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THE IMPACT OF ATTENTIVE SYSTEM ON SLEEP REACTIVITY: A STUDY WITH A PARTICIPANT-APPLIED ELECTROENCEPHALOGRAPHY IN THE HOME ENVIRONMENT

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ABSTRACT

A comprehensive understanding of the processes leading to Insomnia Disorder (ID) is a crucial goal in clinical research. A central role is played by Sleep Reactivity (SR), describes as the extent to which an individual tends to experience sleep disturbances following a stressful event. Heightened SR is one of the most robust predictors of the onset and persistence of ID. Cognitive processes also contribute to triggering and maintaining hyperarousal, and excessive attention to sleep-related cues has been repeatedly identified as a factor promoting ID. However, how specific attentional components influence the sleep response to stress remains unclear.

The present study examined whether attentional performance modulates SR, investigating whether differences across attentional domains help explaining stress-induced sleep alterations in healthy individuals with (IS) and without (GS) ID symptoms. Forty participants were assigned to groups based on the scores of the Sleep Condition Indicator. They completed a two-week Sleep Diary, underwent attentional assessment, and were then exposed to a validated stress-inducing video based on the Trauma Film Paradigm. Objective sleep was recorded using a portable, home-based polysomnography that can be self-administered on the two nights before and after the stressor.

Subjectively, the stressor worsened sleep quality in both groups, but the deterioration in Total Sleep Time (TST) and Sleep Efficiency (SE%) was significantly greater in IS group. Objectively, stress induced a general disruption of sleep continuity and REM stability across the sample, yet IS participants showed a markedly stronger decline in SE%, TST, and REM arousal density, indicating reduced resilience of sleep architecture.

GLMM analyses showed that attention shapes the sleep response to stress. Executive Control (EC) predicted changes in subjective sleep quality: poorer executive functioning was associated with greater vulnerability after stress across groups. Orienting showed a group-specific pattern: in GS, better orienting was linked to more stable objective and subjective sleep efficiency, whereas in IS it was paradoxically associated with worsening indices. EC also modulated REM arousal density, with better abilities predicting reduced fragmentation in GS but increased fragmentation in IS.

Overall, attention emerges as a modulating factor of stress vulnerability. These results highlight the importance of interventions aimed at promoting a more flexible and less hypercontrolled use of attentional resources in individuals vulnerable to ID.

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1. Introduction

Our daily life is becoming, day after day, increasingly stressful, to the point that we are gradually getting used to this much more frenetic, precarious lifestyle, characterized by emotional distress. The fact that we manage to survive effectively does not mean that there are no consequences for our health and functioning. The connection is not always direct and obvious, but as we will see, there are many negative consequences that our organism suffers due to stressful situations or conditions.

Sleep, which represents one of the fundamental biological functions for maintaining homeostasis and our survival, is no exception. It is no coincidence that only in the last twenty years increasing attention has been placed on the reciprocal link between stress and sleep. It has become clear, in fact, that the same stressful event does not affect different individuals, especially their sleep, in the same way. From this observation arises the construct of Sleep Reactivity, which indicates the extent to which an individual is inclined to experience sleep disturbances following a stressful experience (Kalmbach et al., 2018a). The same stressor that may cause only mild and transitory sleep problems in one person may cause significant discomfort in another, up to the more severe consequence of developing a true sleep disorder. The study of this trait characteristic has therefore become increasingly important in recent years, especially in the context of Insomnia Disorder, in order to explore the role that stress plays in the onset of such a widespread and debilitating condition.

We will see that many discoveries have been made in this context; however, experimentally exploring such a phenomenon in a rigorous way is very challenging from various perspectives, and for this reason many questions remain unanswered. We are still far from defining in a robust and consistent manner the relationship between stress, stress reactivity, sleep disturbance, and Insomnia Disorder. The aim of my doctoral project was precisely to try to outline in more detail the framework that connects all these aspects.

One of the main questions driving this research was to identify the possible mechanisms underlying the phenomenon of Sleep Reactivity, since a better understanding of these mechanisms would provide us with more information on where and how to intervene to prevent the pathologization and chronicization of sleep disturbances. Among all cognitive functions, it seemed natural to start by investigating the role of attention, a domain highly implicated and studied in the context of Insomnia Disorder. Its role in the

maintenance of the disorder has been widely explored; however, its potential contribution in the early phases of the disorder, particularly regarding its influence on the stress response, remains an uncharted path that this thesis work set out to begin exploring.

A better understanding of the mechanisms that regulate the sleep response to stress in vulnerable and non-vulnerable individuals could be an excellent resource from both a preventive and therapeutic point of view. It would allow us to gain insights into what to strengthen from a preventive perspective, what to intervene on promptly in the acute phase to avoid chronicization, and which new aspects therapies could potentially target.

2. Understanding Insomnia

2.1. Insomnia Disorder and Its Impact

2.1.1. Clinical Definition and Insomnia Subtypes

Sometimes symptoms associated to a disorder are so widespread and relatable that they transcend clinical boundaries and become part of everyday language. "*I had a night of insomnia*" it's a common statement in casual conversation, and most, often through personal experience, immediately understand what is meant. It's true, indeed, that among the healthy adult population, a range between 30% and 36% experiences at least one insomnia symptoms. At first, this proportion seems to be alarming, are we facing an insomnia epidemic? However, although Insomnia Disorder (ID) is indeed the most common between sleep disorders, the rates decrease considerably if we apply a more accurate definition (Morin & Jarrin, 2022). For example, when the presence of daytime consequences is included, the proportion of healthy people experiencing ID drops to 6%-10% (Ohayon et al., 2009). It is therefore essential to recognize that when referring to ID, we are not simply talking about sleep disturbances, but rather a complex and multifactorial disease. In particular, the 3rd edition of the International Classification of Sleep Disorders (ICSD-3) defined ID as a persistent difficulty with one or more aspects of sleep initiation, maintenance, early morning awakenings, or overall sleep quality, despite the presence of adequate conditions for sleep. These nocturnal symptoms must also result in daytime impairments, such as fatigue, daytime sleepiness, mood dysregulation, or mild cognitive impairments (American Academy of Sleep Disorder [AASM], 2014). This is a much more complete picture, but an important question remains: what if, for instance, I experience this type of symptoms, one night of poor sleep, with, difficulties in falling asleep, that make me feel very sleepy and in a bad mood the day after, only once or twice a month? Would this be sufficient to classify me as an ID patient? Here, the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) offers additional guidance: to meet the diagnostic criteria the symptom described above must be occur at least three times per week for a minimum of three month (DSM-5, 2013). Recent epidemiological evidence supports how these stricter definitions influence prevalence estimates. A large meta-analysis by van Straten and colleagues (2025) found that, when DSM-5 criteria were applied through structured clinical

interviews, the pooled prevalence of ID in the general population was 12.4% (95% CI: 9.0–16.8%), whereas studies relying on self-reported DSM-based questionnaires showed a higher rate of 16.3%, much higher than those reported by Ohayon and colleagues (2009). In contrast, less specific instruments such as the Insomnia Severity Index or the Athens Insomnia Scale yielded much broader estimates (from about 7.5% to 32%). These findings confirm that ID remains highly prevalent worldwide, but also highlight that prevalence rates strongly depend on how strictly diagnostic criteria are applied (van Straten et al., 2025).

