control the rewards system in the brain (see Tomiyama, 2019 for a review). A recent meta-analysis found that being overweight or obese was associated with poorer sleep quality in young adults (Fatima et al., 2016), and a separate meta-analysis found obesity was associated with higher rates of insomnia (Chan et al., 2018). For this reason, BMI (a crude but measurable index of individual's weight-related health) is considered a potential moderator of the relationships between sleep with stress.

2. Method

2.1. Transparency and openness

The protocol and analytic plan for this meta-analysis was preregistered with the Open Science Framework (https: //osf.io/35rmy/?view_only=fd17f195c68e47f4b0d1d69ff45567c1). The current meta-analysis was conducted and reported in accordance with PRISMA guidelines (Moher et al., 2009). For a flow chart depicting the process of study selection for this meta-analysis, see Fig. 1. A copy of the raw meta-analytic data that was extracted is available from the same open science framework link.

2.2. Eligibility criteria

Studies were considered eligible for inclusion in the meta-analysis if they had the following characteristics: (1) included a measure of sleep assessed either objectively or via self-report, (2) included a measure of subjective stress or aggression, (3) participants were a non-clinical

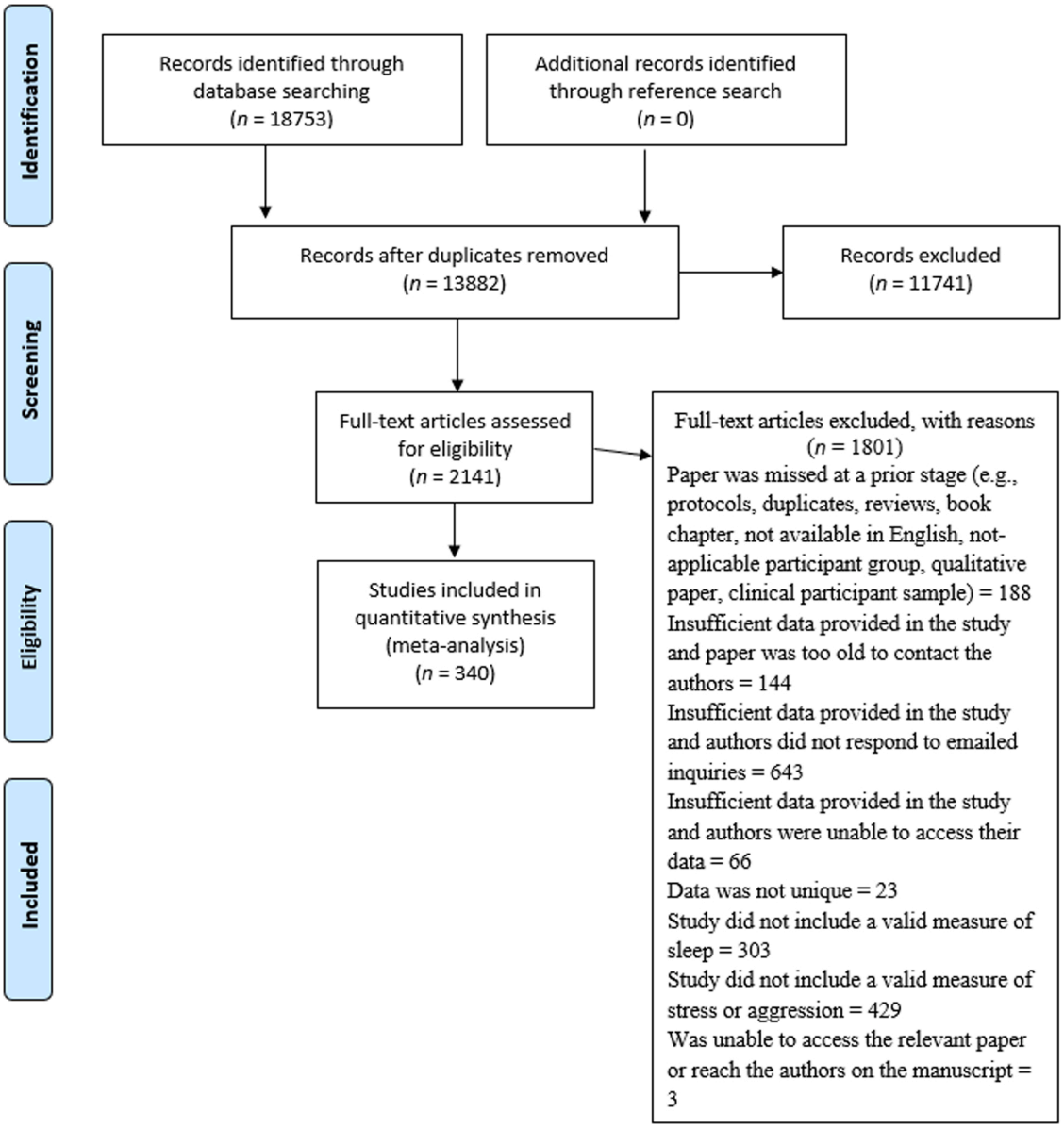

Fig. 1. PRISMA flowchart displaying study screening process.

sample (i.e., no diagnosis of psychiatric or neurological disorders; participants diagnosed with sleep disorders were included), (4) the required statistics to derive effect sizes were published or provided upon request, (5) the studies and their data were unique, and (6) the study was published in a peer-reviewed journal in English. There was no restriction placed on the year in which a study had to be published. In instances where multiple studies used the same participant data, the study with the most relevant measures of sleep, subjective stress or aggression, or largest sample were used.

2.2.1. Sleep eligibility criteria

Sleep is a broad construct and as such can be assessed in a multitude of ways. For instance, sleep measured via polysomnography can be differentiated into which stage of sleep is being assessed, wake after sleep onset, sleep duration, number of awakenings, and so on. Similarly, subjective measures of sleep can assess perceived duration, quality, fragmentation, and numerous other aspects. As the goal of this meta-analysis was to assess the relationship between subjective stress and aggression with overarching sleep, we allowed the inclusion of objective or subjective estimates of sleep.

2.2.2. Stress eligibility criteria

Eligible studies had to include a self-report measure of subjective stress over the past day, week, month, or three months (e.g., The Perceived Stress Scale). As the primary focus of this meta-analysis was subjective stress, physiological indicators (i.e., heart rate, blood pressure, perspiration) were not included. Measures of broader psychological distress (such as the Kessler 6 Psychological Distress Scale) were excluded as these assess a range of negative affective states that include, but are not restricted to, subjective stress (Barkham et al., 2013; Winefield et al., 2012).

2.2.3. Aggression eligibility criteria

Eligible studies had to include a self-report measure of aggression over the past day, week, month, or three months (e.g., the Buss-Perry Aggression Questionnaire). Again, as the primary focus of this meta-analysis was subjective or behavioral aggression, physiological indicators (i.e., heart rate, blood pressure) were not included. Furthermore, physiological measures of aggression were excluded due to ambiguity in determining specific mechanisms underpinning aggression.

2.3. Study selection

A systematic literature search was conducted across PsycINFO, Web of Science, and PubMed databases in January 2021. For each database search, the fields searched were title and abstract. The search terms used were *insomnia* , polysomnogr* , sleep* , actigraph** ; in conjunction with *aggress* , anger, angr* , hostil* , and stress** . This search yielded 19,673 results. To ensure search results were relevant to the aims of the current paper, the following terms were excluded: *autis* , ASD, asperg* , animal* , infan* , child* , oxidative, PTSD, post-traumatic* , post traumatic, posttraumatic, mice, mouse, rat, rats, monk, neonat* , drosophila, fish, psychosis, antisocial personal disorder, psychiatric, inflame, cat, cats, bat, bats, dog, dogs, bird, birds*. Any papers with *review* or *meta* in the title were also excluded. Common animals used in testing (listed in above search terms) were excluded from the search as Web of Science does not provide a function to exclude studies based on population. The revised search using the exclusion criteria yielded 18,753 results. The search terms used were drawn from previous systematic reviews or meta-analyses answering similar questions (Baglioni et al., 2013; Dewald et al., 2010; Duke et al., 2013; Kogler et al., 2015). A backward citation search of studies identified in relevant meta-analyses and systematic reviews was then conducted to ensure all papers relevant to the research question were included (Chen et al., 2020; Polmann et al., 2021; Wang and Bíró, 2021).

Following the exclusion of duplicates, the first author screened 13,903 titles and abstracts to remove ineligible studies (e.g., studies with no mention of sleep, subjective stress, or aggression, systematic reviews or meta-analyses, or animal studies. See Fig. 1). To reduce the possibility of an eligible text being excluded by mistake, a second independent screening of the titles and abstracts was conducted by the third, fourth, and fifth, authors who each screened 4056, 5411, and 4438 titles and abstracts respectively. When conflicts arose regarding the inclusion of a study, the two authors who screened the title and abstract discussed their reasons for including or excluding said study. In instances where the decision to include or exclude a study could not be made, the second author made an independent assessment and final decision; this occurred for 12 titles and abstracts. Once title and abstract screening was complete, the first author screened a total of 2141 full-text papers. In instances where study inclusion was ambiguous, the third author was consulted; this occurred for 25 papers.

