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# Insomnia and emotion dysregulation: a meta-analytical perspective integrating regulatory strategies and dispositional difficulties

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ABSTRACT

Insomnia and emotion dysregulation are intricately related, yet their aggregate association across different domains of emotion dysregulation and the effect of moderating factors including health-related status, age, and gender remain unclear. This systematic review and meta-analysis synthesized data from 57 studies, pooling 119 effect sizes from correlational and 55 effect sizes from group comparison studies. By separate analyses, we assessed both the strength of the association and whether clinically significant insomnia symptoms exacerbate difficulty in regulating emotion. Correlational analyses revealed a significant association between insomnia symptoms and emotion dysregulation, primarily in individuals with serious health-related conditions (Fisher $Z_{no\text{-}serious\ condition} = 0.22$, Fisher $Z_{serious\text{-}conditions} = 0.37$, $p < 0.00001$). Group comparison analyses indicated that clinically significant insomnia symptoms present worse emotion dysregulation regardless of health-related status (Hedges' $g = 0.99$, $p = 0.01$). The reliance on maladaptive emotion regulation strategies and difficulties in dispositional domains of emotion regulation, particularly impulsivity, were more strongly associated with insomnia than challenges related to adaptive strategies. Age and gender did not impact these associations in either type of study. These findings underscore a robust link between insomnia and emotion dysregulation, suggesting the potential benefits of integrating emotion regulation skills into insomnia management to improve therapeutic outcomes.

## 1. Introduction

Insomnia, the second most common mental health condition [1], is associated with high socioeconomic costs and health risks [2,3]. It is characterized by nocturnal symptoms, including difficulty falling, staying asleep throughout the night, or premature waking up in the morning [4,5]. These symptoms are accompanied by impaired daytime social or behavioral functioning [4]. Chronic insomnia disorder entails persistent symptoms that last for a minimum of three months and occur at least three times per week [5]. Insomnia disorder is diagnosed in 10 %, but insomnia symptoms affect 30–35 % of the general population [3].

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**Glossary of terms (in alphabetical order)**

**Adaptive** Reduced use of adaptive strategies
**AIC** akaike information criteria
**Awareness** lack of emotional awareness
**AXIS** appraisal tool for cross-sectional studies
**BIC** bayesian information criteria
**Clarity** lack of emotional clarity
**DERS** difficulty in emotion regulation scale
**DERS-Total** total score of difficulty in emotion regulation scale
**DSM** diagnostic and Statistical Manual of Mental Disorders
**ERQ** emotion regulation questionnaire
**Function** lack of emotional functionality
**Goals** difficulties engaging in goal-directed behavior
**ICD** International Classification of Diseases
**Impulse** difficulties in impulse control
**Impulsivity** deficiency in reducing the urgency of emotion
**ISI** insomnia severity index
**LRT** likelihood ratio test
**Maladaptive** Heightened use of maladaptive strategies
**MARS** meta-analysis reporting standards
**Non-acceptance** non-acceptance of emotional responses
**NOS** newcastle-ottawa scale
**PI** prediction intervals
**PFC** prefrontal cortex
**PRISMA** preferred reporting items for systematic reviews and meta-analyses statement
**RVE** robust variance estimation
**Strategy** inflexible strategy selection
**sgACC** subgenual anterior cingulate cortex
**TOPICS + M** time, outcome, population, intervention, comparison, study design, and moderators
**V** variance-covariance matrix

Insomnia can develop in any individual. However, it often persists in those who exhibit maladaptive sleep patterns in response to stress [6,7]. This vulnerability arises from disruptions in intrinsic sleep-permissive mechanisms, which originate within brain circuits responsible for regulating arousal and emotional distress [8–11]. Individuals with insomnia often demonstrate inappropriate emotional responses to situational demands, reflecting impairments in these neural circuits [12, 13]. Furthermore, insomnia is associated with an increased risk of developing various mental health disorders [14–16] including anxiety, depression, and post-traumatic stress disorder [17–20], which are characterized by dysregulations in arousal and emotional processing [21–23]. Overall, these findings emphasize the strong interplay between insomnia and deficits in emotion processing and regulation.

Emotion regulation is the ability to manage one's emotional state in alignment with the demands of social and environmental contexts [24]. The primary aim of emotion regulation is to facilitate goal achievement and meet individual needs by modulating emotional experiences [25]. Gross's (1998) process model of emotion regulation conceptualizes it as occurring at different stages of the emotion-generative process: situation, attention, appraisal, and response phases [26,27]. Individuals use various regulatory strategies at these stages to initiate, modulate, or terminate emotional responses, adjusting them to the context. While a flexible use of all emotion regulation strategies is functional, some strategies have been described as more adaptive or maladaptive than others in most situations. Adaptive strategies help individuals manage emotions in a healthy and flexible way, often by intervening early in the emotional process. These strategies, such as reappraisal (reinterpreting a situation to alter its emotional impact), promote emotional adjustment and well-being. Some later-stage strategies, like acceptance (allowing emotions without trying to change them), are also adaptive as they do not attempt to counteract emotional responses. In contrast, maladaptive strategies focus on controlling or modifying emotions after they have already occurred and are considered dysfunctional when used rigidly or inappropriately. Examples include suppression and rumination, which can increase emotional distress and reduce psychological flexibility [28].

When emotion regulation is impaired, it could lead to emotion dysregulation, characterized by heightened, poorly controlled emotional reactivity, difficulties in achieving goals, and reduced overall well-being [25]. However, emotion dysregulation is not merely the absence of effective regulation; it is a distinct construct with its own conceptualization. According to Gross's process model, emotion dysregulation occures when individuals over-rely on maladaptive strategies or fail to use adaptive strategies effectively in response to emotional challenges [24]. More recent perspectives expand this understanding by proposing that emotion dysregulation is not only a failure in selecting or implementing specific regulatory strategies but also involves dispositional difficulties—trait-like tendencies that impact an individual's overall emotion regulation patterns across different situations [29]. These difficulties manifest in three key areas [29]: *(i) emotional functionality:* struggles with emotional awareness, clarity, or acceptance; *(ii) reducing the urgency of emotion:* challenges in controlling impulsive behaviors and maintaining goal-directed actions during intense emotional states; and *(iii) flexible strategy selection:* an inability to adaptively choose emotion regulation strategies based on situational demands and personal goals, leading to a rigid reliance on a limited set of strategies. Building on these perspectives, Gratz and Roemer (2004) proposed a multidimensional dispositional model for emotion dysregulation that incorporates these core domains [29]. According to this model, individuals experience emotion dysregulation when they exhibit difficulties in at least one of these dispositional areas. Considering these perspectives on emotion dysregulation, the present study adopts a comprehensive framework that integrates both strategy-based and dispositional aspects, examining how specific adaptive and maladaptive strategies, along with key dispositional difficulties, are associated with insomnia (Fig. 1).

Recent systematic reviews and meta-analyses [30–34] have significantly advanced our understanding of the interplay between sleep disturbances and emotion regulation. However, important gaps in the context of insomnia and emotion dysregulation still remained. Previous research has lacked a unified conceptualization of emotion dysregulation, often relying on broader, overlapping constructs such as emotion, affect, and mood or focusing on emotional reactivity rather than the impaired regulatory processes of emotion. In addition, sleep deprivation is frequently used as an experimental model of insomnia despite being theoretically and clinically distinct from insomnia [35]. This limits the applicability of findings to real-world insomnia conditions. Moreover, no prior meta-analysis has quantitatively synthesized the relationship between insomnia and emotion dysregulation as a distinct, unified construct encompassing both strategy-based and dispositional domains. As a consequence, it remains unclear whether insomnia is more strongly associated with the frequent use of maladaptive strategies, the failure to employ adaptive ones, or difficulties in dispositional domains of emotion regulation. Furthermore, the distinction between assessing the strength of this relationship and determining whether insomnia symptoms or disorder contribute to greater emotion dysregulation remains unexamined. Additionally, while some studies explore this association in populations exposed to significant life stressors or psychiatric disorders, no quantitative synthesis has specifically evaluated these cohorts. Finally, the moderating effects of age and gender—both key determinants of insomnia prevalence—on the relationship between

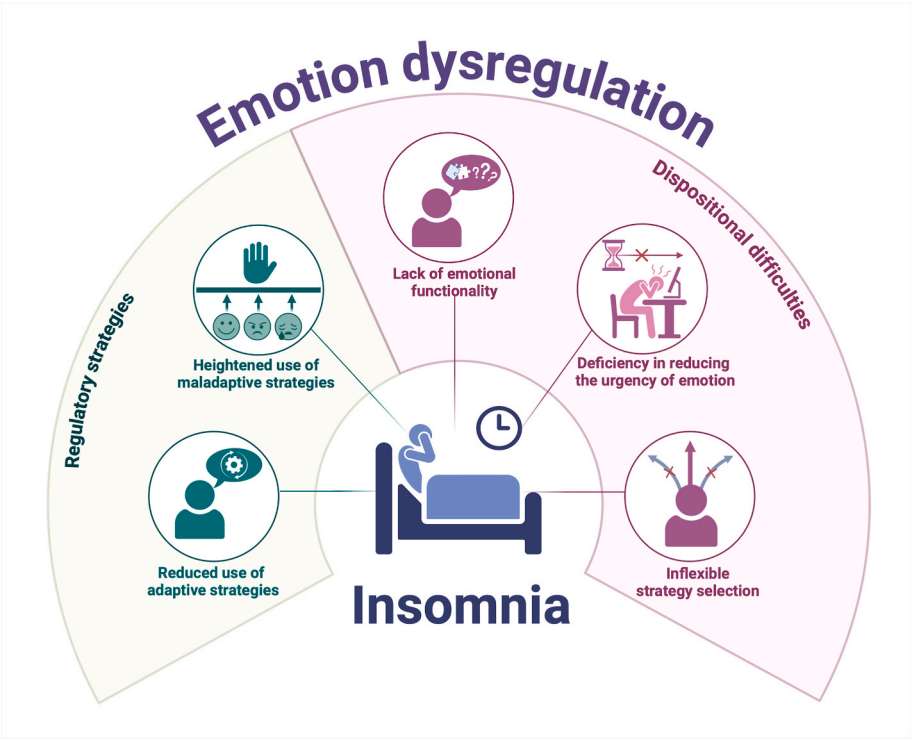


**Fig. 1.** Infographic on the association between insomnia and domains of emotion dysregulation. Created in BioRender. Samea, F. (2025) https://BioRender.com/i48e500.

insomnia and emotion dysregulation have not yet been adequately documented.