While three months may seem a lengthy period, longitudinal evidence highlight the unstable nature of ID: a patient who meets diagnostic criteria at time point zero, may experiences remission in six months, only to relapse into a fully symptomatic state within other six months (Morin et al., 2009). At the same time, there are several pieces of evidence that show ID as a disturb that tend to persist over the years, with considerable variability in its course and persistence rates. Indeed, data from different cohorts around the world reveal high variable one-year-persistence rates, ranging from 31% in the United States (Ford et al., 1989), 44% in Sweden (Jansson-Fröjmark & Linton, 2008), 69% in the United Kingdom (Morphy et al., 2007), and 74% in Canada (Morin et al., 2009), and these rates tend to decrease when longer follow-up are considered (Fok et al., 2010; Kim et al., 2009; Morgan & Clarke, 1997).

Only by diving into its definition, we realize that talking about ID encompasses a family of heterogeneous sleep disturbances that can affect individuals in many different way and follow highly variable trajectories. Reflecting this complexity, in the history of ID different classification followed one another: the ICSD-1 comprised seven subtypes (American Sleep Disorders Association, 1990) that increased to nine in ICSD-2 (AASM, 2005). Nevertheless, studies soon demonstrated that these categorizations lacked reliability and validity, as the described subtypes tended to be unstable over type and often overlapped (Buysse et al., 1994; Edinger et al., 2011). Because of this instability and inconsistency, the diagnostic system proved too complex and impractical in clinical settings, for the absence of clear and specific guidelines tragedized on all the different subtypes (National Institutes of Health [NIH], 2005). To address these limitations, in the ICSD-3 a more functional approach has been adopted. This edition comprises only three possible subtypes, which can be applied to all patients, despite the presence of comorbid

condition, and with a reduced risk of overlap (AASM, 2014). One of the most important changes introduced was the removal of the distinction between primary and secondary ID. Traditionally, primary ID occurs independently of the presence of other medical or psychological conditions, whereas secondary ID refer to a condition in which the ID symptomatology is a consequence of pre-existent disorders, such as psychiatric or medical conditions; for instance, both ICSD 1 and 2 included a subtype named "Insomnia due to drug or substance" (Lichstein et al., 2001). This distinction proved to be artificial, since in clinical practice primary and secondary ID shared similar features (Riemann et al., 2022). Moreover, it's well known that ID not only frequently co-occurs with psychiatric conditions, but, in some cases, it even precedes their onset and eventually evolve into an independent disorder (Khurshid, 2018). For this reason, referring to *secondary* ID appears strongly inappropriate, and the most updated classification no longer emphasize this distinction (AASM, 2014).

ICSD-3 defines three categories as follows:

1. Chronic ID: Defined by the same time-related criteria needed for ID diagnosis in DSM-5: symptoms must be present minimum three times a week for at least three months. This condition is characterized by persistent difficulties with sleep initiation and/or maintenance, accompanied by daytime impairments.
2. Short-Term ID: Presents the same symptomatology of chronic ID but persists for less than 3 months. It's usually associated with a triggering event.
3. Other ID: Includes all the cases of ID-like sleep disturbances that do not meet the criteria for the two previous categories but still warrant clinical attention.

Taken together, these evolving definitions and classifications highlight the complexity but also the importance of better understand ID.

2.1.2. Psychological and Physiological Consequences

Introducing ID, we have strongly emphasized the pivotal role of the associated daytime consequences. These consequences extend beyond the emotional distress that accompanies experiencing sleep disturbances, and reflect the broader, large-scale implications of the disease. In the past decades, more and more studies focused on the impact of untreated, or not appropriately treated ID, showing that it affects not only to the health care system, in terms of economic cost and resource demands, but also daily life

of general population (Olatunde & Patton, 2023). It's pretty common to read the term "*burden*" refer to ID, as its presence its strongly associated with increased health care services used, even compared to other clinical groups. This could be due to the absence of clear and universally shared guidelines for the treatment (Daley et al., 2009). Indeed, although the scientific community widely agrees that Cognitive Behavioural Therapy for Insomnia (CBT-I) represents the first-line treatment (Hertenstein et al., 2022; Jernelöv et al., 2022; Morin & Espie, 2007), ID continues to be associated with and higher numbers of ambulatory visits and frequent prescription of hypnotic drugs (Ozminkowski et al., 2007). Beyond direct health care expenses, it has been estimated that indirect costs represent the largest proportion of the total economic burden of ID (Daley et al., 2009, Wickwire et al., 2016). Indirect consequences are mainly represented by a recent work efficiency (i.e., higher rates of absenteeism and lower productivity) (Chen et al., 2018; DiBonaventura et al., 2015; Hui & Grandner, 2015), an increased risk of accidents (Morin et al., 2016; Watson et al., 2015) and greater vulnerability to health problems (Liu et al., 2016; Thomas et al., 2017). In light of this, it becomes essential to examine more closely the impact of ID from different perspectives, in order to fully capture what cause its burden.

It's impossible to study ID without taking in account its impact on our cognitive and emotional functioning. Numerous studies have analysed the impact of ID symptoms on cognitive performance, and most converge in concluding that ID is accompanied by a reduced performance, from both objective and subjective perspectives (Wardle-Pinkston et al., 2019). Overall, cognitive functioning is impaired in ID patients, but some domains appear more strongly affected than others (Wardle-Pinkston et al., 2019). The most consistent findings involve deficits in executive functions, with difficulties in problem solving (Altena et al., 2008; Fang et al., 2008), as well as impairments in focusing attention (Altena et al., 2008; Varkevisser et al., 2007) and the memory abilities, in particular, working memory (Haimov et al., 2008; Hauri et al., 1997; Varkevisser et al., 2007) and episodic memory (Backhaus et al., 2006; Hauri et al., 1997; Orff et al., 2007). Moreover, individuals with ID often show slower reaction times (Fortier-Brochu et al., 2012; Wardle-Pinkston et al., 2019). Collectively, these impairments directly affect daily functioning, with consequences that extend from professional performance to personal life.

The interplay between ID and emotional dysregulation constitutes a core element of the disease. Not only ID is frequently accompanied by mood symptoms (Levenson et al., 2015), but these symptoms appear to contribute to its maintenance (Baglioni et al., 2010). The mechanisms underlying this bidirectional relationship between sleep and emotion remain unclear. Some authors have proposed that the presence of emotional hyperarousal leads to an increase autonomic reactivity, that interferes with normal sleep processes (Kales et al., 1979; Bradley et al, 2001). In particular, ID is commonly associated with internalizing problems, such as depressive and anxiety symptoms (Taylor et al., 2003; Reid et al., 2009). Even in the general population, there is a strong link between sleep and emotional regulation: it is well established that negative emotional states are associated with a poorer sleep quality (Goldstein & Walker, 2014). Accordingly, it is not surprising that, compare to healthy controls, ID patients show higher levels of negative emotions, lower positive emotions and a grater tendency to use maladaptive emotional regulation strategies as it has been confirmed in a recent metanalysis (Samea et al., 2025). This is particularly relevant not only because it impacts on patients' daily life, but also because it plays a role in perpetuating the disorder (Jansson-Fröjmark & Hossain, 2024). The emotional dysregulation that characterizes individuals with ID can directly contribute to difficulties in the sleep onset phase. For these patients, distancing themselves from negative thoughts and moods accumulated during the day it's more challenging, while, on the other hand, worries related to sleep itself emerge (Schmidt et al., 2011). A well-documented phenomenon in this phase is the tendency to interpret neutral elements as negative, especially when they are sleep-related (Akram et al., 2023; Baglioni et al., 2010). This happens not only at a subjective level: for instance, Howlett and colleagues observed that patients with ID exhibit increased amygdala activation in response to ID-related stimuli (Howlett et al., 2020). In such a state of emotional hyperactivation, falling asleep becomes increasingly difficult, forming the nucleus of the vicious circle. Catastrophic thoughts such as "*I won't fall asleep again tonight, and tomorrow I'll be too tired to function*" increase emotional activation and perceived distress, which in turn further hinders sleep onset and so on (Doghramji, 2014; Vaziri et al, 2021). If extended over time, these emotional impairments may result in social withdrawal or strained relationships, both at work and in personal life (Simon et al., 2020; Vanek et al., 2020).