2.4. Data extraction

The data extracted from the final sample of eligible primary studies included:

1. Data to derive effect sizes for correlational studies (e.g., sample size and correlation coefficients for the relationship between sleep and subjective stress or aggression);
2. Data to derive effect sizes for experimental studies (e.g., the mean and standard deviations to compare sleep deprived and rested participants on measures of subjective stress or aggression);
3. Key participant details/covariates: age (mean and standard deviation), BMI (mean and standard deviation), and sample nationality;
4. How sleep, stress, and aggression were measured (i.e., objective measurement, self-report) and, for aggression, it was noted whether trait or state aggression was measured.

When required data were unavailable, the corresponding author on the manuscript was contacted. The exception being that authors were not contacted if their study was published more than 17 years ago (2004 or earlier), in which case, the data was assumed to be unavailable. In total, 885 authors were contacted, with some emails inquiring after data from more than one paper. Of these, 172 authors responded to the emails and provided the requested data, 66 authors responded to the emails but were unable to access their data, and three authors responded to the request but refused to provide data. The remaining authors did not respond to the request. For a list of references from authors who responded to the request for data please see the Supplementary Document.

For studies that included multiple measures of sleep (i.e., sleep duration, sleep quality, number of awakenings), measures of sleep duration assessed through polysomnography were preferentially used, followed by actigraphy assessed sleep duration, sleep assessed via the Pittsburgh Sleep Quality Index (PSQI), self-reported sleep quality, then self-reported sleep duration. In instances where measures of sleep quality or duration were split across weekend or weekday data, weekday data was used as this allowed for a longer sleep assessment. For studies containing stress measured with both the Perceived Stress Scale (PSS) and the Depression, Anxiety, and Stress Scale (DASS), the PSS was used as this measure solely assesses subjective stress. In the aggression literature, some studies that used the State-Trait Anger Expression Inventory-2 (STAIX-2) reported data from each subscale rather than a global score. When a global score from the full STAIX-2 was not available, we used the global score for the State Anger subscale. When this was also not available, the overall score from the Angry Temperament subscale was used as this reflects the tendency to become angered quickly and easily.

As the relationships between sleep with subjective stress and sleep with aggression were not compared statistically, independence of data was not required. Therefore, studies with eligible data on the relationship between sleep and subjective stress, and sleep and aggression were permitted to contribute to both sets of analyses. In instances where studies reported relevant data on two independent samples (e.g., if a study separated participants by younger and older adults), both applicable data points were used under the assumption these represented independent samples.

To ensure reliability of data extraction coding, a random sample of 20% of the included studies were independently double coded. Due to the number of contributing studies, independent data extraction and coding were performed by the third and fourth authors. For coded categorical variables, we calculated the Cohen's Kappa (κ) coefficient of agreement, which accounts for agreement by chance (Cohen, 1960). We used the suggested interpretations of κ, that κ's of 0.60–0.79 are moderate, 0.80–0.90 are strong, and 0.91–1.00 are almost perfect (McHugh, 2012). For all key coded variables, the percentage agreements were calculated. Because specific benchmarks for their interpretation have not been established (Orwin and Vevea, 2009), we set our own a priori benchmarks of what were considered good agreement as follows: Percentage agreements of 80 or above were considered high, percentage agreement between 60 and 79 were considered moderate, and percentage agreement below 60 were considered low. Disagreements were assessed, and the first author's final coding decision was used for analyses.

The Kappa statistic was calculated using SPSS version 27. In instances where one of the two coders left a numerical or nominal data section blank (e.g., if coder one reported age as 29.39 and coder two left this blank; or if coder one reported the sleep measure as the PSQI and coder two left this blank), the missing numerical value would be recorded as *999* and the missing nominal variable as *MISS*. This ensured the analyses recognised any inconsistencies in coding. The variables that were assessed included the sleep measure used, the stress/aggression measure used, the required effect size (correlation, or mean and standard deviation), sample size, participant type, nationality, age (mean and standard deviation), and BMI (mean and standard deviation).

2.5. Statistical analyses

Meta-analyses were conducted using the Comprehensive Meta-Analysis Version 3 software (Borenstein et al., 2014). Cochran's *Q* was used to measure the extent to which the studies contributing to each aggregate effect size could be regarded as homogenous. For

correlational data, the correlation coefficient *r* effect size was used. For experimental data, a Hedges *g* index of effect size was used (Borenstein et al., 2014). Cohen's conventions were used for size interpretation. Accordingly, correlational effect sizes were deemed small, medium, or large when their values were equal to or larger than 0.1, 0.3, and 0.5 respectively. Hedges *g* effect sizes were deemed small, medium, and large when their values were equal to or larger than 0.2, 0.5, or 0.8 respectively (Cohen, 1988). The effect sizes for each study were coded such that positive values were indicative of the hypothesised relationship (e.g., poorer sleep quality was associated with higher subjective stress or aggression). The random effects model was used as it better accommodates heterogeneous effect size distributions (Lipsey and Wilson, 2001). To run a meta-regression, a minimum of 10 contributing data points per moderator variable is recommended (Higgins et al., 2019). Therefore, a meta-regression assessing variance accounted for by age and BMI was run for the associative relationship between sleep and stress in individuals from the general population, however only age could be assessed in the associative relationship between sleep and aggression. No other relationship investigated had enough data to investigate the effect of these moderator variables.

2.5.1. Study quality assessment

To investigate any potential biases at the level of the individual study, the Quality Assessment with Diverse Studies (QuADS) tool was used (Harrison et al., 2021). This scoring system was chosen over the originally pre-registered option as it provided quality scoring options for multiple study designs. Due to the high number of contributing studies, the first, fourth, and fifth authors each independently scored approximately a third of the contributing papers. The QuADS contains 13 items each rated from 0 to 3. Scores from the QuADS therefore ranged from 0 to 39 with higher scores indicating a higher quality study. The QuADS does not have an inclusion or exclusion cut-off score for studies, instead researchers are advised to discuss the quality assessment findings and use discretion to determine study inclusion.

2.5.2. Publication bias assessment

"The file drawer problem" refers to the higher probability of significant results being published relative to nonsignificant results (Easterbrook et al., 1991; Rosenthal, 1979). We conducted two analyses to test whether the results were likely to have been influenced by publication bias. For each overall effect size (e.g., the relationship between sleep and stress for healthy adults, etc.) the Begg and Mazumdar rank correlation test was used to calculate the correlation between effect size and study size. Egger's test of the intercept was used to provide a linear regression of the effect estimates on their standard errors weighted by their inverse variance. For these, publication bias was indicated by a significant correlation/intercept. To further assess for publication bias, a funnel plot analysis of all contributing studies was conducted. We plotted the effect sizes against their standard errors with standard error decreasing along the y-axis and effect size increasing on the x-axis. An unbiased literature of a true effect was represented by a symmetrical distribution of effect sizes on this graph. A biased literature, where studies were more likely to be published when a significant positive effect is found, was represented by an asymmetrical distribution in a left-skewed pattern thus indicating that the studies we would expect to see due to measurement error were missing, possibly due to publication bias.

2.6. Post hoc assessment of measurement type

It is likely that sleep may relate differently to subjective stress, depending on how it was measured. For instance, many common assessments of sleep such as the PSQI, or single items assessing duration or quality, rely on an individual's memory and their ability to accurately report their experience. On the other hand, objective indicators such as polysomnography can directly measure time spent in each of the sleep phases, as well as report specifics of sleep architecture, and therefore removes potential errors that may occur from memory. It is well established that subjective reports of sleep are often quite different to the objective reality (Landry et al., 2015). Furthermore, the PSQI (the current gold standard in subjective sleep measures) does not correlate well with polysomnography (Buysse et al., 1991, 2008; Mondal et al., 2013). Therefore, we conducted a post hoc meta-regression to test whether sleep measurement type moderated the sleep and subjective stress relationship in individuals from the general population. It should be noted that this assessment of measurement type could not be run on the other key relationship (i.e., between sleep and aggression) due to insufficient data points (i.e., there were < 10 contributing unique effects for some categories). This test was conducted using R metafor version 4.2.0. Sleep measures were categorized into the following groups: (1) objective assessments such as actigraphy or polysomnography data; (2) subjective assessments measured via the PSQI – as this is currently the gold standard for subjective sleep measures; (3) other multi-item sleep questionnaires such as the Insomnia Severity Index; (4) single items assessing sleep duration; and (5) single items assessing sleep quality.