This study addresses the above-mentioned critical gaps in the existing literature on the relationship between insomnia and emotion dysregulation. To overcome the lack of clear constructs for insomnia and emotion dysregulation, we explicitly focused on insomnia symptoms rather than experimental sleep deprivation. In addition, we employed a regulatory framework for emotion dysregulation that specifically includes a well-known repository of maladaptive (i.e., avoidance, rumination, suppression) and adaptive (i.e., problem-solving, acceptance, reappraisal) strategies, along with difficulties in subdomains of Gratz and Roemer's dispositional model [29]. To provide quantitative estimations, we conducted a meta-analytical synthesis of existing findings to generate robust pooled effect sizes. Recognizing the need to differentiate the degree of association from whether the presence of clinically significant insomnia symptoms contributes to greater emotion dysregulation, we included both correlational and group comparison studies, conducting separate sets of analyses for each study design. Within both study types, we examined how the relationship between insomnia and emotion dysregulation varies based on participants' health-related status (e.g., individuals without serious conditions vs. those with serious conditions) and explored the effects of various emotion dysregulation domains as well as age and gender (the female-to-male ratio) on this association.

## 2. Methods

### 2.1. Transparency and openness

We followed the strict methodological recommendations based on the best-practice methods for conducting systematic reviews and meta-analyses in the health sciences [36]. In particular, we first utilized the TOPICS + M framework, considering time, outcome, population, intervention, comparison, and study design alongside potential moderators, to guide the formulation of the research question, and in-/exclusion criteria. Next, to ensure both proper reporting and methodological rigor, we followed the preferred reporting items for systematic reviews and meta-analyses statement (PRISMA) standards [37] and conducted additional quality control measures. Finally, we followed the meta-analysis reporting standards (MARS) guidelines [38] for reporting the findings. The current meta-analysis was pre-registered in the Prospective Register of Systematic Reviews (PROSPERO) (CRD42019123986).

### 2.2. Literature search

We searched multiple electronic databases: PubMed, Scopus, Web of Science, and PsycINFO, as well as gray literature sources: ProQuest, psyRxiv, medRxiv, and OSF. This approach allowed us to identify both peer-reviewed reports and gray literature sources containing relevant findings. Gray literature reports encompassed preprints, conference abstracts, unpublished manuscripts, and theses/dissertations. These databases were searched up to the end of March 2024. The search terms included (emotion) AND ((regulation) OR (suppression) OR (acceptance) OR (avoidance) OR (reappraisal) OR ("problem-solving") OR (rumination)) AND ((insomnia) OR (sleeplessness) OR ("sleep disturbances") OR ("sleep problems") OR ("initiating sleep") OR ("maintaining sleep") OR ("early awakening") OR ("sleep onset latency") OR ("non-restorative sleep")). The search terms were adapted to match the specific structure of each database (see more details in the supplementary material).

### 2.3. Selection criteria

To ensure systematic, consistent, and transparent study selection, we structured the criteria within the TOPICS + M framework. We first predefined potential moderators, as Johnson et al. (2019) recommended, to address study heterogeneity and systematically evaluate their potential effects on the outcomes [36]. Our set of moderators includes individuals' health-related status, age, gender, and various

domains of emotion dysregulation, as follows: maladaptive strategies (avoidance, rumination, suppression), adaptive strategies (problem--solving, acceptance, reappraisal), and difficulties in dispositional domains (emotional functionality, reducing the urgency of emotion, and flexible strategy selection). The study selection criteria were then organized based on these moderators and according to each TOPIC element:

- ***T:*** *Time concerns the period when studies are of interest.*

This meta-analysis did not specify limitations regarding the time periods during which the included studies were conducted or considered relevant.

- ***O****: Outcome refers to the specific measure used to assess the relationship between the variables.*

**Inclusion:** Studies must report the relationship between insomnia symptoms and emotion dysregulation, represented by: *(i)* Pearson's correlation coefficient between insomnia and emotion dysregulation scores. Insomnia symptoms must be measured using validated scales specifically designed to assess insomnia conditions, such as the insomnia severity index (ISI) [39]. Emotion dysregulation scores must be measured using the difficulty in emotion regulation scale (DERS) [29], which measures dispositional subdomains, or validated self-reported scales like the emotion regulation questionnaire (ERQ) [40], which assess our prespecified maladaptive and adaptive emotion regulation strategies, including avoidance, rumination, suppression as well as problem-solving, acceptance, and reappraisal; *(ii)* Mean differences in emotion dysregulation between individuals with clinically significant insomnia symptoms and good sleepers. Clinically significant insomnia symptoms must be identified based on clinical interviews (e.g., diagnostic and statistical manual of mental disorders (DSM), international classification of diseases (ICD)), or above-threshold scores on validated scales like the ISI. Good sleepers must have no clinically significant insomnia symptoms, as determined by the same tools, typically scoring within the normal range. Studies must report the mean difference between the groups on dispositional subdomain scores from the DERS, or adaptive/maladaptive prespecified regulation strategies scores from validated scales such as ERQ.

**Exclusion:** Studies using non-validated tools or reporting irrelevant outcomes, such as odd-ratio for a binary presence or absence of emotion dysregulation in individuals with insomnia symptoms compared to good sleepers, were excluded. Studies assessing distinct variables of emotion generation, emotional reactivity, emotion competency, and experimental investigations on emotion regulation, as well as those focusing on experimental sleep deprivation or examining sleep quality, were excluded.

- ***P****: The populations to which the topic is most relevant.*

**Inclusion:** The samples can be recruited from diverse populations based on health-related status, including individuals exposed to serious conditions like significant life stressors or psychiatric disorders, as well as those with no serious medical or psychological conditions. There are no restrictions on age range or gender. This broader inclusion is intended to examine quantitative distinction within specific populations and assess the moderating effects of demographic factors.

**Exclusion:** Studies focusing exclusively on populations with extremely specific or rare conditions that limit generalizability to broader populations or those that do not clearly define or report the health-related status of participants are ineligible for inclusion.

- ***I:*** *Intervention refers to the particular type of treatment provided to participants.*

**Inclusion:** This study does not concern the effect of treatments. However, interventional studies that report relevant baseline outcomes for the relationships between insomnia and emotion dysregulation are eligible for inclusion.

**Exclusion:** Studies focused solely on interventions without reporting baseline outcomes were excluded.

- ***C:*** *Comparison references the standard to which the experimental group is compared.*

**Inclusion:** For group comparison studies, the group of good sleepers must be recruited from the same population as the individuals with insomnia symptoms.

**Exclusion:** Studies comparing groups with varying levels of insomnia or different sleep disturbances/disorders without including a group of good sleepers were ineligible.

- ***S:*** *Study design refers to the method used to evaluate the phenomenon in question.*

**Inclusion:** Studies must be designed as an investigation involving a specific sample of participants and one or more predefined outcomes examining the association between insomnia and emotion dysregulation in the context of correlational or group comparison analyses.

**Exclusion:** animal studies, case reports, reviews, and meta-analyses were excluded.

### 2.4. Study selection

Given that a "study" might have multiple identified reports in the searched databases or that an identified "report" might contain multiple studies, we initially removed duplicated reports. Two authors (F.S. and N.M.) independently screened titles and abstracts, followed by full-text screening. The agreement rate was 96 % in the first stage and 84 % in the second stage, with Cohen's kappa values [41] of 0.89 and 0.71, respectively. Disagreements were resolved through discussion with the corresponding author (M.T.). Non-relevant literature was excluded at the initial screening stage, and eligible studies that fully met the selection criteria were included after full-text review (Fig. 2A). We contacted the authors twice for studies lacking data for effect size calculation. If there was no response from the authors, those studies were excluded.

### 2.5. Data extraction and study coding

We extracted the following data from each included study: *(i)* sample characteristics (population, sample size, mean age, and female/male ratio); *(ii)* methodological characteristics study design: longitudinal/cross-sectional and correlational/group comparison, measurement scales and reported outcomes); *(iii)* statistical measures necessary for calculating effect sizes. For correlational studies, we obtained the correlation coefficients (r), sample sizes, and effect direction. For group comparisons, we extracted the mean and standard deviation of emotion dysregulation measures and the sample size in both experimental and control groups (Fig. 2B- part 1).