The interaction with emotional dysregulation paves the way to understand the frequent presence of psychiatric comorbidities in patients with ID. The relationship between psychiatric disorders and ID is, in fact, bidirectional: while ID can emerge as a symptom or consequence of a psychiatric condition, it can also act as a risk factor for the development of such conditions (Hertenstein et al., 2023). The most documented and studied is the relationship between ID and depression, as it has long been known that ID frequently precedes the onset of depressive episodes (Franzen & Buysse, 2008). Numerous longitudinal studies converge in showing that ID increase the risk of developing depression and the presence of ID can even predict the degree of the increase in depressive symptom severity (Buysse et al., 2008; Byrne et al., 2019; Suh et al., 2013). On the other hand, evidence also supports the reverse pathway: individuals with depression but without ID are at greater risk of later developing ID (Hertenstein et al., 2019; Sivertsen et al., 2012).

The link between ID and anxiety disorders is equally robust: anxiety symptoms can heighten both physiological and psychological activation, which, as previously discussed, represents one of the core mechanisms perpetuating ID. Indeed, the presence of anxiety at baseline has been demonstrated to predict the development of future ID (Jansson-Fröjmark & Lindblom, 2008; Neckelmann et al., 2007). The opposite pattern has also been observed in different longitudinal studies: the presence of ID constitutes a risk factor for developing anxiety disorders (Breslau et al., 1996; Jansson-Fröjmark & Lindblom, 2008; Johnson et al., 2006).

ID is also a recognized risk factor also for alcohol abuse. Not only is ID frequently present among alcohol abuser (Brower & Perron, 2011), but its symptoms have been shown to correlate with, and even predict, the risk of relapse in both adults (Arnedt et al., 2007; Brower, 2003;) and adolescents abusers (Wong et al., 2015; Johnson & Breslau, 2001).

The relationship between ID and psychosis has been explored as well. ID predicts the increase and the persistence of psychotic experiences (Reeve et al., 2018). Prospective evidence, from a single cohort, further suggests that ID increases the risk of psychotic disorder (Sheaves et al., 2016). Importantly, the ID–psychosis link appears to be partly mediated by negative affect (e.g., anxiety, depression), which amplifies psychotic experiences (Reeve et al., 2018). These findings are consistent with literature that reports

that sleep disturbances can predict the occurrence of psychotic symptoms the following day (Hennig et al., 2020; Simor et al., 2019).

Finally, we must consider Post-Traumatic Stress Disorder (PTSD), as the presence of sleep disturbances constitutes diagnosis diagnostic criterion (DSM-5, 2013). Indeed, ID symptoms are present in up to 80–90% of PTSD patients (Koffel, 2016). Moreover, evidence from the literature shows that ID constitutes a risk factor for future developing of PTSD (Miller et al., 2021; Wang et al., 2018). The overlap between these two conditions likely reflect shared pathophysiology: a state of chronic hyperarousal with sympathetic nervous system up-regulation and Hypothalamic-Pituitary-Adrenal (HPA) axis dysregulation, limbic–prefrontal imbalance (heightened amygdala reactivity with reduced top-down control), and REM-sleep disruption that impairs fear-extinction and emotional memory processing-mechanisms that both precipitate and perpetuate ID and trauma-related symptoms (Bonnet & Arand, 2010; Vgontzas et al., 2001; Goldstein & Walker, 2014; Pace-Schott et al., 2015; van Liempt et al., 2013).

Other psychiatric and mental disorders have been less studied in relation to ID, (e.g., Attention Deficit Hyperactivity Disorder [ADHD], Obsessive-Compulsive Disorder [OCD] or personality disorders) so further research is needed to draw conclusions (Hertenstein et al., 2023). Nevertheless, it can still be expected that ID impacts psychiatric disorders more broadly, and particularly their course, given the well-documented negative effects of ID on quality of life (Khurshid, 2018).

As we previously mentioned, not only ID impact on mental health, but also physical health, since sleep is involved in numerous physiological processes. For instance, several studies show that poor sleep negatively impacts on metabolic functioning, affecting hormones regulation (Duan et al., 2022). In addition, sleep plays a crucial role in immune regulation: circadian system disruption affects the HPA axis, which in turn alters immune function by heightening inflammatory signaling and reducing system response efficiency. Consequently, ID can weaken immune system and increase vulnerability to illness (Besedovsky et al., 2012; Imeri & Opp, 2009; Prather et al., 2012). The impact that ID has on cardiovascular system is also worth to be mentioned. Sleep duration has been identified as a predictor of cardiovascular disease (e.g., hypertension, coronary artery disease, and stroke). Shorter sleep is consistently associated with weight gain, obesity problems, hypertension and heighten stress, all of which impact on cardiovascular health

(Belloir et al., 2022; Cappuccio et al., 2011; St-Onge et al., 2016). Many others are the diseases influenced and worsened by ID because it disturbs body's regulation of pain by amplifying nociceptive responses (Finan et al., 2013). Many other diseases are influenced and worsened by ID, as it disrupts the body's natural regulation of pain by amplifying nociceptive responses (Finan et al., 2013). This occurs because chronic sleep disturbance alters central and peripheral mechanisms involved in pain modulation: it increases sympathetic nervous system activity and cortical arousal, impairs descending inhibitory pathways, and promotes low-grade inflammation and neurochemical imbalances (e.g., reduced serotonin and endorphin availability) (Carter et al., 2018). As a result, individuals with ID exhibit heightened pain sensitivity and reduced pain tolerance, which can negative affect chronic pain conditions (i.e., fibromyalgia, chronic back pain, and tension headaches) and it may further hinder recovery from illness or injury (Choy, 2015; Stiefel & Stagno, 2004).

We can conclude that ID exerts a profound burden (on individual and society) across cognitive, emotional, psychiatric, and physiological domains, reinforcing its status as a systemic disorder with wide-ranging consequences. Recognizing this multidimensional impact is essential for understanding not only the clinical relevance of ID but also the mechanisms that may underlie its persistence.