3. Results

3.1. Literature search

In total, 340 studies met the inclusion criteria. This included 278 studies assessing the association between sleep and subjective stress in individuals from the general population, nine assessing the association between sleep and subjective stress in individuals with sleep disorders, 50 assessing the association between sleep and aggression in individuals from the general population, six assessing the causal impact of sleep restriction on subjective stress, and 10 assessing the causal impact of sleep restriction on aggression. The contributing studies comprised papers and theses published between 1977 and 2021. Overall, the studies included data from a total of 388,934 participants with an average age of 32.19 ($SD = 13.46$). Data was also gathered from countries across Asia, Africa, North America, South America, Europe, and Australia (see Supplementary Document for key characteristics of all studies contributing to the meta-analyses).

3.2. Inter-rater reliability of data extraction

Table 1 reports the key results from the inter-rater reliability of data extraction. The BMI mean and standard deviation variables had moderate Cohen's kappas (0.73 and 0.70 respectively). All other variables had strong to almost perfect Cohen's kappas ranging from 0.81 to 0.96. Percentage agreements for all variables were above 80% and therefore high according to the a priori benchmarks we set (see Methods section). As such, we are confident in the replicability of data extraction and

Table 1
Agreement between coders for each extracted variable.

Variable	Percentage agreement (%)	Kappa	*SE*	*t*	*p*
Sleep measure used	97.18	0.93	0.035	18.759	< 0.001
Stress/Aggression measure used	98.59	0.95	0.028	23.351	< 0.001
Correlation or t-value	95.71	0.94	0.028	58.786	< 0.001
Sample Size	84.51	0.81	0.046	59.826	< 0.001
Participant group	95.77	0.95	0.028	20.435	< 0.001
Nationality	98.59	0.96	0.026	20.900	< 0.001
Age (mean)	91.54	0.87	0.042	36.015	< 0.001
Age (standard deviation)	88.73	0.84	0.046	32.623	< 0.001
BMI (mean)	97.18	0.73	0.111	11.786	< 0.001
BMI (standard deviation)	94.44	0.70	0.122	11.004	< 0.001

coding.

3.3. Meta-analytic results

3.3.1. Correlational data

Table 2 reports the key results from the meta-analyses on correlational data. As predicted, in individuals from the general population, sleep was found to have a medium association with subjective stress ($r = 0.307$, $k = 281$, $p < .001$), and a small association with aggression ($r = 0.258$, $k = 51$, $p < .001$). As predicted, in individuals with sleep disorders, sleep was found to have a medium association with subjective stress ($r = 0.425$, $k = 9$, $p < .001$; see Figs. 2.1, 2.2, 3, and 4 for the correlation and 95% confidence intervals - *CIs* - for each contributing study; see the Supplementary Document for the specific study characteristics).

3.3.2. Experimental data

Table 3 reports the key results from the meta-analyses on causal data. As predicted, sleep had a small effect on subjective stress ($g = 0.403$, $k = 6$, $p = .017$), and a small effect on aggression ($g = 0.330$, $k = 10$, $p = .042$). Figs. 5 and 6 show the Hedges *g* for each contributing study, and their corresponding 95% *CIs* (for the characteristics of contributing studies, see the Supplementary Document).

3.4. Meta-regression results

Neither age nor BMI accounted for significant variance in the relationship between sleep and subjective stress (age: $R^2 < 0.01$, $Q = 0.00$, $p = .973$, $k = 242$; BMI: $R^2 < 0.01$, $Q = 3.39$, $p = .066$, $k = 50$). Similarly, age did not account for significant variance in the relationship between sleep and aggression (age: $R^2 < 0.01$, $Q = 0.08$, $p = .778$, $k = 47$).

3.4.1. Post hoc meta-regression results

Measurement type moderated the relationship between sleep and subjective stress in individuals from the general population ($R^2 = .31$, $QE = 4714.83$, $p < .001$, $k = 280$). We then analysed the sleep and subjective stress correlation, separated by measurement type (see Table 4). When sleep was measured objectively, via the PSQI, via single items assessing sleep quality, and via other multi-item sleep questionnaires, it was significantly associated with subjective stress. When sleep was assessed via single items assessing sleep duration, the relationship with subjective stress was not significant. Omnibus moderation of the association between subjective stress and sleep by measurement type was significant ($QM = 1394.17$, $p < .001$) indicating that measurement type influenced the magnitude of the sleep and subjective stress association. To follow this up, the betas to test argument was used to assess which associations to compare statistically. The results of these comparisons are reported in Table 5 and indicate that measurement type was influential for all pairwise comparisons.

3.5. Heterogeneity of effect sizes

As reported in Tables 2 and 3, Cochran's *Q* was significant for all main effects except for the causal sleep and subjective stress relationship. This indicates substantial heterogeneity among the effect sizes contributing to the meta-analyses excepting those contributing to the analysis of the causal effect of sleep restriction on subjective stress - wherein there was no between-effect size heterogeneity detected (Borenstein et al., 2014). Note however that the non-significant heterogeneity in the causal sleep and subjective stress relationship is likely due to the low number of contributing studies and therefore a potentially underpowered assessment of heterogeneity (see Higgins et al., 2003).

3.6. Study quality assessment

The quality of included studies ranged from 16 to 39 out of 39 ($M = 28.97$, $SD = 4.81$). Although the QuADS criteria has no cut-off score, 330 studies were scored above 50% suggesting at least moderate quality. To view the individual quality score for each contributing study, see the Supplementary Document.

3.7. Publication bias assessment

The association between sleep with both subjective stress and aggression in individuals from the general population demonstrated possible publication bias. Specifically, for the sleep and subjective stress relationship, Egger's test of the intercept was significant. However, the Begg and Mazumdar rank correlation test was non-significant and the funnel plot analysis displayed a symmetrical distribution (see Table 2 and Fig. 7). For the sleep and aggression relationship, the Begg and Mazumdar rank correlation test was also non-significant, however Egger's test of the intercept was significant, and the funnel plot analysis displayed a heavily left-skewed distribution (see Table 2 and Fig. 8). This suggests that there is a low likelihood of publication bias in the correlational sleep and subjective stress literature, and a high likelihood that significant results are published more than non-significant results when reporting on the sleep and aggression association.

For the relationship between sleep and subjective stress in individuals with sleep disorders as well as the causal relationship between sleep with subjective stress and aggression, the Begg and Mazumdar rank correlation test and Egger's test of the intercept were both non-significant, suggesting that publication bias is unlikely to exist in the literature published on these topics (see Tables 2 and 3). This result was further supported by the funnel plot analysis, where data from these relationships all demonstrated symmetrical distributions (see Figs. 9–11).

4. Discussion

The results of this meta-analysis provide important and novel insights into the association between - and causal impact of - sleep with subjective stress and aggression. In line with predictions from neurobiological models, sleep was significantly associated with both subjective

Table 2
Mean effects for sleep with subjective stress and aggression, and tests for publication bias in correlational data.

Subcomponent	*r*	95% CIs						Begg's method kendall's Tau	Egger's method Intercept a
		Lower	Upper	PVAF	*k*	*n*	*Q*		
Stress in individuals from the general population	0.307 ***	0.29	0.33	9.42	278	367,267	8423.83 ***	-0.07	0.90 *
Stress in individuals with sleep disorders	0.425 ***	0.27	0.56	18.06	9	1777	56.45 ***	0.14	1.62
Aggression individuals from the general population	0.258 ***	0.21	0.31	6.66	50	19,236	546.35 ***	0.08	2.42 **

Note. *$p < .05$, * * $p < .01$, * ** $p < .001$. A positive effect size indicates a detrimental effect from poorer sleep. PVAF = percentage of variance accounted for. k = the number of contributing studies. n = the number of participants that contributed to the effect. Imputed mean is random effects.