Subsequently, we assessed the methodological quality of correlational studies using the appraisal tool for cross-sectional studies (AXIS) [42,43]. For the group comparison studies, we used the newcastle-ottawa scale (NOS) [44,45], recommended for observational case-control studies. Two authors assessed the quality of the studies (F.S. and N.M.), with disagreements resolved through discussion with the corresponding author (M.T.). According to AXIS, a study is considered "fully qualified" when it meets all the criteria for the introduction, methods, and results sections. Otherwise, it is rated as "partly qualified". The NOS quality score ranges from 0 to 7 (i.e., high quality (score $>6$), four of moderate quality ($3 <$ score $<6$), and three of low quality (score $<3$)). These features facilitate post-hoc observations and help evaluate

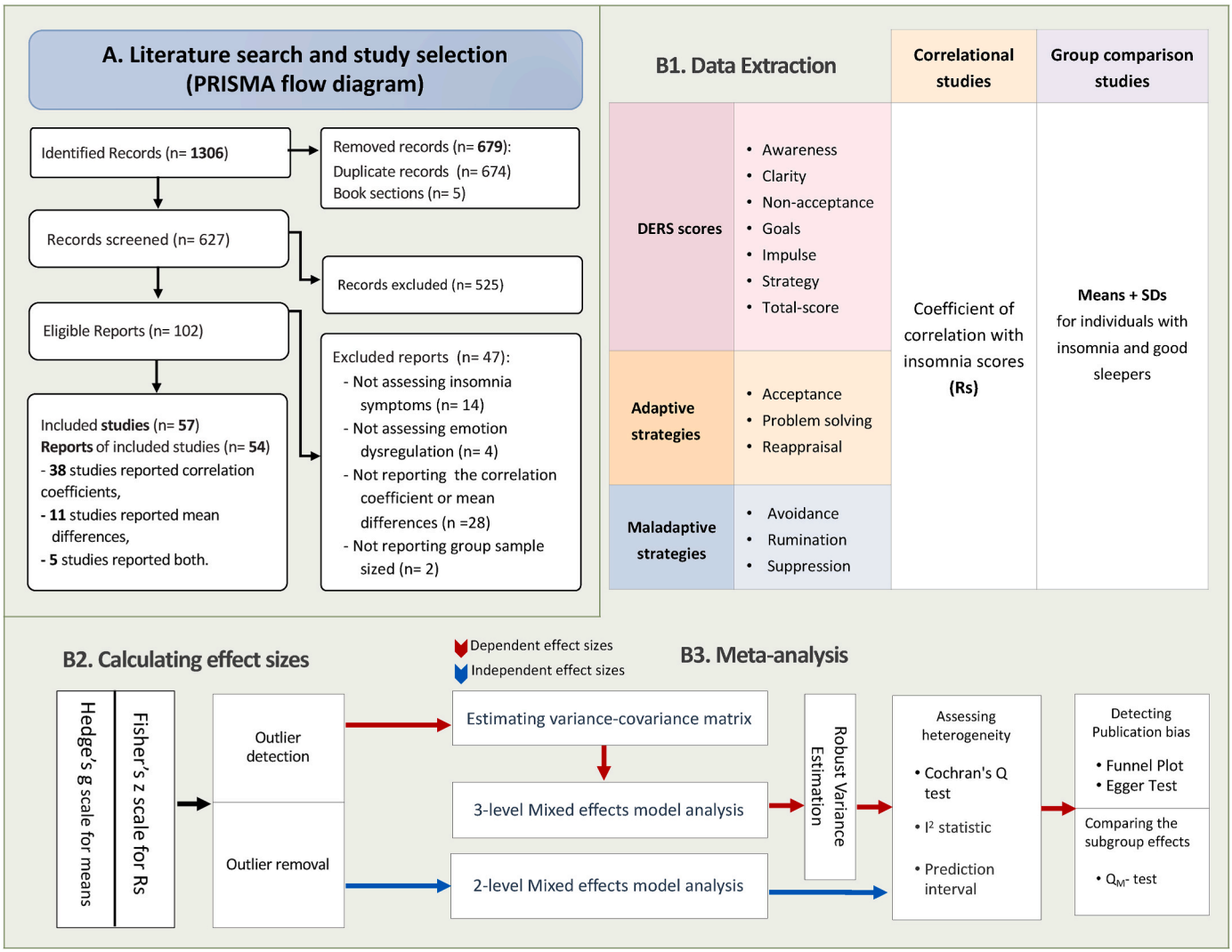


**Fig. 2.** PRISMA flow diagram of literature search and study selection (A), and details of the meta-analysis procedure (B)
**Note:** "Report" refers to any document, such as a journal article, conference abstract, unpublished manuscript, or thesis, providing relevant information. "Study" is an investigation with a defined group of participants and one or more outcomes and may have multiple reports. **Awareness:** lack of emotional awareness, **Clarity:** lack of emotional clarity, **DERS:** difficulty in emotion regulation scale, **Goals:** difficulties engaging in goal-directed behavior, **Impulse:** difficulties in impulse control, **non-acceptance:** non-acceptance of emotion, **Strategy:** inflexible strategy selection.

whether the inclusion of studies with lower rigor influences the conclusion of the meta-analysis.

### 2.6. Effect size calculation

For correlational studies, we used Fisher's z-transformation to stabilize variance and improve the accuracy of the pooled effect size [46]. For group comparison studies, we used the standardized mean difference to account for variations in measurement scales across studies. Specifically, we calculated Hedges' g, a bias-corrected version of Cohen's d, to adjust for potential overestimation in studies with small sample sizes, ensuring more reliable and comparable effect size estimates [47]. We also identified potential outliers and influential effect sizes that could notably influence the results of our analyses by evaluating Cook's distance values [48]. Effect sizes with a Cook's distance larger than 0.4 were flagged as outlier/influential cases.

### 2.7. Pooling effect sizes approach

*Given the varying* effect sizes *across studies and the absence of a singular true* effect size *for the association between insomnia and* emotion dysregulation*, a random-effects model should be used to estimate the mean of the true effect size distribution based on our data* [49]. *However,* many studies reported multiple outcomes for the same samples, resulting in a complex data structure that violates the assumption of outcome independence in the *random-effects model* [50]. To address the outcome interdependence issue, we applied an extended model of the traditional random-effects approach, allowing us to account for both within-study and between-study variance properly. Therefore, we first constructed an approximate variance-covariance matrix (V) for the dependent estimates (i.e., multiple effect sizes from the same samples). Then, we fit a three-level *random-effects model* to these effect sizes, using V as the variance-covariance matrix. In the fitted models, *the restricted maximum likelihood estimator* [51] *was used to calculate unbiased estimates of the variance of true* effect sizes ($\tau^2_{between\ study}$, $\tau^2_{within\ study}$). *Tests of the estimated coefficient and the corresponding confidence interval were based on t- and F-distributions.* In addition, recognizing that V is often just a rough approximation and that the random-effects structure may not fully capture all dependencies in the underlying true effects, we applied a cluster-robust inference method known as Robust Variance Estimation (RVE) to the fitted models. This method provided cluster-robust tests and confidence intervals [52], further enhancing the reliability of our

meta-analytic results (Fig. 2B- part 2,3). To assess whether three-level models provide a better fit to our data compared to two-level models, we employed akaike and bayesian information criteria (AIC, BIC), along with the likelihood ratio test (LRT) [49]. A lower AIC or BIC value and a significant result from the LRT indicate a superior fit of the model.

### 2.8. Heterogeneity detection

Heterogeneity, defined as the extent to which effect sizes vary within a meta-analysis, was assessed using Cochran's *Q* test [53]. This test evaluates whether the variability in the observed effect sizes is greater than what would be expected based on sampling error alone. Additionally, we calculated a multilevel version of the $I^2$ statistic, including $I^2_{between\ study}$ *and* $I^2_{within\ study}$. These statistics represent the proportion of variance in observed effect sizes not attributable to sampling error, thereby determining how much of the heterogeneity is due to within-study differences and how much is due to between-study differences. Cochran's Q and $I^2$ are influenced by the number and/or sample size of included studies, increasing with larger meta-analyses and sample sizes. To address these limitations, we also calculated 95 % prediction intervals (PIs) to provide a range into which we can expect the effects of future studies to fall based on the present evidence [54].

### 2.9. Publication bias detection

To address potential publication bias and enhance the validity of the meta-analysis results, we initially incorporated gray literature into our literature search (see above). This approach helps to prevent overestimation of actual effects [55]. We also evaluated the presence of publication bias using a contour-enhanced funnel plot that visually represented the symmetry of effect sizes. Subsequently, we conducted Egger's regression test to examine the symmetry in the funnel plot, statistically [56].

### 2.10. Meta-analysis procedure

We conducted separate analyses for correlational and group comparison studies. For each type of study, we categorized the effect sizes based on the individuals' with "no serious" and "serious health-related" conditions. We then pooled the effect sizes within each category using a three-level mixed-effects meta-analysis model, a *random-effects model* that considers the health-related status as a categorical moderator. To ensure that our findings were not affected by a small number of influential cases, we performed sensitivity analyses, checking how the pooled effect size estimates changed when potential outliers or influential cases were omitted. We also conducted moderating analyses, through (multiple) meta-regression models, on full or subsets of effect sizes to explore the impact of pre-specified moderating factors, their interactions, or linear combinations on the association between insomnia and emotion dysregulation. This included emotion dysregulation domains, mean age, and female/male ratio. For emotion dysregulation domains, we specified six sub-domains, including *(i)* Heightened use of maladaptive strategies (maladaptive), *(ii)* Reduced use of adaptive strategies (adaptive), *(iii)* lack of emotional functionality (function), *(iv)* deficiency in reducing the urgency of emotion (impulsivity), *(v)* inflexible situational-appropriate strategy selection (strategy), and *(vi)* the total scores of dispositional difficulties (DERS-Total). The omnibus test (QM-test) to test the null hypothesis that all the moderator effects are equal to zero was based on F-distribution with $m$ and $k-p$ degrees of freedom (i.e., with $m$ denoting the number of coefficients tested and $p$ the total number of model coefficients). We used a two-level mixed-effects meta-analysis model for subsets of independent effect sizes.

## 3. Results

### 3.1. Description of included studies

We identified 57 studies (Fig. 2A–Table S1 and S2), all with independent samples, meaning the participants in each study were independent and unrelated. The samples included individuals from various populations: those without serious health-related conditions, those with affective or other mental health issues, individuals who had experienced recent traumatic life events, and those coping with stress (Table 1). Among these studies, 38 reported correlation coefficients, reflecting the strength and direction of the relationship between insomnia and emotion dysregulation. Eleven studies reported mean differences, comparing emotion dysregulation between individuals with severe insomnia symptoms and those without. Five studies reported both correlation coefficients and mean differences.

We extracted 119 effect sizes based on correlation from 47 studies (38 studies reporting correlation coefficients + 5 reporting both correlation coefficients and mean differences) and 55 effect sizes based on group mean from 16 studies (11 studies reporting mean differences + 5 reporting both). Applying the AXIS tool, the correlational studies did not meet all the criteria for full qualification (Table S2). However, all were included in the analyses as they met the introduction criteria and demonstrated high or acceptable methodological quality. The NOS assessment of group comparison studies showed that nine were of high quality (score >6), four were of moderate quality (3 < score <6), and three were of low quality (score <3) (Table S3). A sensitivity analysis was conducted on the high and moderate-quality studies to assess the impact of low-quality studies on pooled effect sizes (see section b2 for details).