2.2. The Aetiopathogenesis of Insomnia Disorder: Current Perspectives and Unresolved Questions

2.2.1. Considering Insomnia Pathogenesis

Knowing how a disorder emerges, develops and the mechanisms that underlie its maintenance is fundamental to a better and more timely management. Many studies have investigated the aetiopathogenesis of ID, but so far, no single, universally shared model has been proposed (Levenson et al., 2015). This is probably because, as described in the previous chapters, we are dealing with a complex and multifactorial disease, and a model that truly explains the causes of ID, and how from this causes lead to its development, should take into account and clarify the heterogeneity intrinsic to ID.

On one hand, the aetiology of ID is still under vivid debate; on the other hand, the most well-know and widely pathogenic framework is the behavioural-cognitive model proposed by Spielman in 1987, known as "*3P Model*"[1]. This model posits that acute ID results from the co-occurrence of traits that make an individual more vulnerable to ID, called *Predisposing factors* (1); the presence of events or situations in the individual's life that cause him distress, called *Precipitating factors* (2); and, if maladaptive coping behaviours are added to this pictures, these act as *Perpetuating factors* (3) which lead the individual toward the chronicization of the disease (Figure 1) (Spielman et al., 1987). In details:

1. Predisposing factors: These comprise a plethora of different innates traits that can characterized the individual, comprising both biological and psychological dimensions.

   Biological factors include age and biological sex, as the vulnerability to ID increases with age and its prevalence is generally higher in women. Aging is associated with a partial degradation of sleep-regulation mechanism, with changes that affect both macro- and micro- sleep architecture (e.g., the well-known phenomenon of advance sleep phase, reduce sleep continuity, decreased slow-wave density, etc.) (Taillard et al., 2021). Moreover, these two factors can interact to further increased ID vulnerability: during the menopause phase, women experience symptoms, ranging from hot flashes to psychological distress, which negatively impact sleep quality (Zeng et al., 2025). Indeed, the prevalence of sleep disorder in the postmenopausal phase is alarmingly high, at around 50% (Salari et al., 2023). (Spielman et al., 1987). Others predisposing factors are more directly related to an individual's everyday sleep experience. For example, chronotype: people with an evening chronotype are at greater risk of developing ID (Zhao et al., 2025). Genetic influences also play a significant role A large body of research focuses on the role of genes in ID, and the overall evidence suggests that ID is not liked to a singular gene, but is a polygenic condition: mani loci contribute, each

[1] In the scientific literature, references to the 3P Model can be found under both the terms aetiopathogenetic model and pathogenetic model. In the present work, I emphasize that we are dealing with the latter, because in order to speak about aetiology, a model should propose an explanation of the causes that determine the disorder. This is not what Spielman did in his original conceptualization: it is instead limited to describing the different phases of disease development.

with small effects and complex interactions (Lane et al., 2019; Watanabe et al., 2022). Candidate genes include circadian clock genes, genes implicate in neurotransmitter regulation (especially the ones involved in the serotonergic system) or genes related to the HPA axis regulation (Byrne et al., 2013; Gehrman, 2013; Lind & Gehrman, 2016; Zhao et al., 2024). Nevertheless, family and twin studies have found that the hereditability of ID to be moderate (the 22-25% circa) (Lind et al., 2015), underscoring the importance of considering the weight of gene-environment interaction and the role of other contributing factors. Psychological predisposing factors include personality traits, such as neuroticism, which is characterized by heightened emotional responses, excessive worry (i.e., repetitive and ruminative thinking), and difficulties in coping with stressful situations (Shao et al., 2025). Other personality traits that exhibit an association with ID are perfectionism and introversion (Akram, 2023; Zakiei et al., 2024), both may foster excessive self-criticism, social withdrawal, and persistent cognitive activity, all of which can interfere sleep initiation.

2. Precipitating factors: Understanding predisposing factors is important, as let us recognize individuals at-risk, a key aspect of disease prevention. However, if predisposing factors alone were sufficient to determine ID, every menopausal woman with neurotic traits would receive an ID diagnosis. In reality, a person whose disposition makes them more likelihood to develop ID, needs to face a precipitant situation in order for sleep disturbances to emerge. Precipitating factors can be describe as life events that impact on the regular functioning of the individual's sleep system (Perlis et al., 2010). We can translate this definition in life events that are often, *but not always*, stressful experiences (Harvey, 2002). Considering the following two examples:
    a. A man, following his divorce, is involved in ongoing legal issues concerning money and custody. As a result, he is constantly worried about the situation and its potential outcomes, which produce a state of psychological hyperactivation characterized by ruminative thinking and intrusive thoughts that occur not only throughout the day but also at bedtime. This leads to difficulty in maintaining sleep and preserving sleep continuity.

b. Another man receives a long-awaited promotion, which makes him very satisfied and happy. This promotion required him to adjust to an earlier working schedule. To get used, he begins to act monitoring strategies (e.g., monitoring the clock at night to ensure sufficient rest). While such strategies are helpful during the day, their inadvertently increase cognitive arousal at bedtime, making it harder to fall asleep and causing early morning awakenings.

Both cases show how sleep disturbances may arise in two individuals with predisposing factors, even if they have perceived the two events in very different ways. This distinction is relevant as this work will primarily focus on how the sleep system reacts to stressors in the phase that could precede ID onset. However, it's important to note that not every precipitating factor must be subjectively perceived as stressful to exert a negative impact on the sleep system.

3. Pepertuating factors: When does an acute sleep disturbance, something that almost everyone has experienced once in a lifetime - became a long-lasting problem? This occurs when a person that is experiencing acute ID begins to adopt strategies, usually perceived as compensatory, that in fact produce the opposite effect. This approach crystallizes the problem and contribute to the development of chronic ID. Such strategies include negative behaviours, thoughts and association that further disrupt sleep. Even when the original event disappears, these learned tendences continuing to disturb sleep (Spielman et al., 1987).

   For example, let's go back to our unfortune, newly divorced man: over time, he becomes increasingly concerned about his sleep situation. In response, he decides to go to bad earlier in the evening to "gain" more sleep and takes long naps during the day to cope with sleepiness. He also begins checking the clock repeatedly during the night and reduces social and physical activity, especially in the evenings, to "save energy". As months pass, the legal situation came to an end, yet he continues to struggle with. His compensatory strategies, though well intentioned, paradoxically reinforce his worries about sleep, heighten his cognitive arousal in the sleep environment and reduce sleep drive. Thus, even though the stressful event has ended these behaviours perpetuate ID symptoms.

It has been demonstrated, thought if it may seem counterintuitive, that excessive efforts to control sleep, including rigid pre-sleep routines and safety behaviours, often backfire. By focusing attention on the act of sleep, they increase cognitive arousal and make sleep even harder to achieve (Lancee & Kamphuis, 2025). This happens because the individual is trying to induce and control a process that should be spontaneous, thereby disrupting its natural course. Safety behaviours are helpful in reducing sleep-related anxiety, but they have been shown to predict ID symptoms at one-year follow-up (Fairholme & Mamber, 2014; Lancee & Kamphuis, 2025). Such behaviours are often induced, and indeed they have been found to be predicted by the presence of dysfunctional beliefs about sleep. These include unrealistic expectations (e.g., "*I must fall asleep as soon as I go to bed, otherwise my night will be ruined.*"), wrong ideas about what cause sleep disturbances (e.g., "*The reason I don't sleep is that I'm not following my bedtime routine perfectly*"), and catastrophic attributions of its consequences (e.g., "*I must get 8 hours or I'll be useless*") (Morin et al., 2007; Woodley & Smith, 2006). Moreover, ID severity is linked to the extend of dysfunctional beliefs, particularly in patients with comorbid mood disorders (Chang et al., 2020; Karsan et al., 2024). Cognitive perpetuating factors therefore play a crucial role as well. Worry, rumination, and catastrophic thinking are repeatedly identified as maintaining factors (Perlis et al., 2024). In the precipitating phase, worry and ruminations tend to be related to the life, precipitating, event, while in the chronic phase, the focus of subject's concerns shifts toward sleep itself (Harvey, 2002; Jansson-Fröjmark et al., 2011; Lemyre et al, 2020). Such negative and repetitive thoughts have been found to correlate with ID symptoms severity (even after controlling for psychiatric symptoms) (Karsan et al., 2024; Muthuraman et al., 2024), because they increase pre-sleep arousal, interfering with both sleep initiation and maintenance.