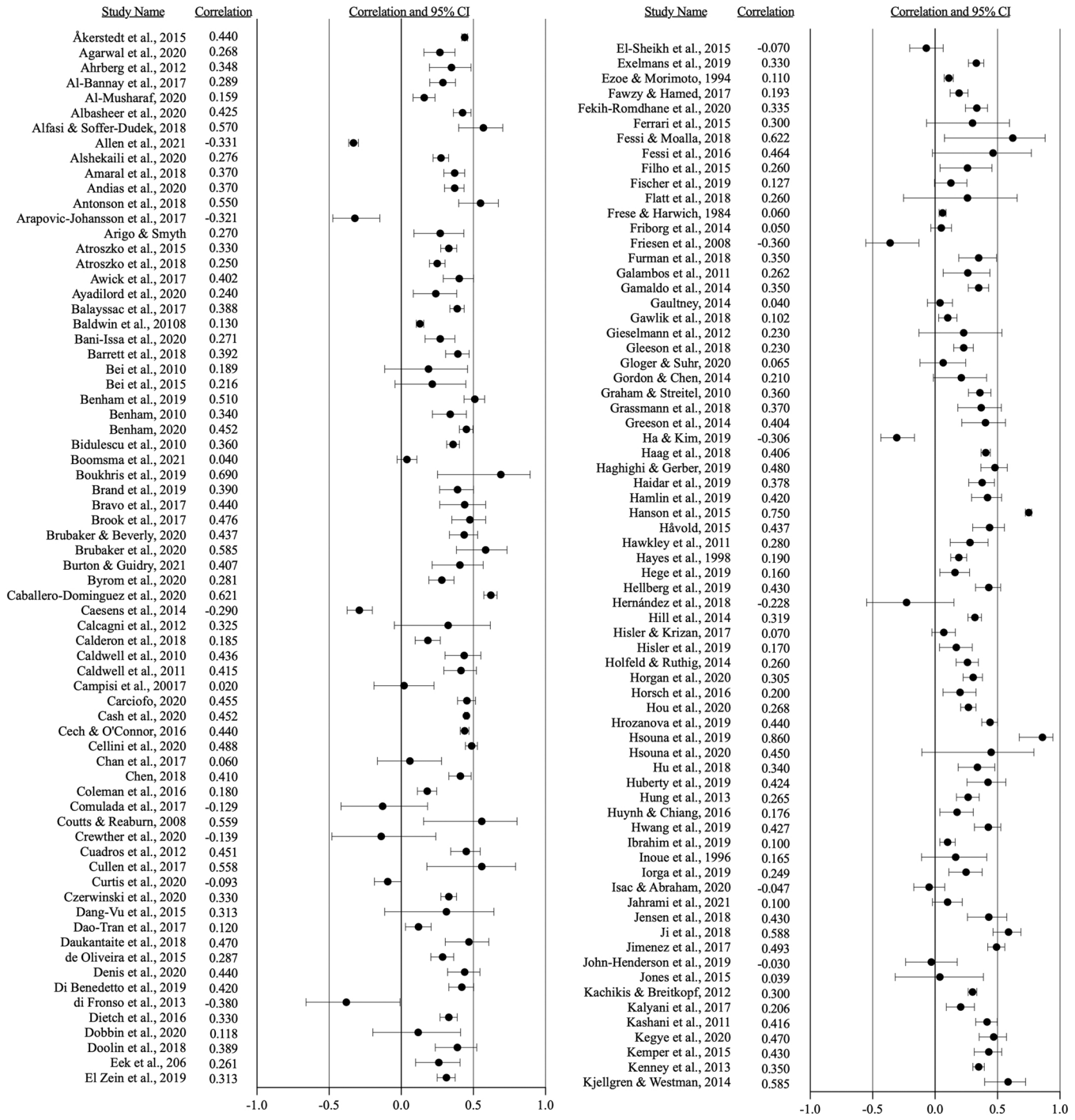

Fig. 2.1. Forest plot displaying the correlation and 95% confidence intervals for the first 140 studies contributing to the sleep and subjective stress association in individuals from the general population.

stress and aggression in individuals from the general population. In individuals with sleep disorders the predicted inverse association was also found between sleep and subjective stress. These findings indicate that poorer sleep is associated with elevated feelings of stress in both individuals from the general population and individuals with sleep disorders. Also as predicted, experimentally manipulated sleep restriction led to increased levels of subjective stress and aggression, suggesting that sleep restriction can directly cause increased subjective stress and aggression.

As noted previously, a relationship between sleep and subjective stress was anticipated based on broader findings from the neurobiological literature. Sleep deprivation has been shown to reduce activity in key neural regions required for the stress response (Javaheripour et al., 2019), and increase cortisol levels, which are also elevated in a stress response (Goodman et al., 2017; Leproult and Van Cauter, 2010; Nejtek, 2002; Redwine et al., 2000; Reinhardt et al., 2012; Stone et al., 2001; Wright et al., 2015). Indeed, in the neuroendocrine literature, sleep deprivation is often viewed as a stressor. This is because sleep appears to have suppressive effects on neuroendocrinological stress systems, whereas sleep deprivation and fragmentation tend to maintain the activity of the autonomic sympathetic system (see Meerlo et al., 2008 for a review). Such findings suggest that poor sleep can

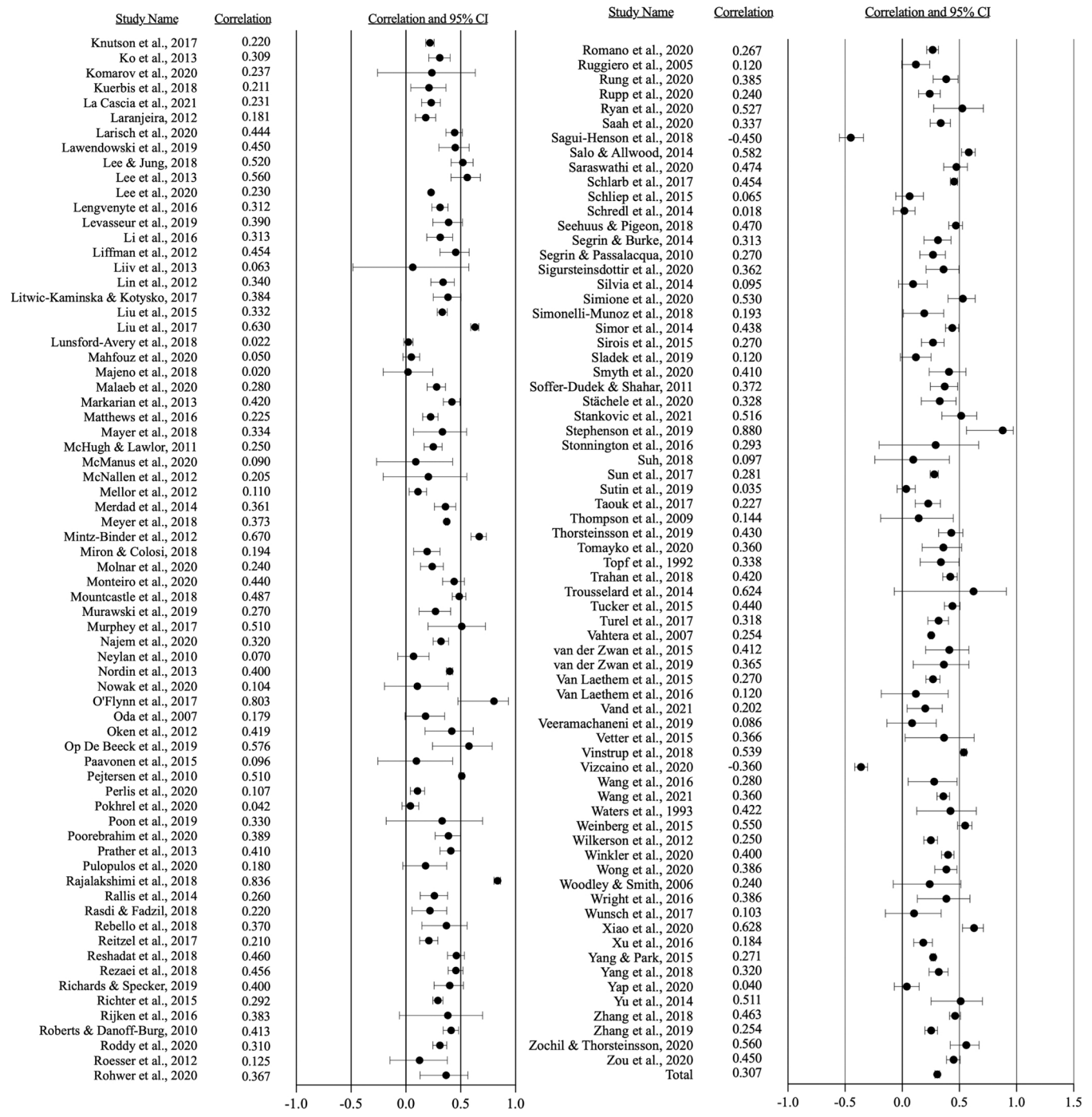

Fig. 2.2. Forest plot displaying the correlation and 95% confidence intervals for the latter 140 studies contributing to the sleep and subjective stress association in individuals from the general population.

directly increase stress. Specifically, that due to poor sleep there may be prolonged activation of the sympathetic nervous system and that this may lead to disruption in neuroanatomical regions responsible for regulating stress. More research is now required to understand how prolonged sleep restriction, fragmentation, or general disturbance – commonly evident in various sleep disorders – interacts with neurobiological aspects of the stress response over time.