### 3.2. Meta-analysis findings of correlational studies

#### 3.2.1. Main analysis

We conducted a three-level mixed-effects meta-analysis to pool 119 effect sizes (Fisher's z) from correlational studies, incorporating individuals' health-related status as a moderator. We selected the three-level model as it provided a significantly better fit compared to two-level models, where between-study or within-study heterogeneity was constrained to zero (Table S4A). The pooled effect sizes for both groups of individuals were significant ($p < 0.00001$). The effect size for individuals without serious conditions (Fisher's $z = 0.22$) was smaller than for those with serious conditions (Fisher's $z = 0.37$). The results showed heterogeneity, with variance components of $\tau^2_{between} = 0.016$ and $\tau^2_{within} = 0.05$, indicating that 24.17 % of the total variation was due to between-study heterogeneity and 73.75 % to within-study heterogeneity (Table 2A, Fig. 3A). The QM-test ($p < 0.0001$) indicated that health-related status significantly influences the correlation between insomnia and emotion dysregulation (Table 2A, Fig. 4A).

#### 3.2.2. Outliers and sensitivity analysis

One outlier/influential case [57], with Fisher's $z = -0.09$, was identified among the correlational studies on samples with serious conditions (Fig. S1). However, a sensitivity analysis indicated that removing this study did not notably affect the pooled estimated correlation between insomnia and emotion dysregulation and did not reduce the heterogeneity (Table 2B). Therefore, we did not exclude it in the following analyses.

#### 3.2.3. Meta-regression/Subgroup analyses

*3.2.3.1. Mean age and female/male ratio.* Adding mean age or female/male ratio to our main meta-analysis model revealed that neither mean age, nor female/male ratio affected the relationship between insomnia and emotion dysregulation (p-values >0.1) (Table 2C, Fig. 5A). While

**Table 1**
Description of 57 included studies

| Study | Health-related status | | N | K | Range of mean-age | Range of sample-size |
|---|---|---|---|---|---|---|
| Correlational | no serious condition | – | 26 | 69 | 14.48–71 | 46–10148 |
| | serious condition | Affective issues | 5 | 8 | 18.33–48.4 | 40–880 |
| | | Trauma or major life event | 7 | 25 | | |
| | | Experiencing coping stress | 5 | 12 | | |
| | | others | 4 | 5 | | |
| Group Comparison | no serious condition | – | 12 | 42 | 26–50 | 39–653 |
| | serious condition | Affective issues | 1 | 7 | 38.23–48.9 | 40–255 |
| | | Trauma or major life event | 1 | 4 | | |
| | | Experiencing coping stress | 0 | 0 | | |
| | | others | 2 | 2 | | |

**K:** Number of effect sizes, **N:** number of studies.

the majority of studies focused on adults, with age ranges spanning from young to older adulthood, three correlational studies had a mean participant age below 17 and were categorized as adolescent samples. To assess the impact of age-related variability, we conducted two analyses: one including all studies (from adolescence to older adulthood) and another excluding the adolescent studies. Results were consistent across both models, indicating that the association between insomnia and emotion dysregulation is stable across age groups and not moderated by age.

*3.2.3.2. Individuals' health-related condition × emotion dysregulation domains.* Considering the interaction between the individuals' health-related condition and various domains of emotion dysregulation as a moderator in a mixed-effects meta-analysis model, we found that the difference between estimated effect sizes for individuals with and without serious conditions varied across different domains of emotion dysregulation. This difference was only for two domains: impulsivity and maladaptive strategies (p-values <0.05). Moreover, the main effect of health-related status was not significant, while the main effect of emotion dysregulation domains, except for impulsivity, was significant (Table 2C). The result of the QM-test indicated that this interaction significantly affects the link between insomnia and emotion dysregulation (p-value <0.05). Moreover, incorporating this interaction as a moderator in the meta-analysis model significantly improved the model fit compared to the main model. (Table S4A).

### 3.2.4. Focal analyses on a subset of correlational studies

*3.2.4.1. Serious health-related conditions.* Pooling the subset of effect sizes for individuals with various health-related conditions, using a three-level mixed-effects meta-analysis model with the condition as a moderator, revealed that the type of serious health-related condition significantly moderated the association between insomnia and emotion dysregulation. Our findings indicated significant estimated effect sizes for samples with affective issues, those who have experienced recent traumatic life events, and individuals dealing with coping stress, but not for individuals with other mental health issues. The strongest association was observed in the subgroup of individuals with affective symptoms (Table 2D, Fig. 4A).

*3.2.4.2. Specific emotion dysregulation domains.* Utilizing a two-level mixed-effects meta-analysis model on distinct subsets of effect sizes for three domains, including lack of emotional functionality as well as adaptive and maladaptive emotion regulation strategies, we found that the type of emotional function (i.e., awareness, clarity, and acceptance) and the type of strategies can moderate the link between insomnia and the specific domain of emotion dysregulation ($p\text{-value}_{QM} < 0.0001$). Regarding emotional functionality, insomnia was associated with clarity (p-value <0.01) and non-acceptance (p-value <0.001) but not with awareness. Among problem-solving, acceptance, and reappraisal, insomnia had a negative correlation with reappraisal (p-value <0.05). Furthermore, rumination (p-value <0.00001) and suppression (p-value <0.01) were both associated with insomnia (Table 2D, Fig. 4A). There were insufficient effect sizes for avoidance.

## 3.3. Meta-analyses findings on group comparison studies

### 3.3.1. Main analysis

We pooled 55 effect sizes (Hedges' g) from 16 group comparison studies using a three-level mixed-effects meta-analysis model, with individuals' health-related status included as a moderator. In the subgroup of individuals without serious conditions, those with insomnia exhibited more emotion dysregulation problems than good sleepers (Hedges' g = 1.26, p-value <0.05). However, among individuals with serious conditions, there was no significant difference between the two groups (p-value = 0.65). Of note, the number of studies for the subgroup with serious conditions was limited (N = 4, number of effect sizes = 13). The results displayed heterogeneity, with estimated variance components of $\tau^2_{between} = 1.51$ and $\tau^2_{within} = 0.38$, indicating that 78.4 % of the total variation could be attributed to between-study heterogeneity and 20.12 % to within-study heterogeneity (Table 3A, Fig. 3B). The non-significant result of the QM-test (p-value = 0.07) indicated that the health-related status of samples could not influence the pooled estimated effect of insomnia on emotion dysregulation (Table 3A, Fig. 4B). The three-level model pooling approach provided a significantly better fit than two-level models, where between-study or within-study heterogeneity was constrained to zero (Table S4B). However, the mixed-effects meta-analysis model did not provide a better fit compared to the *random-effects model* (Table S4B) that estimates a significant overall association between insomnia and emotion dysregulation (Hedges' g = 0.99, p-value <0.05) (Table 3A).

### 3.3.2. Outliers and sensitivity analysis

We identified one outlier/influential case [58] with the Hedges' g = 4.86 among studies on samples without serious conditions (Fig. S1). Sensitivity analysis demonstrated that removing this outlier reduced the magnitude of estimated Hedges' g for this subgroup of individuals. In addition, a part of the observed between- or within-study heterogeneity can be attributed to this outlier (Table 3B). Furthermore, an additional sensitivity analysis excluding the three identified low-quality studies indicated that the inclusion of those studies also contributes to the observed heterogeneity between or within studies (Table 3B). Given the results of the main and sensitivity analyses, we excluded the identified outlier and low-quality studies and proceeded with additional moderating analyses irrespective of individuals' health-related status.

### 3.3.3. Meta-regression/subgroup analyses

*3.3.3.1. Mean age and female/male ratio.* In a three-level random-effect meta-regression, we explored the impact of adding mean age or female/male ratio as moderators. The findings indicated that both mean age and female/male ratio did not have a moderating effect (p-values >0.4)