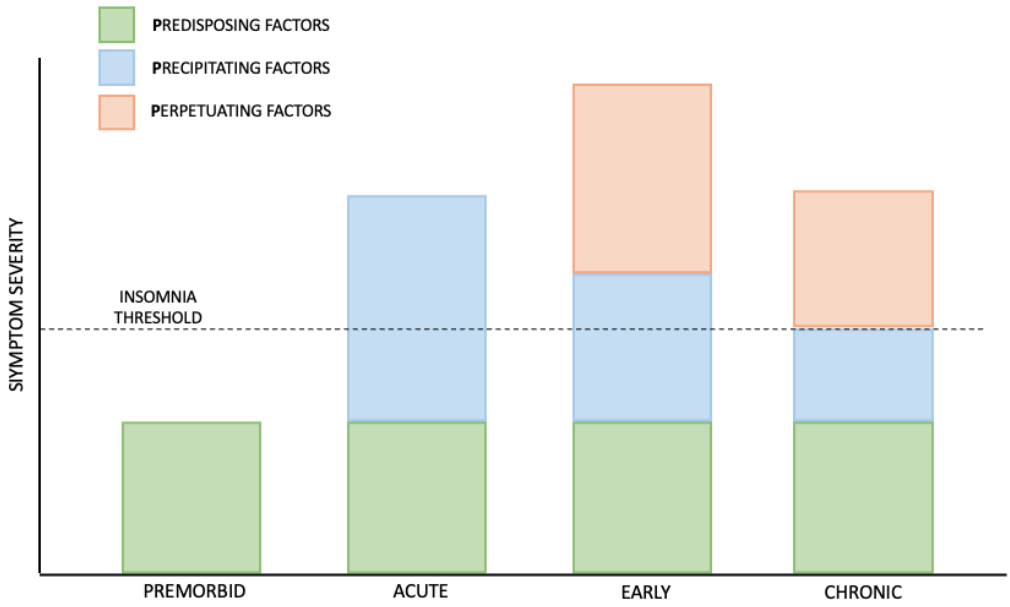


Figure 1. *Schematic representation of Spielman's 3P model, recreated and adapted by the author (Spielman, 1987; License number: 6115391002331).*

2.2.2. The Reference Framework: The Hyperarousal Hypothesis

Since the very beginning of this dissertation, one concept has persistently resurfaced: arousal. It refers to a state of physiological and/or psychological activation in which the body mobilizes its resources to be prepared to respond to a stimulus or a situation (Isaac et al., 2024). From this definition, it's easily to understand that arousal it's evolutionary useful, allowing individuals to cope with abnormal situation with the necessary resources. However, if this state persists beyond the circumstances that would requires a mobilization, it can intuitively lead to maladaptive consequences. This is precisely the assumption underlying the model first formalized by Bonnet and Arand in 1997: the Hyperarousal Model, which is is the most wildly shared model of ID aetiopathogenesis. It posits that ID results from an underling state of increased psychological and physiological arousal (Bonnet & Arand, 1997; Riemann et al. 2010). The model is particularly relevant because it focuses on the precence of an alternated state of arousal, an element that many other ID theories has highlighted, including the Cognitive Model (Harvey, 2002), the Neurocognitive Model (Perlis et al., 2007), and models emphasizing

the role of emotion regulation and of Rapid Eye Movement (REM) sleep alteration (Feige et al., 2008; Riemann et al, 2012; Van Someren, 2021).

In its most recent conceptualization, the Hyperarousal Model describes a multidomain condition that affect functioning not only at night but throughout the entire day (Dressle & Riemann, 2023). One line of research has drawn attention to a phenomenon that characterized ID patients that appears to be counterintuitive: although these patients are in a state of sleep debt that causes daytime symptoms such as fatigue, sleepiness and connected consequences, one might expect daytime sleepiness to be common and easy in this population. Nevertheless, from Multiple Sleep Latency Test (MSLT)[2] experiments emerged that ID patients exhibit longer Sleep Latency (SL) compared to healthy controls (Li et al., 2014; Roehrs et al., 2011), a finding that distinguishes them from simply sleep-deprived subjects, who typically present shorter SL (Arand et al., 2014). Consistently, recent evidence from Fasiello and colleagues (2023) shows that individuals with ID rarely report excessive daytime sleepiness on subjective measures (measured with Epworth Sleepiness Scale, [ESS]), instead displaying predominant fatigue, further supporting the hyperarousal model of ID (Fasiello et al., 2023).

There is abundant physiological evidence, across multiple systems, supporting this hypothesis.

Considering sleep itself first, from and electrophysiological perspective ID is consistently associated with increased fast-frequency activity (i.e. beta and gamma frequencies), and decreased low-frequencies activity (i.e. delta power), during both NREM and REM (Dressle et al., 2023; Kang et al., 2022; Zhao et al., 2021). Patients also exhibit increased alpha frequency during the onset period (Berra et al., 2024) and modest increases in alpha and sigma during REM (Zhao et al., 2021), reflecting persistent cortical arousal throughout the night. Moreover, high-frequency EEG, in particular gamma band, correlates positively with maladaptive pre-sleep cognitive arousal (Dressle et al., 2023).

[2] The MSLT is a standard procedure in sleep medicine used to measure the physiological tendency to fall asleep during the day. Usually, it is employed to evaluate excessive daytime sleepiness, During the test, the patient is asked to lie down in a quiet, darkened room with a polysomnograpich (PSG) setup. They are given five nap opportunities of 20 minutes each, spaced approximately two hours apart, from morning to late afternoon. Sleep Latency (SL) is taking in account: a SL under 8 minutes indicates the presence of pathological daytime sleepiness, while between 10 to 15 minutes is the normal range (Carskadon, 1986).

Research also highlights REM sleep fragmentation as a key correlate of ID. A significant increase in microarousals, specifically during REM, has been observed in ID, and is closely related to longer subjective wake time (Feige et al., 2008; Ren et al., 2023). Converging evidence links REM instability to impaired overnight emotional processing: fragmented REM prevents the usual resolution of emotional distress. Indeed, ID patients show increased distress reactivity after sleep, whereas healthy sleepers show reductions (Wassing et al., 2016; Wassing et al., 2019a). Neurobiologically, this is attributed to persistent noradrenaline release due to incomplete silencing of the locus coeruleus during REM, which prevents normal amygdala down-regulation and synaptic depotentiation (Osorio-Forero et al., 2022; Wassing et al., 2019a). As a result, ID patients show continued limbic reactivity to past negative memories, unlike healthy individuals who disengage emotionally from them (Wassing et al., 2019b).