For the sleep and subjective stress association, it is possible that this relationship is bidirectional. Studies have shown that increased levels of stress at night lead to decreases in slow wave sleep, REM sleep, sleep efficiency, and general sleep quality (Åkerstedt et al., 2012; Kim and Dimsdale, 2007). The correlation between sleep and stress in the current meta-analysis might therefore reflect poorer sleep contributing to increased stress, increased stress leading to poorer sleep, or some combination of the two. Thus, although in the present study, we found a direct effect of sleep restriction on subjective stress (which suggests that poor sleep can lead to increased subjective stress), because the direct effect of stress on sleep was not investigated, we cannot conclude that the reverse-causal relationship does not also exist. Future studies using longitudinal or experimental methods are needed to further clarify the direction of the relationship between sleep and subjective stress.

The findings surrounding the sleep and aggression relationship also align with the broader neurobiological literature. Again, as noted previously, key cortical and subcortical structures known to be critical for

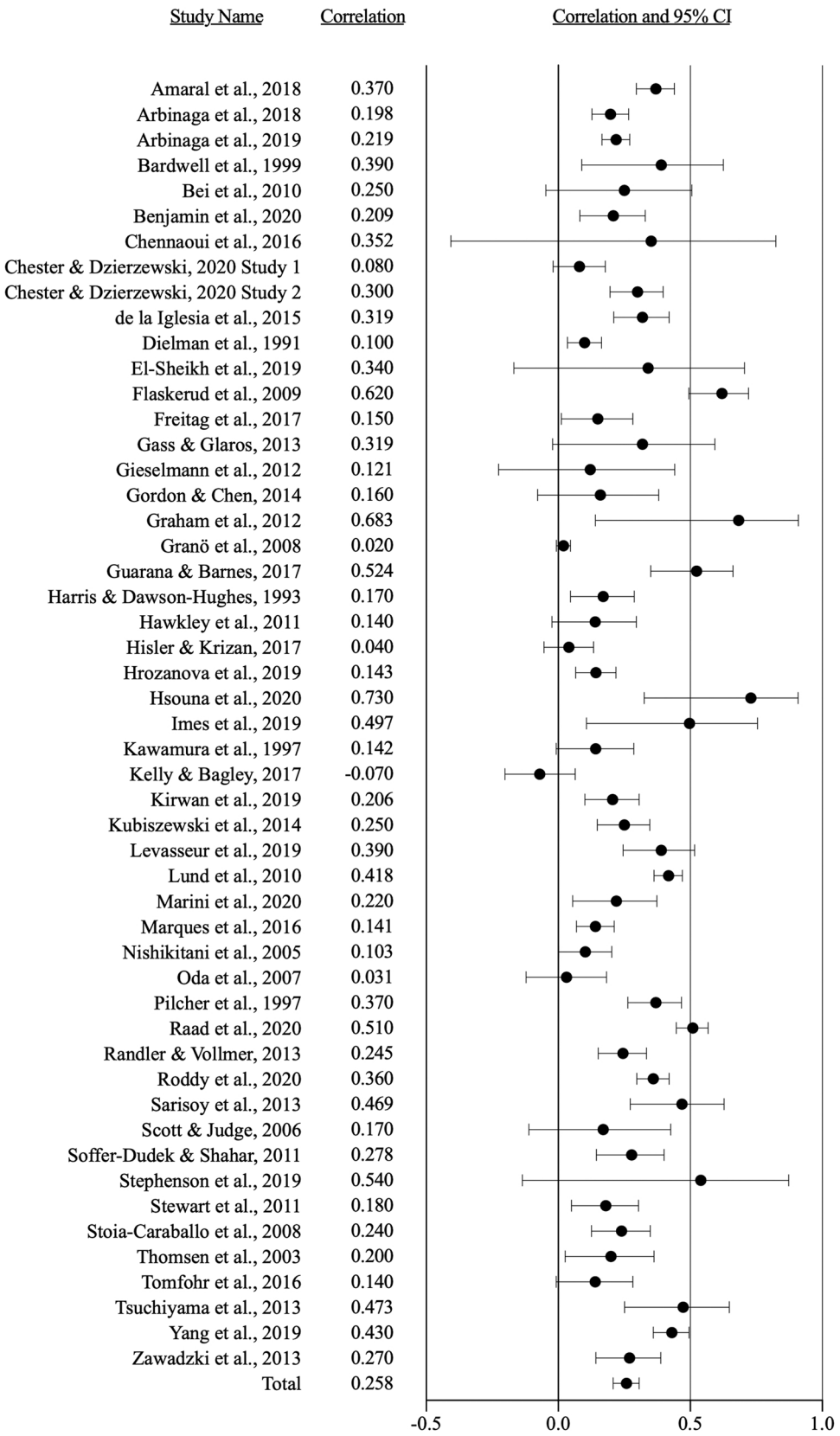

Fig. 3. Forest plot displaying the correlation and 95% confidence intervals for studies contributing to the sleep and aggression association.

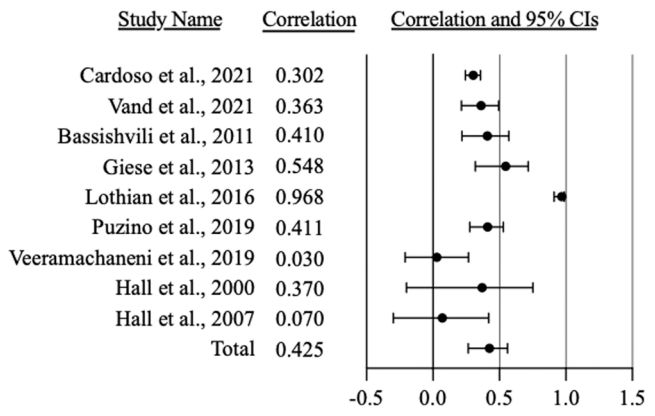

Fig. 4. Forest plot displaying the correlation and 95% confidence intervals for studies contributing to the sleep and subjective stress association in individuals with sleep disorders.

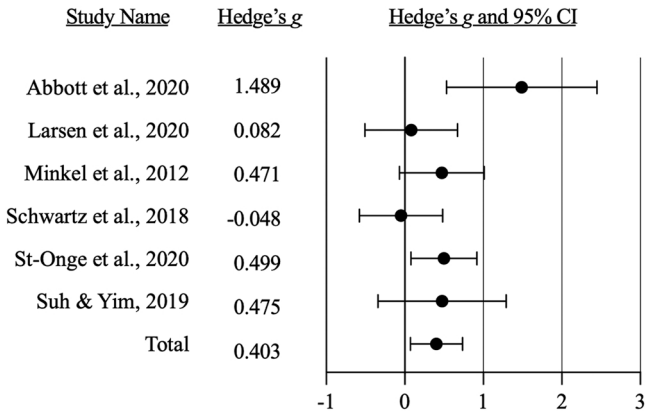

Fig. 5. Forest plot displaying the Hedge's *g* and 95% confidence intervals for studies contributing to the causal sleep and subjective stress relationship.

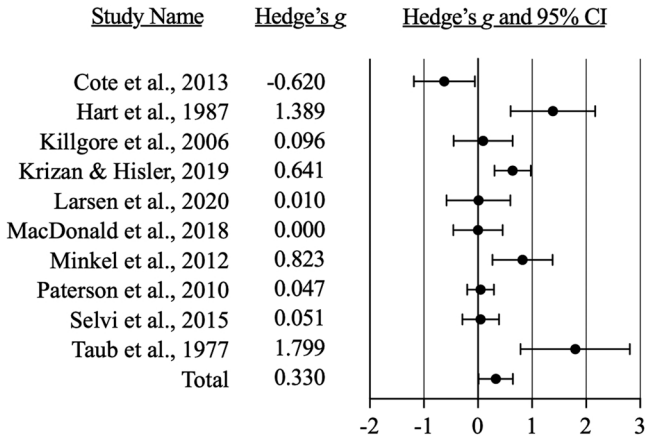

Fig. 6. Forest plot displaying the Hedge's *g* and 95% confidence intervals for studies contributing to the causal sleep and aggression relationship.

moderating aggressive behavior - including the prefrontal and posterior parietal cortices - demonstrate reduced cerebral metabolic rate for glucose after sleep deprivation (Bosch et al., 2013; Rosell and Siever, 2015; Shao et al., 2014; Thomas et al., 2000; Yoo et al., 2007). A leading hypothesis on the neurobiological link between sleep and aggression is that sleep deprivation can result in poor prefrontal cortical functioning which then negatively influences the ability to anticipate, delay, and initiate behavioral responses based on cognitive and social contexts (see Kamphuis et al., 2012). Furthermore, sleep deprivation has been found to lead to behavioral anomalies (e.g., weakening of goal directed behavior and an instability in emotional responses) which are associated with prefrontal cortical functioning (see Kamphuis et al., 2012 for a review). The neuroendocrine literature suggests a more complex relationship between sleep and aggression. Whilst sleep deprivation does lead to changes in testosterone and increases in levels of cortisol and serotonin, the relationship these changes have on aggression can be influenced by other factors that include sex, psychopathy and personality (Abu-Samak et al., 2018; Elmenhorst et al., 2012; Leproult and Van Cauter, 2010; Lopez-Rodriguez et al., 2003; Maggio et al., 2013; Oh et al., 2012; Redwine et al., 2000; Senthilvelan et al., 2006; Wright et al., 2015). Nevertheless, there is increasing evidence to suggest a role of serotonin in influencing aggression (see Kamphuis et al., 2012 for a review), and having now provided the strongest evidence to date that a significant association does exist between sleep and aggression, future research is required to gain a more precise understanding of the mechanisms by which sleep influences the neuroendocrinological underpinnings of aggression.