**Table 2**
Statistics for meta-analysis of correlational studies

| Subset of effect sizes | Moderators | | | n | k | PE | SE | [95 % CI] | p-value | [95 % PI] | Q | $I^2$ | | $\tau^2$ | | $Q_m$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | $I^2_{btn}$ | $I^2_{wtn}$ | $\tau^2_{btn}$ | $\tau^2_{wtn}$ | |
| **A. Main analysis** | | | | | | | | | | | | | | | | |
| all | serious condition | No | | 26 | 69 | 0.22 | 0.04 | [0.14, 0.29] | <0.00001 | [-0.33, 0.76] | 5276.34 | 24.17 | 73.75 | 0.016 | 0.05 | 37.35* |
| | | Yes | | 20 | 50 | 0.37 | 0.06 | [0.25, 0.49] | <0.00001 | [-0.19, 0.94] | | | | | | |
| | – | | | 46 | 119 | 0.28 | 0.03 | [0.21, 0.35] | <0.001 | [-0.26, 0.83] | 5572.57 | 23.67 | 72.34 | 0.02 | 0.05 | – |
| **B. Sensitivity analysis** | | | | | | | | | | | | | | | | |
| Without outlier[a] | serious condition | No | | 26 | 69 | 0.22 | 0.05 | [0.14, 0.29] | <0.00001 | [-0.30, 0.69] | 5084.62 | 17.51 | 80.12 | 0.010 | 0.047 | 50.93* |
| | | Yes | | 19 | 47 | 0.41 | 0.05 | [0.30, 0.51] | <0.00001 | [-0.12, 0.93] | | | | | | |
| **C. Multiple meta-regression analyses** | | | | | | | | | | | | | | | | |
| all | serious condition + mean age | No | | 46 | 119 | 0.20 | 0.07 | [0.05, 0.35] | 0.01 | [-0.34, 0.74] | | | | | | |
| | OR female/male ratio | Yes | | | | 0.32 | 0.08 | [0.16, 0.48] | <0.001 | [-0.24, 0.88] | 5122.23 | 21.59 | 76.15 | 0.0137 | 0.049 | 24.93* |
| | | Mean age | | | | 0.001 | 0.002 | [-0.00, 0.01] | 0.77 | [-0.54, 0.54] | | | | | | |
| | | No | | | | 0.24 | 0.09 | [0.03, 0.45] | 0.02 | [-0.39, 0.86] | | | | | | |
| | | Yes | | | | 0.36 | 0.08 | [0.17, 0.55] | <0.01 | [-0.25, 0.97] | 5180.70 | 21.47 | 76.34 | 0.014 | 0.05 | 22.22* |
| | | Female/male ratio | | | | −0.034 | 0.13 | [-0.32 0.25] | 0.80 | [-0.66, 0.60] | | | | | | |
| | serious condition × domains of emotion dysregulation | Main effect | No | | | 0.01 | 0.06 | [-0.14, 0.17] | 0.80 | [-0.51, 0.55] | 4739.82 | 11.58 | 85.25 | 0.005 | 0.04 | 39.35· |
| | | | Yes | | | −0.07 | 0.06 | [-0.27, 0.11] | 0.31 | [-0.73, 0.57] | | | | | | |
| | | Main effect | DERS-Total | | | 0.38 | 0.07 | [0.22, 0.54] | <0.001 | [-0.10, 0.86] | | | | | | |
| | | | Function | | | 0.21 | 0.08 | [0.03, 0.40] | 0.02 | [-0.33, 0.76] | | | | | | |
| | | | Impulsivity | | | 0.20 | 0.10 | [-0.05, 0.45] | 0.10 | [-0.35, 0.75] | | | | | | |
| | | | Strategy | | | 0.273 | 0.123 | [-0.01, 0.55] | 0.05 | [-0.28, 0.83] | | | | | | |
| | | | Maladaptive | | | 0.25 | 0.08 | [0.06, 0.44] | 0.01 | [-0.26, 0.77] | | | | | | |
| | | Interaction effect | Yes × DERS-TOTAL | | | 0.20 | 0.10 | [-0.02, 0.43] | 0.06 | [-0.29, 0.70] | | | | | | |
| | | | Yes × Function | | | 0.19 | 0.12 | [-0.09, 0.47] | 0.15 | [-0.35, 0.73] | | | | | | |
| | | | Yes × Impulsivity | | | 0.32 | 0.14 | [0.01, 0.63] | 0.04 | [-0.23, 0.88] | | | | | | |
| | | | Yes × Strategy | | | 0.31 | 0.15 | [-0.04, 0.67] | 0.07 | [-0.29, 0.92] | | | | | | |
| | | | Yes × Maladaptive | | | 0.31 | 0.11 | [0.05, 0.57] | 0.02 | [-0.22, 0.85] | | | | | | |
| Mean age >17 | serious condition + mean age | No | | 44 | 116 | 0.26 | 0.09 | [0.07, 0.44] | <0.01 | [-0.31, 0.82] | | | | | | |

(*continued on next page*)

**Table 2** (*continued*)

| Subset of effect sizes | Moderators | | n | k | PE | SE | [95 % CI] | p-value | [95 % PI] | Q | $I^2$ | | $\tau^2$ | | $Q_m$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | $I^2_{btn}$ | $I^2_{wtn}$ | $\tau^2_{btn}$ | $\tau^2_{wtn}$ | |
| | | Yes | | | 0.36 | 0.08 | [0.18, 0.54] | <0.001 | [-0.22, 0.94] | 3311.93 | 20.23 | 75.31 | 0.014 | 0.052 | 25.35* |
| | | Mean age | | | −0.001 | 0.03 | [-0.01, 0.01] | 0.84 | [-0.55, 0.55] | | | | | | |
| **D. Focal analyses of subsets of effect sizes** | | | | | | | | | | | | | | | |
| serious conditions | Affective | | | | 0.48 | 0.19 | [-0.14, 1.12] | 0.04 | [-0.72, 1.69] | 812.92 | 42.75 | 53.55 | 0.043 | 0.055 | 6.01 |
| | Trauma | | | | 0.30 | 0.09 | [0.07, 0.55] | 0.02 | [-0.55, 1.16] | | | | | | |
| | Coping stress | | | | 0.38 | 0.10 | [0.11, 0.65] | 0.01 | [-0.45, 1.21] | | | | | | |
| | Others | | | | 0.35 | 0.16 | [-0.16, 0.87] | 0.11 | [-0.76, 1.47] | | | | | | |
| difficulty in function domains | Awareness | | 7 | 19 | 0.15 | 0.06 | [-0.03, 0.33] | 0.08 | [-0.28, 0.58] | 141.98 | 87.5564 | | 0.0226 | | 34.50 |
| | Clarity | | | | 0.36 | 0.08 | [0.15, 0.58] | <0.01 | [-0.08, 0.81] | | | | | | |
| | Non-acceptance | | | | 0.39 | 0.04 | [0.28, 0.50] | <0.001 | [0.01, 0.78] | | | | | | |
| adaptive strategies | Problem solving | | 13 | 25 | −0.002 | 0.06 | [-0.14, 0.14] | 0.97 | [-0.61, 0.61] | 595.23 | 96.7453 | | 0.0659 | | 3.19· |
| | Reappraisal | | | | −0.214 | 0.065 | [-0.4, −0.06] | 0.01 | [-0.83, 0.40] | | | | | | |
| | Acceptance | | | | 0.217 | 0.155 | [-0.16, 0.60] | 0.21 | [-0.52, 0.95] | | | | | | |
| maladaptive strategies | Maladaptive + health-related condition | Rumination | 27 | 34 | 0.30 | 0.03 | [0.24, 0.37] | <0.00001 | [-0.08, 0.70] | 293.77 | 93.0556 | | 0.0316 | | 39.80· |
| | | Suppression | | | 0.18 | 0.03 | [0.09, 0.27] | <0.01 | [-0.27, 0.63] | | | | | | |
| | | Health-related condition | | | 0.18 | 0.08 | [0.00, 0.36] | 0.04 | [-0.23, 0.60] | | | | | | |

**Awareness:** lack of emotional awareness, **btn:** between study, **Clarity:** lack of emotional clarity**, CI:** confidence interval, **DERS-Total:** total score of difficulty in emotion regulation scale (DERS), **Function**: lack of emotional functionality**, Impulsivity:** deficiency in reducing the urgency of emotion, **K:** number of effect sizes, **Maladaptive:** Heightened use of maladaptive strategies**, n**: number of studies, **Non-acceptance:** non-acceptance of emotion, **PE:** pooled effect size estimate, **PI:** predict interval, **Q:** Q-value of cochran's Q test, **SE:** standard error, **Strategy:** inflexible strategy selection, **wtn:** within study.
p-value $\leq$ 0.05* p-value <0.0001, ** p-value <0.00001.
[a] **Outliers/influential cases:** Lee et al., 2019.

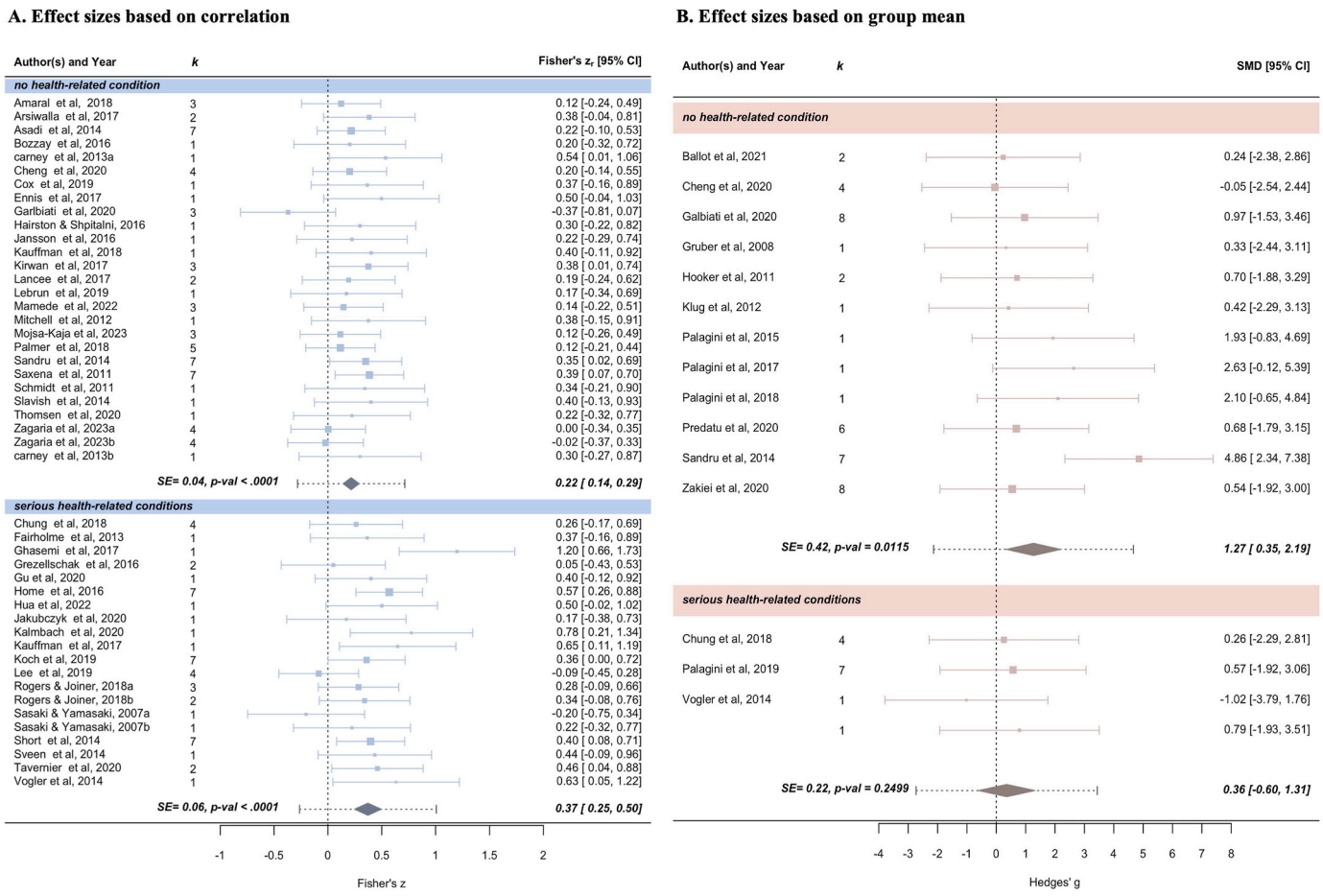


**Fig. 3.** Forest plots displaying aggregated effect sizes calculated from each correlational (A) and group comparison (B) studies
**Note: K:** number of effect sizes, **SE:** standard error.