From the genetic point of view, as we have already discussed, ID is influenced by multiple genes, many of which are implicated in arousal response/regulation. Examples includes genes regulating adenosine receptors, implicated in sleep pressure, (Byrne et al. 2012; Tartar et al. 2021), GABA receptor genes, that plays a in the inhibitory control a mechanism strongly related to sleep initiation, (Xiang et al. 2023; Zhu et al. 2024) and circadian clock genes (Emekli et al. 2020; Lee et al. 2023).

One of the main systems affected is the autonomic nervous system (ANS): several studies have documented elevated heart rate (HR) in patients with ID both during the pre-sleep period and throughout the night, together with reduced parasympathetic activity and increased sympathetic tone (Jarrin et al., 2018). Body temperature has also been studied as a marker: ID patients show elevated nocturnal body temperature compared to controls, though not consistently during the day, highlighting the importance of circadian influences (Lack et al., 2008; Lushington et al, 2000). Furthermore, an increased basal metabolic rate has been reported in ID, indicating that heightened physiological activation extends beyond cardiovascular regulation (Bonnet & Arand, 1995; Bonnet & Arand, 2003).

Research on cortisol, the key hormone of the HPA axis, points to increased functioning of the neuroendocrine system in ID. Several studies have found elevated evening, nocturnal, or morning cortisol secretion in patients with ID (Passos et al., 2023; Yap et al., 2024).

Immune system alterations have been linked to ID as well. Evidence includes a reduced number of natural killer cell during ID patients' night sleep (Tang et al., 2025), increased interleukin-6 secretion (Burgos et al., 2006), which is also interestingly modulated by subjective poor sleep quality (Lee & Park, 2024), and associations between deteriorated sleep indices and other inflammatory markers such as C-reactive protein and the Tumor necrosis factor (Dzierzewski et al., 2020; Okun et al., 2009).

Neuroimaging studies provide direct evidence of structural and functional brain alterations in ID, further supporting the Hyperarousal Model. Single Photon Emission Computed Tomography (SPECT) studies have revealed hypoperfusion in multiple regions at sleep onset, most prominently the basal ganglia, with normalization after CBT-I (Smith et al., 2002; Smith et al., 2005). Positron Emission Tomography (PET) studies have consistently shown increased global glucose metabolism during both wakefulness and Non-REM (NREM) sleep, with smaller declines from wake to sleep in arousal-related regions, such as the thalamus, hypothalamus, and ascending reticular activating system, as well as reduced metabolism in the prefrontal cortex (Nofzinger et al., 2004). Structural Magnetic Resonance Imaging (MRI) identified bilateral and subfield hippocampal volume reductions in ID (Cavaillès et al., 2025; Joo et al., 2014). Functional MRI (fMRI) has revealed hypoactivation of medial and inferior prefrontal cortices during cognitive tasks, which returns to normal levels following CBT-I, suggesting compensatory recruitment during performance (Altena et al., 2008; Kim et al., 2017). Finally, Magnetic Resonance Spectroscopy (MRS) has shown globally reduced GABA levels in ID (Winkelman, 2008), indicating impaired inhibitory control in central nervous system function. Overall, these findings converge to suggest that ID is characterized by altered brain function, compromised inhibition–excitation balance, and structural changes in key cognitive and emotional circuits.

At last, Event-related potential (ERP) studies also reveal that ID patients exhibit altered cortical responses to auditory stimuli, during wakefulness and sleep onset. Findings include increased P300 amplitudes after poor nights of sleep (Devoto et al., 2003) and larger N100 amplitudes (Turcotte & Bastien, 2009; Xu et al., 2025; Yan & Lo, 2007). Results for later components are less consistent: one study found increased P220 and reduced N350, during the sleep onset (Turcotte & Bastien, 2009), whereas another observed decreases in both components during stage 2 sleep (N2) (Yan & Lo, 2007).

Subjectively, ID patients often report the sensation of having a constantly "*overactivated mind*", particularly at night. Many psychological mechanisms are involved, and most of them already discussed in relation to perpetuating psychological factor, such as excessive worry, rumination, and intrusive thoughts, which are especially pronounced in the pre-sleep period (Carney et al., 2010; Lancee et al., 2017; Wicklow & Espie, 2000). Studies implying the Pre-Sleep Arousal Scale (PSAS), a questionnaire that measures cognitive and subjectively perceived physiological hyperarousal, consistently show higher pre-sleep cognitive activity in ID compared to good sleepers (Harvey, 2002; Lebrun et al., 2019; Sharman, et al., 2022). However, this hyperarousal activation is not confined to nighttime, evidence supports a 24-hours hypercognitive activation, that correlates with objectively and subjectively poorer sleep (Dai et al., 2024; Kallestad, 2010; Spiegelhalder et al., 2012).

Cognitive hyperarousal can also produce emotional distress and dysregulation, which, as highlighted before, are commonly present in ID (Jansson-Fröjmark & Hossain, 2024; Samea et al., 2025). This can trigger intrusive thoughts and worries which, combined with sleep-related concerns, heighten arousal and interfere with sleep initiation, thereby creating a vicious cycle that perpetuates ID.

Despite this broad evidence base, it remains unclear whether the alterations observed in these systems represent causal mechanisms driving sleep disturbance or rather secondary consequences of chronic sleep loss. Still, even if some alterations are consequences, this does not invalidate the model: the Hyperarousal Hypothesis claims the presence of complex, bidirectional relationship between arousal systems and sleep-regulating mechanisms. Indeed, Dressle and Riemann in 2023, suggest that most of the elements shaping different phases of ID progression, especially the predisposing, and perpetuating factors, but also precipitating factors (such as illness-induced worry and physiological arousal), can be interpreted as s both markers of hyperarousal and consequences of it (Dressle & Riemann, 2023). What remains essential is to disentangle this pervasive state of hyperactivation.

3. The Strong Interplay Between Stress and Sleep Disturbances

3.1. What We Are Talking About When We Talk About Stress

As we argued in the introduction, the main focus of this work is understanding how our sleep system reacts to the induction of a stressor. Therefore, to better understand the relationship between sleep and stress it is first fundamental define what stress is.

Nowadays experiencing stress seems to be an integral part of modern life. Concerning data on stress rates seem suggest the fact that we are undergoing a "*stress pandemic*". A large study analysed, using data from the Gallup World Poll, stress trends in 149 countries, through an annual questionnaire, over 14 years (2007-2021). At the beginning of the study, 26% of participants reported having experienced stress the day before; between 2012 and 2019 this percentage rose to 36%, reaching its peak (38%) at the end of the study period. This increase was largely consistent across included nations, with almost 85% of countries showing an upward trend (Piao et al., 2024). One might argue that these results were strongly influenced by the COVID-19 pandemic, but date from the consequent Gallup Global Emotions Report show only a slight decrease. In 2023, 37% of the respondents still reported having felt stressed the previous day (Gallup, 2024). Whether or not this is related to COVID-19, the data underline that we are facing a silent stress emergency.