The results of this meta-analysis therefore suggest that sleep may play a role in understanding important aspects of our everyday behavior. Although the finding that these associations exist is not surprising in light of a considerable volume of prior research on this topic, the results of this meta-analysis provide the clearest evidence to date of the typical strength of these relationships, which speaks directly to their practical importance. For instance, it is clear that sleep restriction has a moderate effect on subsequent subjective stress and a small effect on subsequent aggression. This suggests that improving sleep in individuals could have

Table 4
Sleep and stress correlation in individuals from the general population separated by sleep measurement type.

Measure	Effect size (*r*)	Lower 95% CI	Upper 95% CI	*k*
Objective sleep measures	0.149	0.084	0.213	28
PSQI	0.246	0.175	0.316	121
Other multi-item sleep questionnaires	0.249	0.174	0.324	64
Single items assessing sleep quality	0.226	0.139	0.313	31
Single items assessing sleep duration	0.017	-0.064	0.099	38

Note. CI is Confidence Interval. PSQI is the Pittsburgh Sleep Quality Index.

Table 3
Mean effects for sleep with subjective stress and aggression, and tests for publication bias in experimental data.

Subcomponent	*g*	95% CIs						Begg's method kendall's Tau	Egger's method Intercept a
		Lower	Upper	PVAF	*k*	*n*	*Q*		
Subjective Stress	0.403 * *	0.07	0.74	4.80	6	174	9.12	0.20	2.21
Aggression	0.330 *	0.01	0.65	3.20	10	480	41.51 ***	0.40	2.21

Note. *$p < .05$, **$p < .01$, ***$p < .001$. A positive effect size indicates a detrimental effect from poorer sleep. PVAF = percentage of variance accounted for. *k* = the number of contributing studies. *n* = the number of participants that contributed to the effect. Imputed mean is random effects.

Table 5
Pairwise comparisons between sleep measurement type and subjective stress in individuals from the general population.

Comparison Measures	*QM*	*P*-value
Objective vs PSQI	793.07	< 0.001
Objective vs multi-item sleep questionnaires	421.06	< 0.001
Objective vs SIdur	62.78	< 0.001
Objective vs SIqual	178.67	< 0.001
PSQI vs multi-item sleep questionnaires	50.21	< 0.001
PSQI vs SIdur	91.13	< 0.001
PSQI vs SIqual	47.45	< 0.001
Multi-item sleep questionnaires vs SIdur	71.05	< 0.001
Multi-item sleep questionnaires vs SIqual	43.63	< 0.001
SIdur vs SIqual	36.01	< 0.001

Note. QM = test statistic of the Wald-type test of the model coefficients. Objective = objective measures of sleep such as polysomnography or actigraphy. PSQI = the Pittsburgh Sleep Quality Inde. SIdur = single items assessing sleep duration. SIqual = single items assessing sleep quality.

quite meaningful effects on an individuals' subsequent stress but relatively less impact on their subsequent aggression. Any attenuation of either type of behaviour however should have positive effects on the many broader aspects of health and wellbeing which are known to be negatively impacted by both stress and aggression (e.g., cardiac risk, morbidity, anxiety, depression etc.; Boals and Banks, 2012; DeLongis et al., 1988; Quick et al., 1987).

4.1. Moderators

Surprisingly, neither age nor BMI accounted for significant variance in the relationship between sleep and subjective stress. Age also did not account for any variance in the relationship between sleep and aggression. One possibility for these findings is that, although sleep has been found to worsen with age (Bowman et al., 2020), skills such as emotion regulation (which generally increase with age; Brummer et al., 2014; Orgeta, 2009; Yeung et al., 2011) 'buffer' the effect poor sleep may have on stress and aggression. Regarding BMI, its relationship with sleep and subjective stress or aggression is likely highly nuanced. For instance, although BMI is a risk factor for sleep disordered breathing (e.g., obstructive sleep apnea; Young et al., 2005), the impact of BMI on sleep disordered breathing appears to be more apparent in the morbidly obese range (>35 kg/m^2). This is reflected by cut-off scores in tools designed to screen for sleep disorders, whereby a BMI higher than 35 kg/m^2 is used in combination with other predicters to determine risk of sleep apnea (Chung et al., 2008). This may explain why no variance was accounted for by BMI as only one study in the meta-analysis reported participants with a mean BMI higher than 35 kg/m^2 (McNallen et al., 2013). It is also possible that BMI may share a bidirectional relationship with sleep. A meta-analysis of longitudinal associations between sleep duration and obesity among adults found that shorter, but not longer sleep durations, were associated with later obesity (Wu et al., 2015). More research should be conducted to better understand which aspects of sleep are predictive of increases in BMI (e.g., subjective sleep quality, time spent in different sleep stages etc.).

We also examined sleep measurement type as a moderator variable for the sleep and subjective stress correlational relationship in individuals from the general population. After categorizing sleep assessments into five different types (objective assessments, PSQI scores, single items assessing sleep quality, single items assessing sleep duration, and other multi-item sleep questionnaires used), the results revealed that measurement type moderated the relationship between sleep and subjective stress. Further sub-analyses demonstrated that sleep was associated with subjective stress for all measurement types except when measured with single items assessing duration. When sleep was assessed objectively, the magnitude of this relationship was small. Contrastingly, when sleep was assessed via the PSQI, other multi-item sleep questionnaires, or a single item assessing sleep quality, the magnitude of this association was small to moderate. The failure to identify any association between single items assessing sleep duration and subjective stress is notable as this is a common method for assessing subjective sleep. Indeed, 44 studies contributing to the sleep and subjective stress relationship assessed sleep using this approach. This null finding, coupled with the finding of a significant association between a single item indicator of subjective sleep quality and subjective stress, provides clear guidelines for future research studies focused on the relationship between sleep and stress that are only able to include a single survey item to index subjective sleep. Specifically, it suggests that

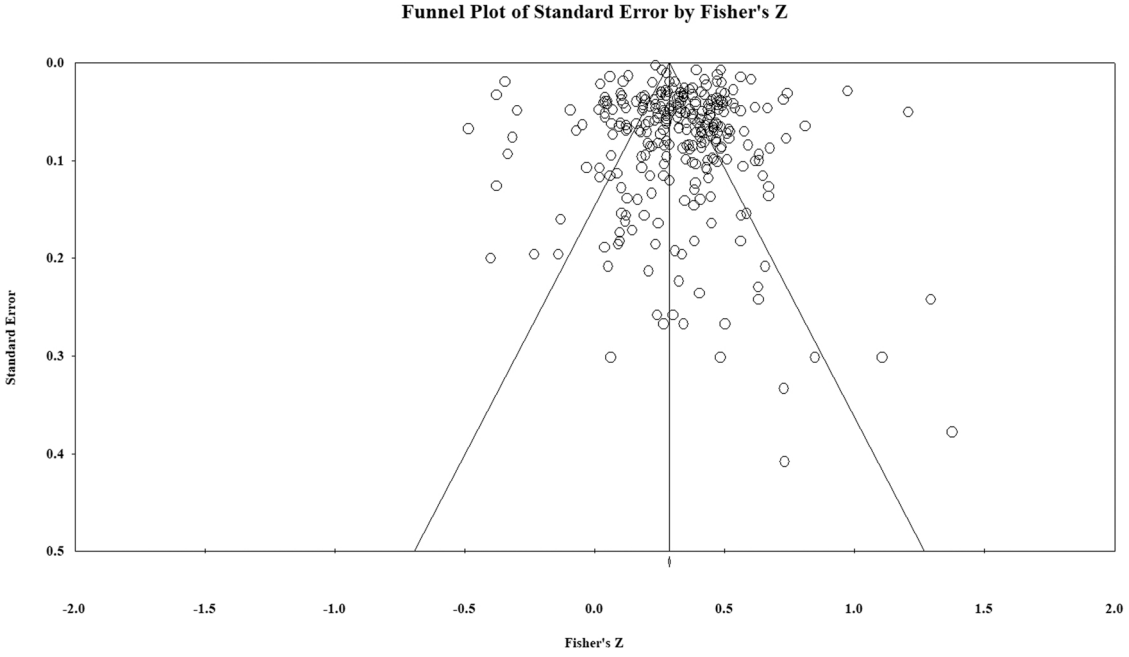

Fig. 7. Funnel plot displaying the symmetrical distribution of effect sizes plotted against their standard error for studies contributing to the relationship between sleep and subjective stress in individuals from the general population.