(Table 3C, Fig. 5B).

*3.3.3.2. Emotion dysregulation domain.* Including the emotion dysregulation domain as a moderator in a three-level mixed-effects meta-analysis model demonstrated that different domains can moderate how emotion dysregulation problems for individuals with insomnia differ from those of good sleepers (p-value$_{QM}$ < 0.0001). While individuals with insomnia suffered from impulsivity, limited access to appropriate strategies, and operated more maladaptive strategies, compared to good sleepers (p-values <0.01), there were no differences in inability to use adaptive emotion regulation strategies and in lack of emotional functionality (p-values >0.1) (Table 3C, Fig. 4B). There were insufficient number of studies to evaluate whether and how insomnia affects the lack of different emotional functionality as well as different types of strategies.

### 3.4. Publication bias

The Egger's regression test showed no asymmetry in the funnel plot of correlational studies, suggesting no evidence of publication bias (p-value = 0.36) (Fig. 6A). There was an asymmetry in the funnel plot of group comparison studies (p-value <0.001) (Fig. 6B). However, the detected asymmetry was caused by the recognized outlier for group comparison studies, and after outlier removal, the result of egger's regression test was not significant (p-value $_{\text{without outlier}}$ = 0.14) (Fig. 6C).

## 4. Discussion

In this large-scale meta-analysis, we synthesized 174 effect sizes from 57 studies using correlational or group comparison designs to examine the relationship between insomnia and emotion dysregulation. Emotion dysregulation was investigated within a framework that integrates both emotion regulation strategies and dispositional difficulties. We conducted separate meta-analyses for correlational and group comparison studies. In correlational studies, we quantified the strength of the association between insomnia symptoms and emotion dysregulation. In group comparison studies, we examined whether individuals with clinically significant insomnia symptoms exhibit greater emotion dysregulation. Within these two sets of analyses, we explored how various domains of emotion dysregulation, individuals' health-related status, their potential interactions, as well as age, and gender influenced the relationship between insomnia and emotion dysregulation.

### 4.1. Neurobiological underpinnings of the insomnia-emotion dysregulation association

Our meta-analyses revealed a significant positive association between insomnia symptoms and emotion dysregulation, and showed that individuals with clinically significant insomnia symptoms exhibited higher levels of emotion dysregulation compared to good sleepers. The hyperarousal theory of insomnia offers a useful framework for understanding these findings. According to this theory, individuals with insomnia symptoms/disorder experience elevated emotional arousal, which occurs both during the day and at night [3,59,60]. From a neurobiological perspective, hyperarousal in insomnia is thought to stem from an imbalance between overactive excitatory circuits and weakened inhibitory control mechanisms. Disruptions in cortical–subcortical circuits, which regulate both emotion and sleep [8,9]

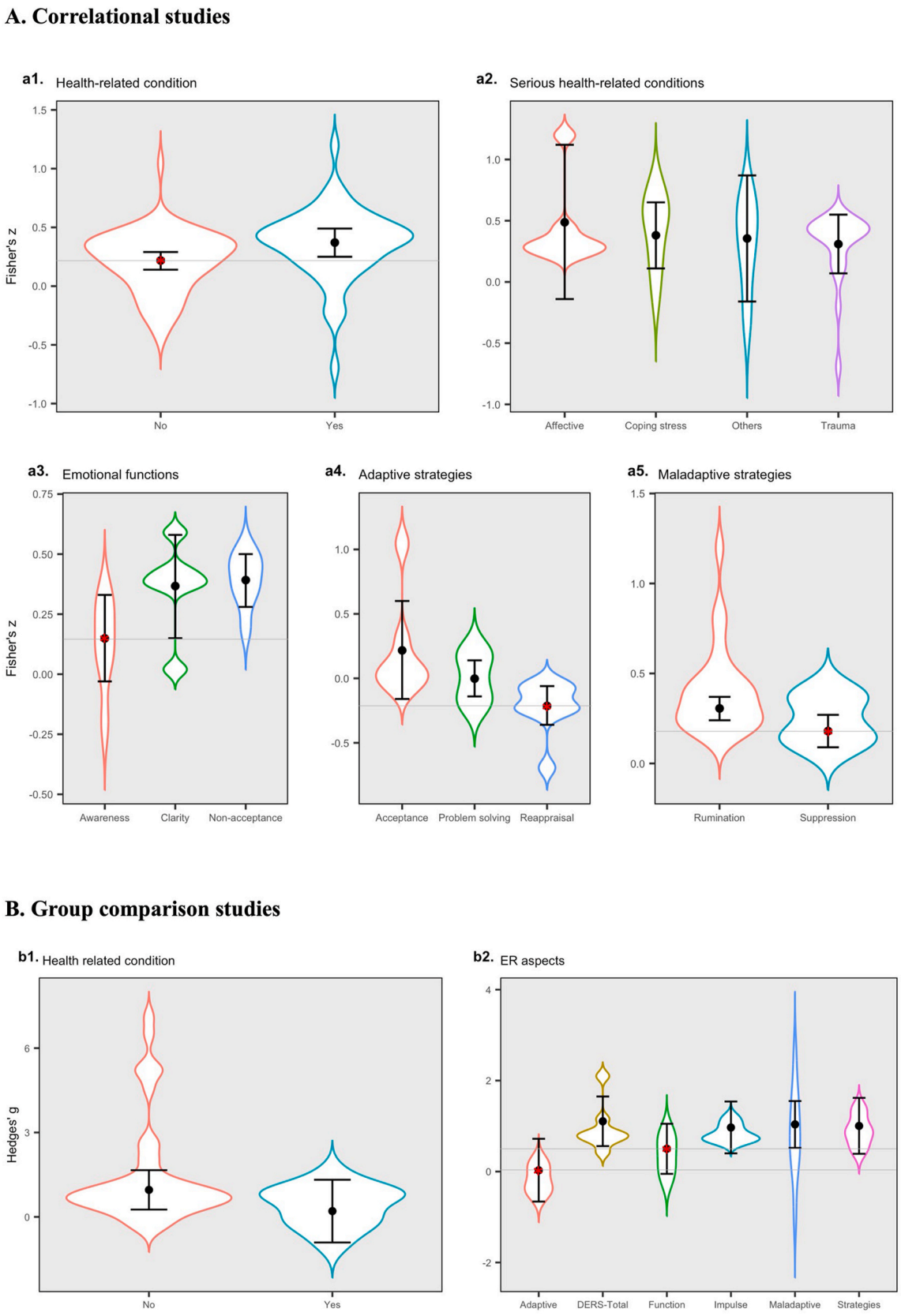


**Fig. 4.** Distribution of effect sizes of correlational (A) and group comparison (B) studies
**Note:** The effect size means that falls outside the confidence intervals differ significantly from others. **Awareness:** lack of emotional awareness, **Adaptive:** Reduced use of maladaptive strategies**, Clarity:** lack of emotional clarity**, DERS-Total:** total score of difficulty in emotion regulation scale (DERS), **Function**: lack of emotional functionality**, Impulsivity:** deficiency in reducing the urgency of emotion, **Maladaptive:** Heightened use of maladaptive strategies**, non-acceptance:** non-acceptance of emotion, **Strategy:** inflexible strategy selection.

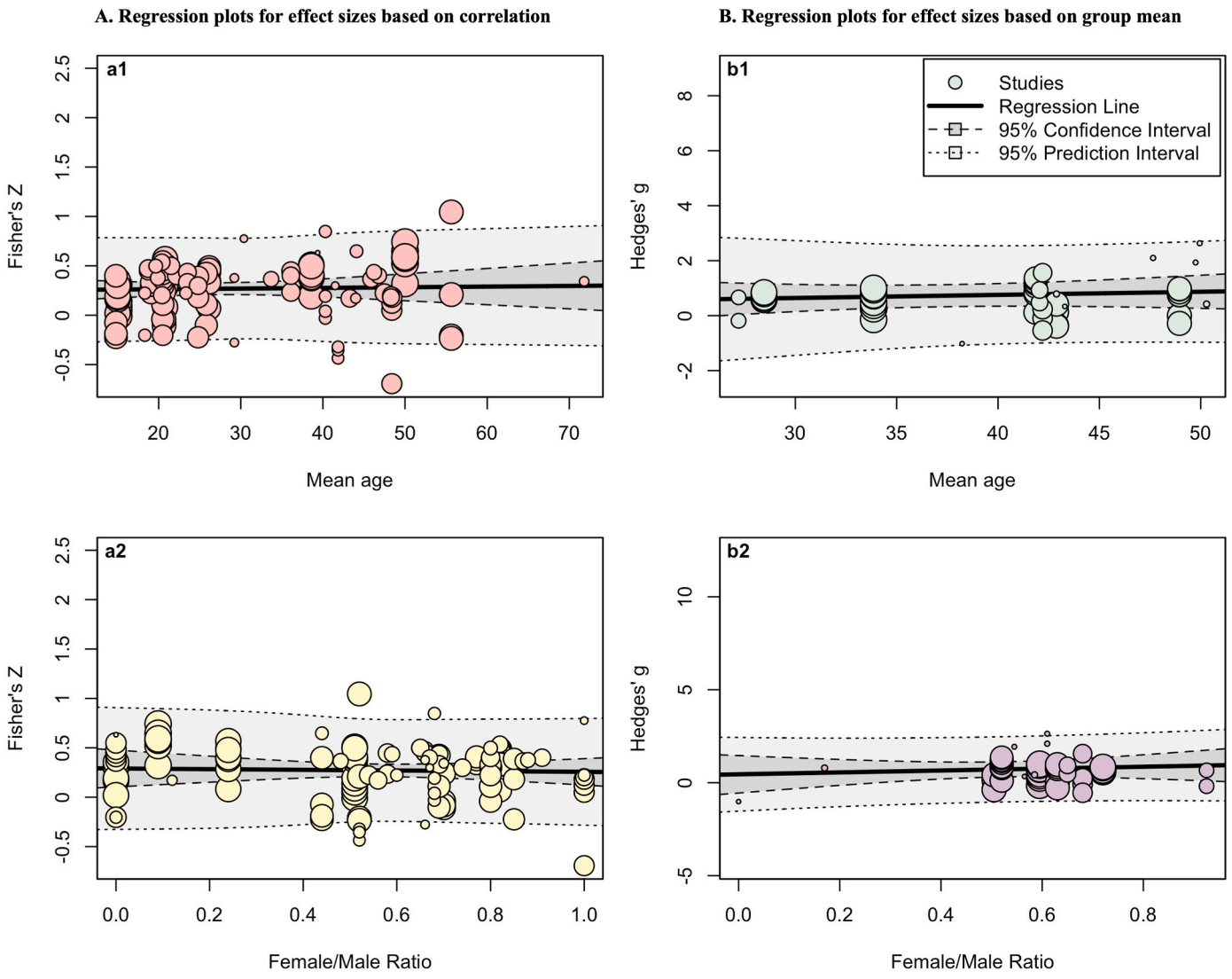


**Fig. 5.** Meta-regression plots displaying the relationship between effect sizes and mean age/female-to-male ratio for each (A) correlational or (B) group comparison study.