As with arousal in the previous chapter, stress is not negative perse. It constitutes a psychological and physiological response to an environment demand perceived as challenging or threating (Lazarus & Folkman, 1984; Schneiderman et al., 2005; Selye, 1950; Selye, 1956). When adaptive it's defined as *eustress*, enhancing motivation and performance, whereas when excessive or prolonged we talk about *distress*, leading to negative consequences (Bienertova-Vasku et al., 2020)

Historically the concept of stress was introduced by Hans Selye in 1936, who defined it as "*a nonspecific physiological response to a wide range of harmful agents*". He also focused on how our body react to a prolonged in time stressor, in what he named the General Adaptation Syndrome, composed of three phases (Seyle, 1936):

1. Alarm: The immediate reaction to the stressor, involving activation of the sympathetic system and the HPA axis. This leads to the release of adrenaline, noradrenaline and cortisol producing manifestations like tachycardia, pression

increase, sweetening (Herman et al., 2016; Motta E Motta et al., 2020). This state of hypervigilance culminate in a fight or flight response (Godoy et al., 2018).

2. Resistance: If the organism it's not capable to resolve the stressor, it need to adapt to its continued presence. Stress-related systems (e.g., the HPA axis) remain activated, but at a lower, more sustainable level, while vital functions are maintained in a heightened state. Nevertheless, while continuing its regular functioning, maintaining this state of ongoing readiness comes at a biological cost. (Guidi et al., 2020). It often produces continue fatigue, tension, frustration and interfering with other systems functioning. It can last even months, and the longer this state persists, the grater the negative impact (Mariotti, 2015; Yaribeygi et al., 2017).
3. Exhaustion: Human body cannot sustain this functioning indefinitely. So when stress becomes chronic, the systems activated for too long lose efficiency. In particular, the negative feedback mechanism that regulates cortisol secretion to maintain homeostasis begin to fail, leading to hormonal imbalance (James et al., 2023; Karin et al., 2020). At this stage, physiological (and psychological, for what we know now) functioning collapses, with serious consequences.

While this model remains relevant, it was originally limited to physiological responses, overlooking psychological consequences and individual differences.

To bridge this gap, Lazarus and Folkman (1984) developed a Transactional Model of Stress, which emphasized the role of the cognitive assessment of coping resources in response to the environmental demands. According to this model, stress is a psychological process involving continuous interactions between an individual and their environment. Stress response it's determined by a specific outcome resulting from two processes: primary appraisal (1), in which individuals assess whether an event is neutral, positive, or stressful, and secondary appraisal (2), in which they evaluate availed coping resources. Stress arises when environment demands are perceived as exceeding coping abilities (Lazarus & Folkman, 1984). In this view, stress is primely a psychological process, that has also a physiological impact and, at the same time, it explains why the same event may be stressful for one person but not for another.

Building on these frameworks, McEwen (1998) provided a more integrative perspective by emphasizing the interplay among the involved systems: the immune

system, the ANS, and the HPA axis. He introduced the concept of allostatic response: the organism it's not simply capable of returning to a homeostatic baseline after stress, but instead engages in allostasis, a process of achieving stability through change. The multiple physiological systems involved in allostasis adjust continuously to meet the perceived environmental demands. When stress is manageable, this response is adaptive. However, when stressors are chronic, repeated, or unresolved, this repeated activation accumulates as allostatic load, producing long-term negative effects (Juster et al., 2010; McEwen, 1998).

Most recently, Mueller and colleagues (2022), deepened the role and relationship of biological system involved in stress regulation. They suggested that stress reactions involved multiple biological systems operating at different levels (from molecular to neural networks) modulating by time and context and prior experiences. This means the biological stress response can vary greatly across individuals and situations (Mueller et al., 2022). According to their model, stress is best understood as a multifaceted, dynamic process that involves reciprocal communication between central neuronal regulatory systems (e.g., prefrontal cortex, amygdala, and hippocampus) and peripheral physiological systems (e.g., HPA axis, ANS, and immune system). In this view, psychological appraisals can directly influence physiological processes like inflammation, cardiovascular reactivity, and endocrine function, and in the same way physiological alterations can, in turn, shape mood, cognition, and behaviour (Mueller et al., 2022).

As we have stressed here, many are the systems negatively affected by failed stress management, and sleep is one of the most affected, given its strong and bidirectional relationship with stress, which we will in deep in the following chapters.

3.2. Sleep Response to Stress: the Role of Sleep Reactivity

3.2.1. Same Stressor, Different Reactions

About 20 years ago, a new line of research aimed to investigate and define the role of stress in sleep regulation began to spread, gradually gaining importance in studying ID. In 2006, Charles Drake and Thomas Roth, analysing ID aetiology, drew attention on the role of stressful events and posed a key question: why can the same stressor induce sleep disturbances in one individual but not in another? Within the 3P Model framework, they concluded that this variability must be dependent on individual predisposing factors, particularly the pre-existing tendency of an individual to be affected by a stressful event and in which measure. In this context, they introduced the concept of Sleep Reactivity (SR), a trait-like characteristic that determine the degree to which the sleep system tend to be perturbated by the introduction of a stressor (Drake et al., 2011; Drake & Roth, 2006). To illustrate this construct, Kalmbach propose an example (Figure 2) that is worth quoting. Let us consider two individuals, Mark and Matt, with very similar lifestyle, sleep habits and same chronotype. They both work at the same company, which is doing staff cuts, and they both lose their jobs. Dismissal is commonly a very stressful event, indeed, aside of the emotional consequences, both man struggle with initiating and maintaining sleep in the days immediately following the event. However, as the time goes by, Mark quickly returns to his usual sleep schedule, while Matt's sleep difficulties worsen. We can deduce that Mark has a resilient sleep system: it is affected by the stressor, moderately and for a limited duration, so he easily returns to baseline. Thus, Mark has a low SR. Matt's sleep system, on the other hand, it's more fragile and vulnerable to stressors; their impact it's greater and persists over time. In this picture, Matt has high SR and is clearly at higher risk of develop ID (Kalmbach et al., 2018a).

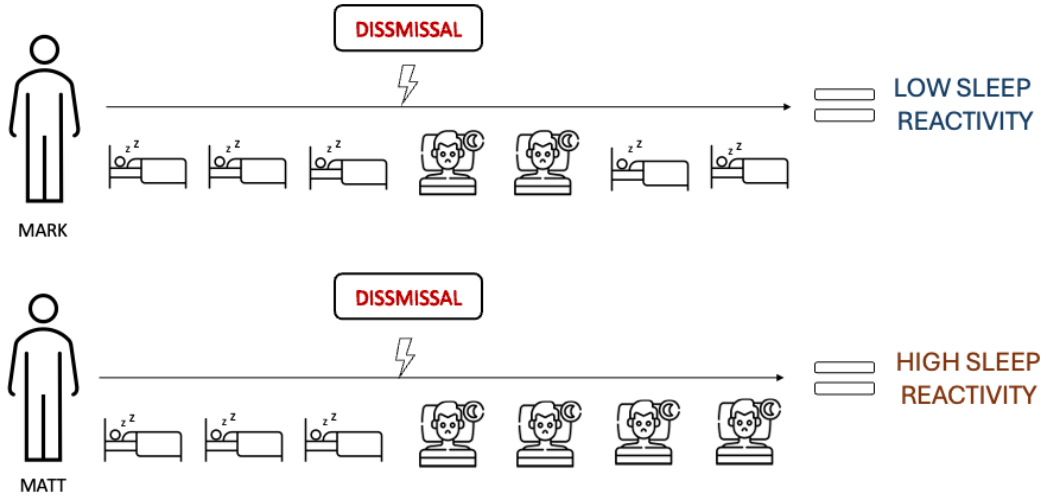


Figure 2. *Schematic representation of how two individuals with different levels of SR react to the same stressor.*