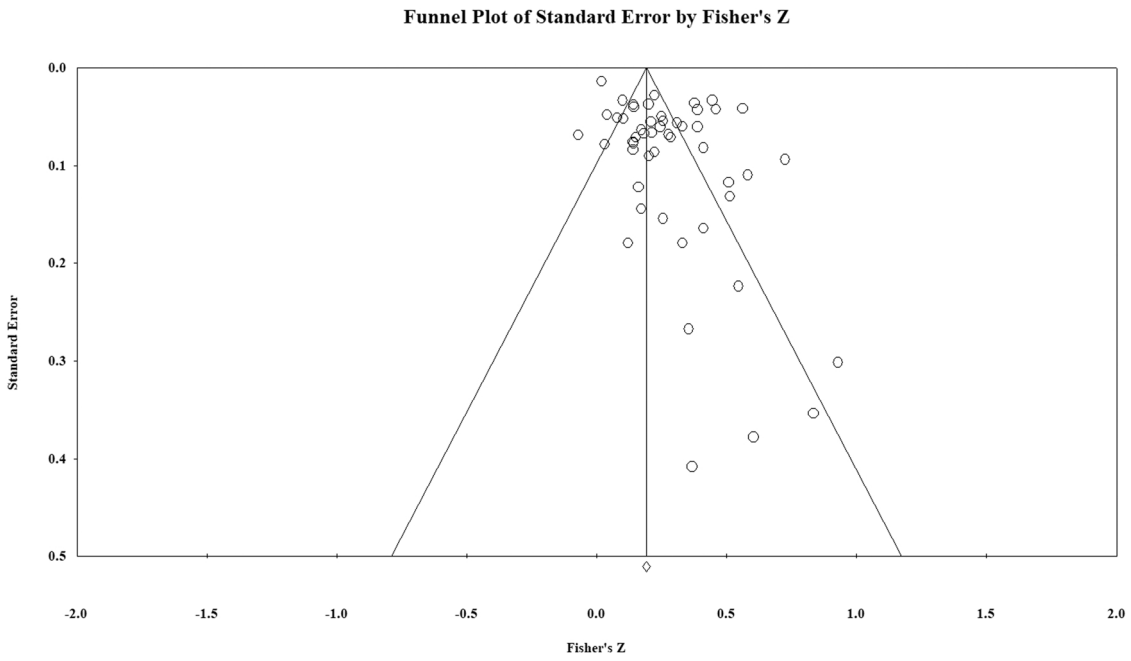

Fig. 8. Funnel plot displaying the asymmetrical distribution of effect sizes plotted against their standard error for studies contributing to the relationship between sleep and aggression.

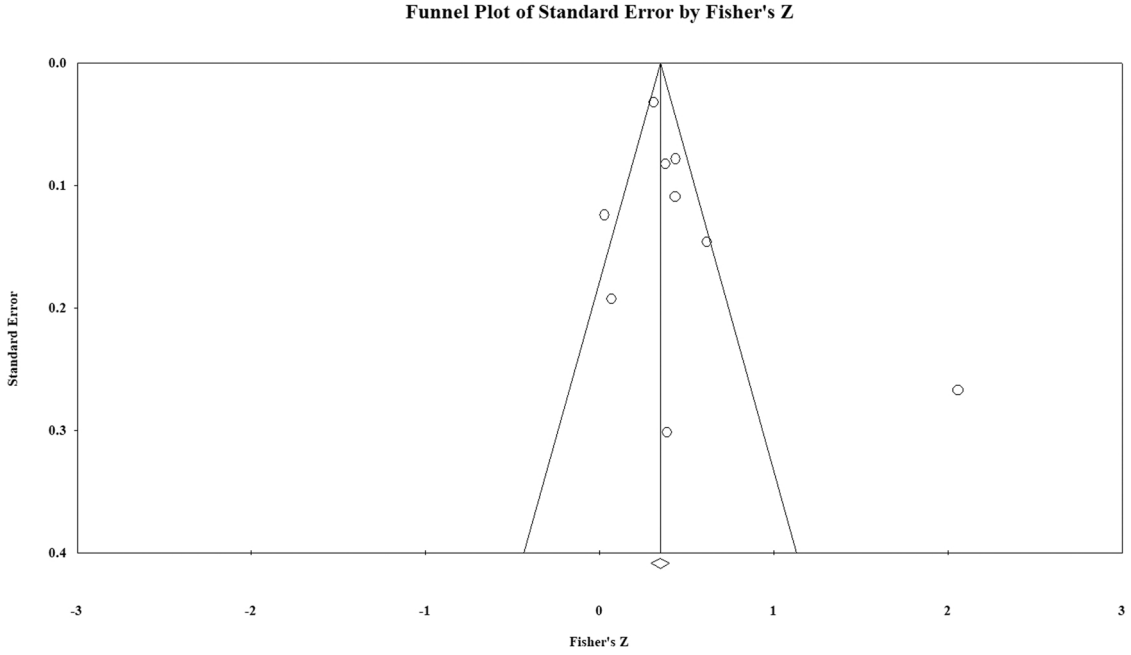

Fig. 9. Funnel plot displaying the symmetrical distribution of effect sizes plotted against their standard error for studies contributing to the relationship between sleep and subjective stress in individuals with sleep disorders.

in such studies, subjective sleep quality should be measured in preference to subjective sleep duration.

However, it is important to caution that these analyses were conducted post hoc, and consequently we had no a priori predictions for these effects. Further research is therefore now needed to directly test the mechanisms that might drive these measurement effects. One possibility is that the larger observed effects for subjective relative to objectively indexed sleep, and for sleep quality relative to sleep duration, reflect a 'meaning response,' whereby psychological changes occur as a result of the meaning associated with an experience or object (Moerman, 2002). By this view, individuals may perceive their sleep quality as poorer than desired and subsequently come to have the psychological experience they believe is appropriate given their sleep experience (i.e., reporting feeling more stressed). More research is now needed that directly compares associations between relevant behavior, and both objective and subjective estimates of sleep, because these experiences of sleep may differentially relate to behaviour and emotion. Future studies should be designed to determine whether an individual's perception of their sleep quality impacts their stress regardless of their objective sleep (i.e., by controlling for objective sleep measures).

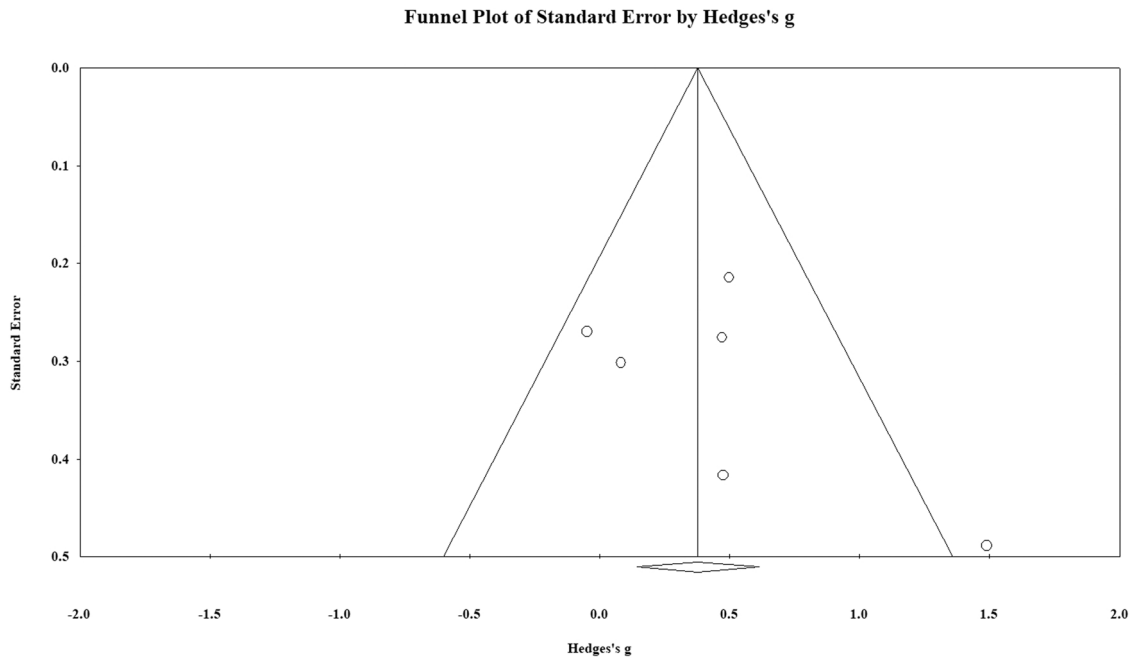

Fig. 10. Funnel plot displaying the symmetrical distribution of effect sizes plotted against their standard error for studies contributing to the causal sleep and subjective stress relationship.