may weaken this balance, thereby contributing to the hyperarousal. In particular, abnormalities in the prefrontal cortex (PFC), which is crucial for the top-down regulation of subcortical limbic regions such as the amygdala, as well as salience-processing areas like the insula and subgenual anterior cingulate cortex (sgACC) [3,61,62] can lead to increased activation in these emotion-generating systems [13,63–67]. The amygdala hyperreactivity contributes to the manifestation of emotional distress and interferes with sleep initiation and maintenance. In addition, heightened activity in the insula and sgACC may amplify emotional salience and interfere with interoceptive processing, thereby further exacerbating hyperarousal [69]. The insula, in particular, plays a central role in integrating interoceptive signals with emotional experiences, potentially amplifying internal sensations of discomfort or threat. This heightened interoceptive sensitivity may sustain internal arousal, contributing to the chronic hyperarousal state of insomnia. Increased sgACC activation, a key node mediating PFC–amygdala communication [68], is associated with exaggerated emotional salience attribution and heightened autonomic arousal, which may compromise the PFC's capacity to downregulate limbic activity. This impaired regulatory loop may disrupt emotional homeostasis and further intensify the emotional and physiological arousal characteristic of insomnia. Supporting this view, our recent neuroimaging meta-analyses confirmed the involvement of both the amygdala and sgACC across insomnia and other sleep disorders [66,69].

Beyond top-down regulation, the association between insomnia and emotion dysregulation may also reflect impairments in basic, evolutionarily conserved emotional processes such as habituation, sensitization, Pavlovian conditioning, and extinction. These basic mechanisms automatically calibrate emotional responses based on prior experiences and are critical for emotional adaptation [70]. When disrupted, they can amplify amygdala reactivity and delay emotional recovery, especially in the context of compromised cognitive control [70]. Sleep, and particularly REM sleep, plays a key role in supporting these bottom-up regulatory processes. Poor sleep may impair the consolidation of emotional memories within these systems [71], potentially through alterations in REM sleep architecture [10,11]. While REM sleep seems to facilitate the integration and extinction of emotional memories in healthy individuals, it may instead strengthen the consolidation of stress-related memories in patients with insomnia disorder [72,73]. Thus, the interplay of weakened top-down control and disrupted bottom-up regulatory mechanisms may jointly explain the robust and pervasive association between insomnia symptoms and emotion dysregulation.

### 4.2. *Specific emotion dysregulation domains*

We found that insomnia symptoms are more strongly associated with

**Table 3**
Statistics for meta-analysis of group comparison studies

| Subset of effect sizes | Moderators | | n | k | PE | SE | [95 % CI] | p-value | [95 % PI] | Q | I2 | | τ2 | | Qm |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | $I^2_{btn}$ | $I^2_{wtn}$ | $\tau^2_{btn}$ | $\tau^2_{wtn}$ | |
| **A. Main analysis** | | | | | | | | | | | | | | | |
| all | health-related condition | No | 12 | 42 | 1.265 | 0.420 | [0.34, 2.19] | 0.01 | [-1.91, 4.44] | 968.89* | 78.4 | 20.12 | 1.51 | 0.38 | 4.00 |
| | | Yes | 4 | 13 | 0.184 | 0.369 | [-1.00, 1.36] | 0.65 | [-4.38, 4.75] | | | | | | |
| | – | | 16 | 55 | 0.99 | 0.34 | [0.26, 1.72] | 0.01 | [-2.10, 410] | 969.19 | 78.04 | 19.83 | 1.60 | 0.38 | – |
| **B. Sensitivity analysis** | | | | | | | | | | | | | | | |
| Without outlier[a] | health-related condition | No | 11 | 39 | 0.886 | 0.239 | [0.35, 1.42] | 0.004 | [-0.98, 2.75] | 692.67* | 68.92 | 27.21 | 0.45 | 0.18 | 6.01 |
| | | Yes | 4 | 13 | 0.200 | 0.351 | [-0.93, 1.33] | 0.60 | [-2.61, 3.01] | | | | | | |
| Without low quality studies | health-related condition | No | 8 | 21 | 0.960 | 0.304 | [0.26, 1.66] | 0.013 | [-1.20, 3.12] | 552.13* | 64.64 | 31.25 | 0.52 | 0.25 | 4.35 |
| | | Yes | 4 | 13 | 0.206 | 0.345 | [-0.91, 1.32] | 0.59 | [-2.85, 3.26] | | | | | | |
| **C. Meta-regression analyses** | | | | | | | | | | | | | | | |
| Without outlier | Mean age + Female/male ratio | Mean age | 13 | 48 | 0.01 | 0.014 | [-0.02, 0.05] | 0.48 | [-3.11, 3.16] | 681.7* | 80.59 | 18.08 | 0.44 | 0.18 | 7.07 |
| | | Female/male Ratio | | | 0.50 | 0.913 | [-2.24, 3.26] | 0.61 | [-1.39, 5.04] | | | | | | |
| | domains of emotion dysregulation | DERS-Total | | | 0.946 | 0.206 | [0.48, 1.41] | 0.0011 | [-0.87, 2.76] | 367.1* | 82.37 | 13.6 | 0.52 | 0.08 | 7.72* |
| | | Function | | | 0.343 | 0.208 | [-0.14, 0.83] | 0.141 | [-1.55, 2.24] | | | | | | |
| | | Impulsivity | | | 0.811 | 0.183 | [0.38, 1.24] | 0.003 | [-1.09, 2.71] | | | | | | |
| | | Strategy | | | 0.846 | 0.241 | [0.26, 1.43] | 0.012 | [-1.15, 2.84] | | | | | | |
| | | Adaptive | | | −0.151 | 0.412 | [-1.71, 1.41] | 0.744 | [-3.51, 3.20] | | | | | | |
| | | Maladaptive | | | 0.884 | 0.282 | [0.27, 1.50] | 0.008 | [-0.93, 2.70] | | | | | | |

**btn:** between study, **CI:** confidence interval, **DERS-Total:** total score of difficulty in emotion regulation scale (DERS), **Function**: lack of emotional functionality, **Impulsivity:** deficiency in reducing the urgency of emotion, **K:** number of effect sizes, **Maladaptive:** Heightened use of maladaptive strategies, **n**: number of studies, **PE:** pooled effect size estimate, **PI:** predict interval, **Q:** Q-value of cochran's Q test, **SE:** standard error, **Strategy:** inflexible strategy selection, **wtn:** within study.
* p-value <0.05.
[a] **Outliers/influential cases:** Sandru et al., 2014.

maladaptive emotion regulation strategies than with adaptive ones. Individuals with clinically significant insomnia tend to rely more on maladaptive strategies, while showing comparatively fewer difficulties in using adaptive strategies than good sleepers. Insomnia was also broadly associated with greater dysregulation across several dispositional domains; however, its link to emotional functionality was relatively weak and, in some analyses, non-significant. This pattern may be explained by higher emotional intensity, driven by hyperarousal in individuals with insomnia, which predisposes them to impulsivity, reduces their ability to use appropriate strategies, and shifts their emotion regulation toward maladaptive methods. This tendency is significant because adaptive regulation strategies, while effective in mild to moderate emotional distress, tend to be less effective when emotions are more intense [74]. Emotional functionality may also require emotional stability, which is difficult to achieve in the context of chronic hyperarousal and intense distress related to severe insomnia symptoms.

Although insomnia was not strongly related to emotional functionality or adaptive strategy use in general, these associations varied depending on the specific emotional function (e.g., awareness, clarity, acceptance) or strategy (e.g., reappraisal, problem-solving, acceptance). Our findings suggest that individuals with insomnia are typically aware of their emotional states but struggle with clarity and acceptance. They also have difficulty using reappraisal and tend to underutilize other adaptive strategies such as problem-solving and acceptance. This pattern indicates that while they can identify their emotions, they often struggle to manage or reinterpret them, especially under heightened emotional arousal. Consequently, they are more likely to rely on maladaptive strategies like rumination and suppression, commonly triggered in high-stress situations [74]. Neorobiologically, cognitive adaptive strategies, in particular the reappraisal, depends on effective recruitment of the PFC to downregulate activation in emotion-generating regions such as the amygdala and insula [21,25,75]. Insufficient inhibition or amplified amygdala reactivity may disturb the implementation of these strategies [25]. Further, maladaptive strategies such as suppression and rumination can increase activity in the amygdala and default mode network [75,76,77], potentially reinforcing emotional rigidity. This impact may be particularly pronounced under conditions of high arousal, where adaptive strategies are less effective, and maladaptive responses tend to dominate.