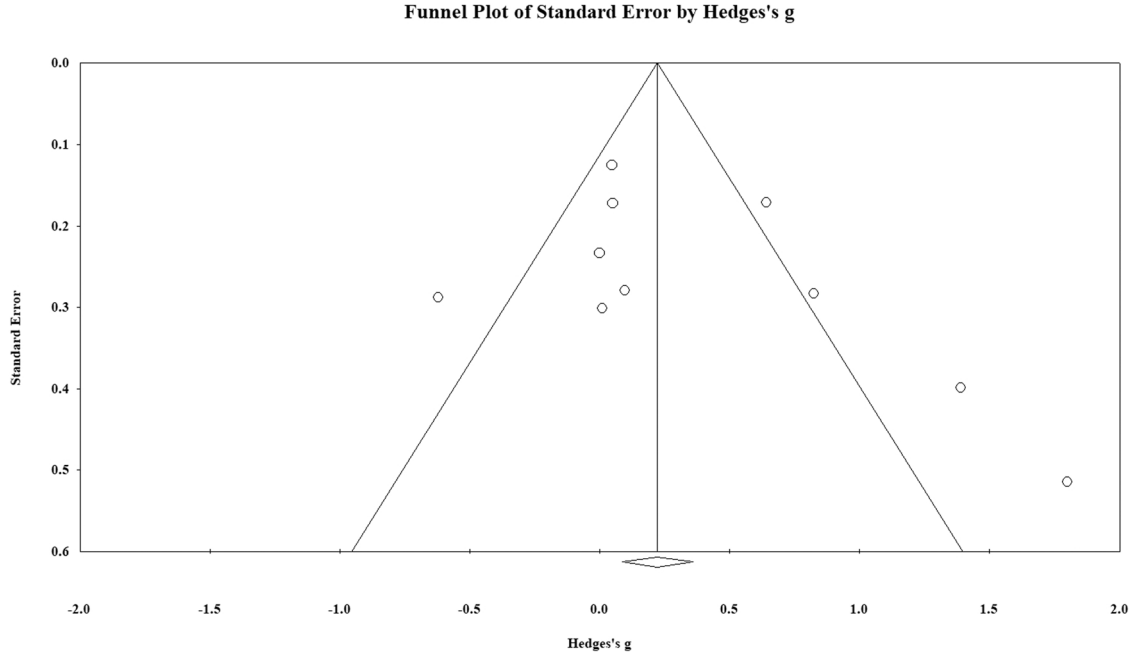

Fig. 11. Funnel plot displaying the symmetrical distribution of effect sizes plotted against their standard error for studies contributing to the causal sleep and aggression relationship.

4.2. Broader implications

Sleep disturbances are increasing in prevalence in non-clinical populations (Adams et al., 2016; Ford et al., 2015; Knutson et al., 2010; Kronholm et al., 2008), and are also a common problem in clinical disorders (Petit et al., 2004). For instance, it is estimated that approximately two thirds of individuals with Alzheimer's disease and 83% of individuals with depression suffer from sleep disturbances (Guarnieri et al., 2012; Nutt et al., 2008). Aggressive behavior is common in neurological disorders (Bidzan et al., 2012; Connor et al., 2010; Dowson and Blackwell, 2010; Fisher et al., 2014; Hoptman et al., 2014; Josephs Jr et al., 2011; Lobbestael et al., 2013; Reus et al., 2018; Zhao et al., 2015), and stress levels tend to be elevated in neuropsychiatric disorders (Anand et al., 2016; Combs et al., 2015; Lackschewitz et al., 2008; Liu et al., 2017; Nunnemann et al., 2012). According to the results of this meta-analysis, sleep disturbances may be directly contributing to these heightened levels of stress and aggression. The results therefore highlight the importance of prioritising healthy sleep to minimise stress and aggression, and thus their negative consequences, both in the general population and clinical populations. Furthermore, as this meta-analysis included data from a wide range of countries spanning six out of seven continents, the potential impact of these relationships are likely to be

generalisable to individuals world-wide.

4.3. Future directions

The integration of data from many different countries is an important strength of this meta-analysis and provided considerable confidence in the key findings: sleep is both associated with and causally impacts subjective stress and aggression in individuals from the general population, and is significantly associated with subjective stress in individuals with sleep disorders. However, having now established that these relationships exist, the next important step in this literature is to unpack the nuances of these relationships, as well as investigate the sleep and aggression relationship in individuals with sleep disorders.

There is a particular need for longitudinal research methods, as these directly speak to whether long-term sleep disruption might lead to particularly elevated levels of stress and aggression. Our literature search revealed that, to date, very few longitudinal studies have looked at these associations in healthy adult populations. Although two studies investigated the association between sleep and stress over 12 days (Yap et al., 2020) and 42 days (Åkerstedt et al., 2012), neither assessed whether the magnitude of this relationship changed across the duration of the study. We are also not aware of any longitudinal studies assessing the impact of continuous sleep restriction on aggression. We therefore consider extended longitudinal research the next important step in this literature. Specifically, it would be beneficial to monitor neuroanatomical and neuroendocrinological changes across continuous sleep restriction (or in individuals with sleep disorders), and how these are related to real-life aggressive or stress responses. Such an approach would provide the clearest picture to date of the temporal dynamics of these relationships, but also, how these associations map onto underlying neurophysiology (thereby speaking to neurobiological mechanisms that drive these associations). This would be especially helpful in the neuroendocrinological aggression and sleep literature where this relationship appears to be particularly complex, and less well understood.

This meta-analysis also highlighted an important gap, in showing that the acute effects of poor sleep on subjective stress and aggression in individuals with sleep disorders remains to be established. Experimental work is now needed, particularly since competing predictions seem possible. On the one hand, it may be that individuals with sleep disorders are particularly susceptible to the acute negative consequences of sleep disruption since sleep losses over time will accrue. Alternatively, it may be that this group is better able to cope with any effects of sleep disruption because they are better practiced at doing so. Future work is needed to test which of these possibilities is correct.

It is also possible that other variables not investigated here may moderate the observed relationships. For instance, it is well documented that there are sex differences in stress responses across the lifespan (see Bale and Epperson, 2015 for a review), as well as in aggression (see Archer, 2004 for a meta-analysis). Similarly, sex differences have been identified across objective and subjective measures of sleep, whereby females show better objective sleep quality but poorer subjective sleep quality than males (see Mong and Cusmano, 2016 for a review). In the studies contributing to the current meta-analysis, only one reported on sex differences between sleep and stress, and this found that the correlation between sleep and stress was larger for female relative to male participants (Amaral et al., 2018). Only one study reported an assessment of sex differences between sleep and aggression and this revealed that the association between sleep and aggression was larger for males than it was for females (Kelly and Bagley, 2017). Unfortunately, it was not possible to formally test the role of sex as a moderator in the present meta-analysis because sex-stratified data were seldom reported, but this is an important area for future research.

Despite the clarity and consistency of the findings, it is important to caution that this meta-analysis found some evidence to suggest publication bias. Evidence of publication bias was detected for individuals from the general population in the association between sleep and stress, as well as between sleep and aggression. This suggests that the absolute size of the mean effects detected may represent an over-estimate of the true population effects for these two specific associations. However, no evidence of publication bias was identified for any other mean effects, and perhaps most importantly, did not emerge in the literature examining the causal effect of sleep deprivation on subjective stress or aggression. The finding of this bias does though reinforce the need for future studies to be clearly preregistered, and publication practices to continue changing so that the quality of the study, and not the significance of the results, is the primary basis for accepting a manuscript for publication.

4.4. Conclusion

Sleep disturbances are very common in modern society and are predicted to continue increasing in prevalence. The results of this meta-analysis provide compelling evidence that poorer sleep is associated with - and causes - increased subjective stress and aggression. Future research is now needed to clearly understand the temporal dynamics of this relationship, as well as the precise neurobiological mechanisms involved.

Financial disclosure

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Data Availability

A link is provided in the methods section that leads to the data used in the study and preregistration files.

Acknowledgements

The authors would like to thank all individuals who provided further information about their studies that contributed to this meta-analysis (please see the Supplementary Document for a list of all authors who provided further information for this meta-analysis).

Conflict of interest

None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2022.104732.

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