### *4.3. Health-related status and the insomnia-emotion dysregulation association*

The overall association between insomnia symptoms and emotion dysregulation was stronger among individuals with serious health-related conditions than those without. However, this amplifying effect was domain-specific, intensifing the link with impulsivity and maladaptive strategies in particular. This aligns with our prior findings that insomnia is more strongly associated with maladaptive than adaptive strategies. This pattern supports the notion that individuals with serious health-related conditions like those with insomnia disorder tend to rely on less effective means of managing emotional distress. Moreover, in individuals with clinically significant insomnia, the presence of additional health-related conditions did not further increase levels of

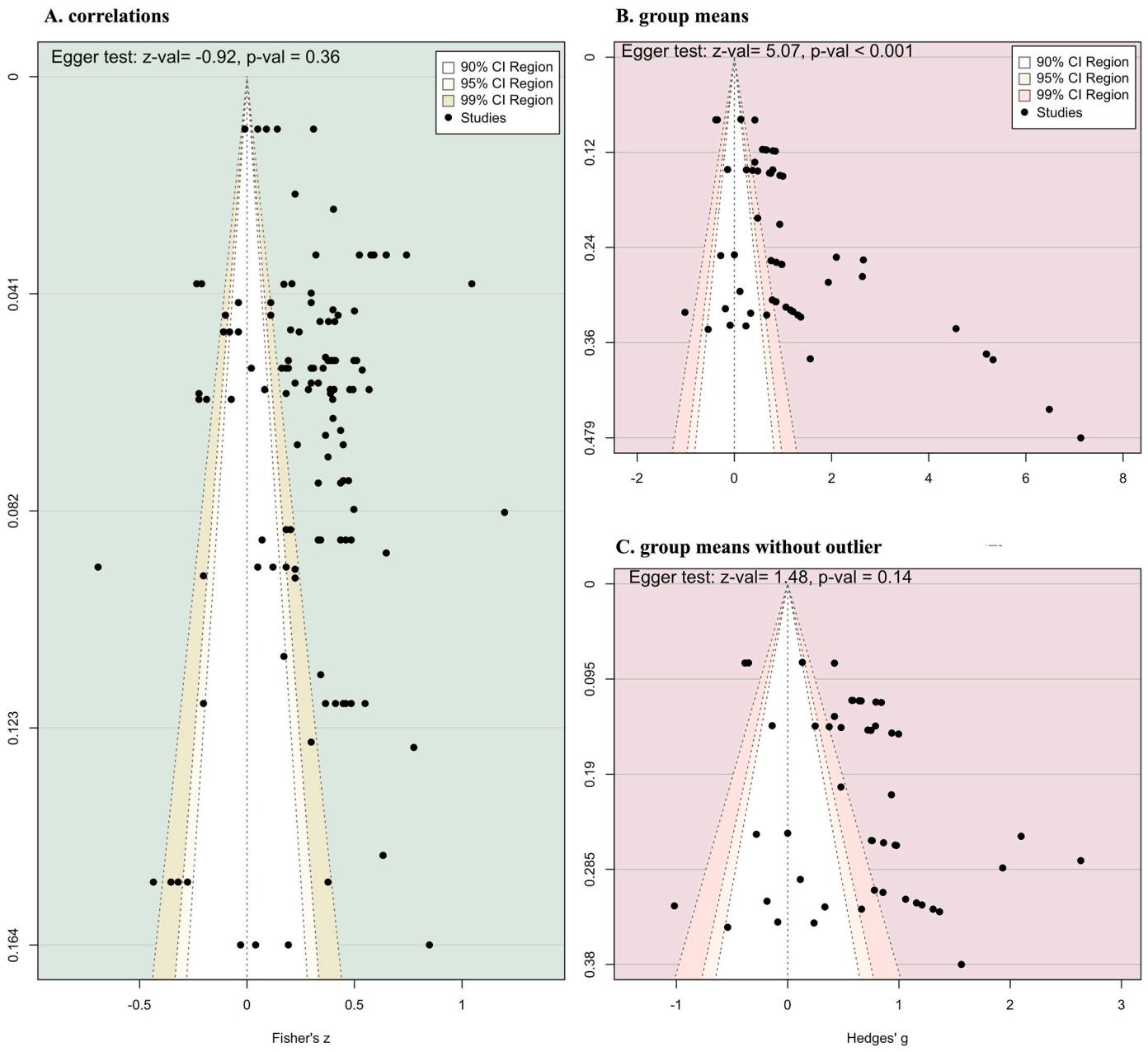


**Fig. 6.** Contour-enhanced funnel plots displaying all effect sizes from (A) correlational and (B) group comparison studies.

emotion dysregulation. This may be due to the severity of insomnia symptoms, which already represent a state of persistent hyperarousal [78]. It suggests that the chronic hyperarousal due to insomnia, could already impair the neural circuits involved in emotion and arousal regulation, leaving limited capacity for further disruption.

Taken together, our findings emphasize that the intensity of emotional distress, regardless of its origin, is key in predicting reliance on impulsivity or maladaptive strategies. This view is supported by research showing reduced use of adaptive strategies like cognitive reappraisal across a range of psychiatric disorders, such as major depressive disorder, bipolar disorder, borderline personality disorder, PTSD, substance use disorder, panic disorder [21,80], in which emotion dysregulation is a core transdiagnostic feature [81]. A recent neuroimaging meta-analysis identified convergent regional abnormalities in the dorsomedial PFC associated with reappraisal tasks across different mental disorders [79]. At the same time, insomnia and poor sleep quality are risk factors for developing severe mental health conditions, including depression and anxiety [78,81,82]. These effects are potentially mediated by shared disruptions in neurobiological systems that regulate sleep, mood, and emotion [83].

### 4.4. Age/gender in the insomnia-emotion dysregulation association

Our findings indicated that neither age nor gender (i.e., the female-to-male ratio) significantly moderates the relationship between insomnia and emotion dysregulation. This stability in the association contrasts with some prior studies that reported age and gender might influence the severity of insomnia [9] or its relationship with emotion regulation [33], with variations in symptoms and coping strategies observed across different life stages. The contrast between our results and previous studies underscores the enduring nature of the link between insomnia and emotion dysregulation, while acknowledging that demographic factors influence individual experiences and the manifestation of the consistent association. While the overall association is

stable, gender- and age-specific stressors such as hormonal changes, life transitions, and societal expectations can still influence how emotion dysregulation manifests and how individuals cope with insomnia [84–86]. These stressors might affect sleep-related emotional reactivity and symptom expression, leading to variations in personal experiences of emotion dysregulation and insomnia without changing the fundamental link between them.

### 4.5. Methodological strengths, key contributions, and open questions

We conducted this meta-analysis following best-practice guidelines for research syntheses in the health sciences [36], ensuring accuracy in search strategy, study selection, effect size coding, analysis, interpretation, and reporting. To our knowledge, this is the first quantitative synthesis of the link between insomnia and emotion dysregulation, employing a state-of-the-art meta-analytic approach that accounts for effect size dependencies, yielding more precise estimates. Our study advances prior studies by addressing the lack of a clear framework for emotion dysregulation and an explicit focus on insomnia symptoms, distinguishing overall association from the contribution of insomnia symptoms to greater emotion dysregulation, and examining the influence of health-related conditions and key moderators like age, gender, and specific emotion dysregulation subdomains. However, some questions remain unanswered due to limitations in the existing literature. For example, most studies focused on adults, with only three examined children or adolescents, leaving unexplored lifespan differences in the insomnia-emotion dysregulation link. Moreover, there is lack of longitudinal evidence on the interplay between insomnia and emotion dysregulation. Although our search strategy was not restricted to behavioral studies, the available literature extensively focused on psychological aspects, with little investigation into direct neurobiological mechanisms.

## 5. Conclusion

This meta-analysis provides a quantitative overview on the relationship between insomnia and emotion dysregulation. It demonstrates that insomnia is closely associated with emotion dysregulation, with individuals experiencing more severe insomnia symptoms showing greater emotion dysregulation compared to good sleepers. An increased tendency toward impulsivity and using maladaptive emotion regulation strategies primarily drives the association. While serious health-related conditions amplify the degree of this relationship, they do not alter the extent to which insomnia contributes to higher emotion dysregulation. This pattern remains consistent across age and gender groups. Our findings suggest the need for integrated interventions targeting both insomnia and emotion dysregulation, with a focus on controlling impulsivity and reducing reliance on maladaptive strategies. Future studies should assess the complex neurobiological mechanisms of the association between insomnia and emotion dysregulation, their predictive role on developing mental health conditions e.g., using machine learning methods, and design treatment strategies applicable across genders and age groups to ensure their effectiveness in diverse populations.

## Practical points

- Insomnia is linked to emotion dysregulation, making emotion dysregulation more pronounced in individuals with clinically significant insomnia symptoms compared to good sleepers.
- The association is primarily characterized by a shift towards impulsivity and applying maladaptive strategies rather than a lack of emotional functionality or difficulty using adaptive strategies.
- The association between insomnia and emotion dysregulation is not moderated by age and gender.
- Serious health-related conditions can intensify this association. However, their impact does not exceed the effect of sever insomnia symptoms on greater dysegulation.

## Research agenda

- Our cross-sectional findings highlight the need for future longitudinal, large-scale neuroimaging studies, as well as mega- and meta-analyses utilizing open-access data and international data collection initiatives such as the ENIGMA-Sleep consortium [87] to delve deeper into *(i)* how demographic-specific stressors influence the relationship between insomnia, brain, and emotion dysregulation, providing detailed neurobiological insights into their interaction; and *(ii)* how the strength of the association is more closely tied to the insomnia severity than the presence of additional health-related conditions.
- Future research should design clinical trials to assess targeted interventions that address different aspects of emotion dysregulation as part of insomnia treatment. These trials should also examine the applicability of these interventions across diverse demographic groups to help develop universal strategies for effective insomnia management.

## Authors' contributions

MT developed the idea. FS and NM conducted the literature research, study selection, and data extraction. FS registered the protocol at PROSPERO and performed all analyses in R. FS and MT drafted the latest version of the manuscript, while GMR, AE, MZ, HK, ANG, KS, CB, AAS provided comments and reviewed the manuscript. All authors read and approved the final version.

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## Declaration of competing interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

## Appendix A. Supplementary data

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