This example makes it clear that people with high levels of SR are at greater risk of sleep disorders. Given its trait-like nature, SR tends remains stable across different situations and over time, making it a predisposing factor throughout the individual's life. The stability of this characteristic has been measured with the Ford Insomnia Response to Stress Test (FIRST), a questionnaire designed to assess individual's predisposition to experience sleep disturbances following a variety of common stressful life situations (Drake et al., 2004). It's considered a valid tool to measure SR. Indeed, higher scores on the questionnaire correlate with a stronger *first-night effect*[3], in terms of decreased Sleep Efficiency (SE%) and increased SL. In the same study, subjects also underwent an MSLT protocol, and those in the high SR group based on FIRST, had significantly longer SL during naps (Drake et al., 2004). The FIRST stability has been tested over time. In a sample of healthy adults, SR was shown to remain stable across two six-month follow-ups (Jarrin et al., 2016). Moreover, compare to other traits, SR not seems to change with age: in a study analysing three different age groups in ID patients, FIRST score did not

[3] The first-night effect is a well-known phenomenon that occurs when subjects sleep in an unfamiliar environment for the first time (e.g., in a sleep laboratory). It is common to experience sleep disturbances as a consequence of being in a non-familiar setting.

differ between groups while, whereas hyperarousal, measured with the Arousal Predisposition Scale (APS), showed a pattern of decline with age (Altena et al., 2016).

Nevertheless, important evidence suggests that SR can be modulated through exposure to stressful situations or through the onset of ID itself. Indeed, in a sample of healthy subjects has been found that SR increased in individuals who later developed ID: 68% of those with low baseline SR were reclassified as high SR at the follow-up. Moreover, the presence of major life stressors was associated with increases in SR, as well. The authors named that phenomenon "*sleep system sensitization*", suggesting that SR is not such a crystalized phenomenon (Kalmbach et al., 2016a). This finding is consistent with stress research showing that repeated exposure reduces the ability to regulate stress in general (McEwan, 2000).

Individual differences play a role in influencing SR. Women, for example, show significantly higher SR compared to men, pointing to possible gender-related vulnerabilities (Drake et al., 2011). SR has also been associated with psychological traits, such as neuroticism, the Type D personality profile, also named *distressed* (Uygur et al., 2023) and lower resilience (Palagini et al., 2018). These findings are consistent with the results regarding vulnerability factors in ID discussed in the previous chapter. We have already underlined the genetic component of ID, and evidence indicates that SR is a moderately heritable trait as well, with influences that substantially overlap with ID (Drake et al., 2011; Lind & Gehrman, 2016). Interestingly, hereditability rates vary by gender (Female: 29%; Male: 43%) (Drake et al., 2011).

It's important to emphasise that SR should not be considered something pathological per se. It's a universal characteristic that became problematic only above a certain threshold. Below this level, SR may be considered adaptive, as heightened vigilance in response to stress could have been protective in evolutionary terms, placing SR on a continuum from physiological to pathological. As illustrated in Figure 3, it is normal to experience moderate, short-lived sleep disturbances after stress; risk arises when sleep disturbances are greater in severity and duration and persist after the stressor has been removed (Kalmbach et al., 2018a). Consistently in a longitudinal study lasted three years, individuals with high SR were nearly 60% more likely to develop ID symptoms and were twice as likely to develop chronic ID within the following two years compared to low-reactive sleepers. This relationship was independent of prior sleep disorders, presence of

depressive symptoms, and stress exposure, and remained significant even after controlling for APS score. These results support SR as a sleep-specific vulnerability that is distinct from general trait hypervariability (Jarrin et al., 2014).

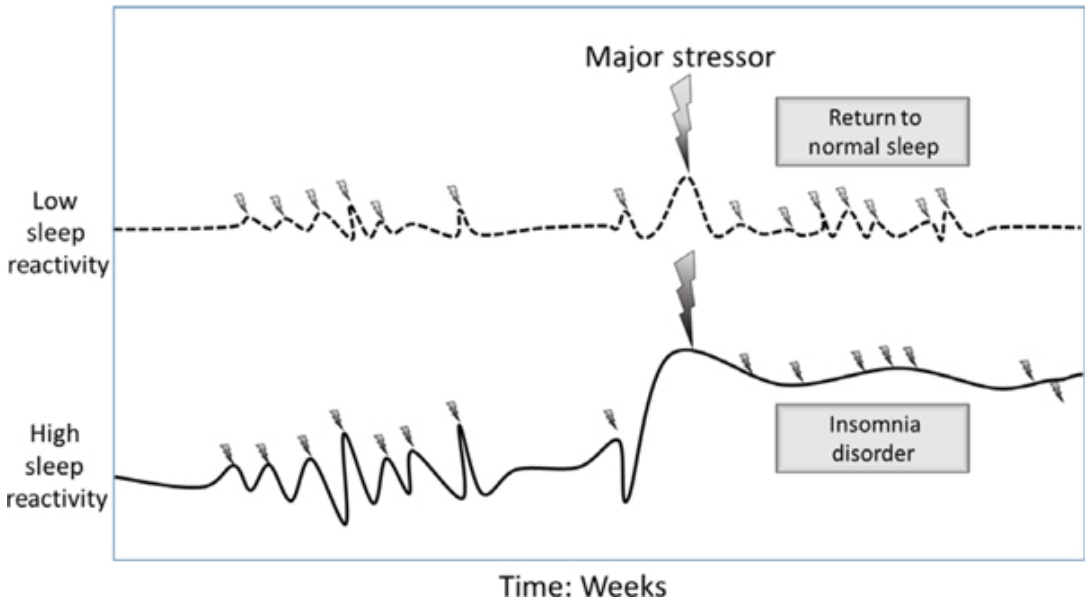


Figure 3. *Schematic representation of the different impact of stress on sleep system in individuals with different levels of* SR *(Source: Kalmbach et al. 2018)*

High SR seems to be associated with other common characteristics of the ID profile. Fernández-Mendoza and colleagues, for example, found no differences between healthy individuals with high SR and data reported in the literature on ID patients in terms of cognitive and somatic hyperarousal, ruminative thinking and presence of emotion-oriented and coping strategies (Fernández-Mendoza et al., 2010). Other studies have reported associations between FIRST score and the depressive symptoms (Nakajima et al., 2017; Vargas et al., 2020) as well as anxiety (Okajima et al., 2023). Both are well known to be strongly linked to ID and its severity.

This suggests that SR may interact with trait hyperarousal in determining vulnerability to ID: in individuals with high SR, the presence of a hyperarousal profile shapes their emotional and cognitive response to stressors, thereby increasing the likelihood of experiencing sleep disturbances (see Figure 4 for a schematic overview of these interactions).

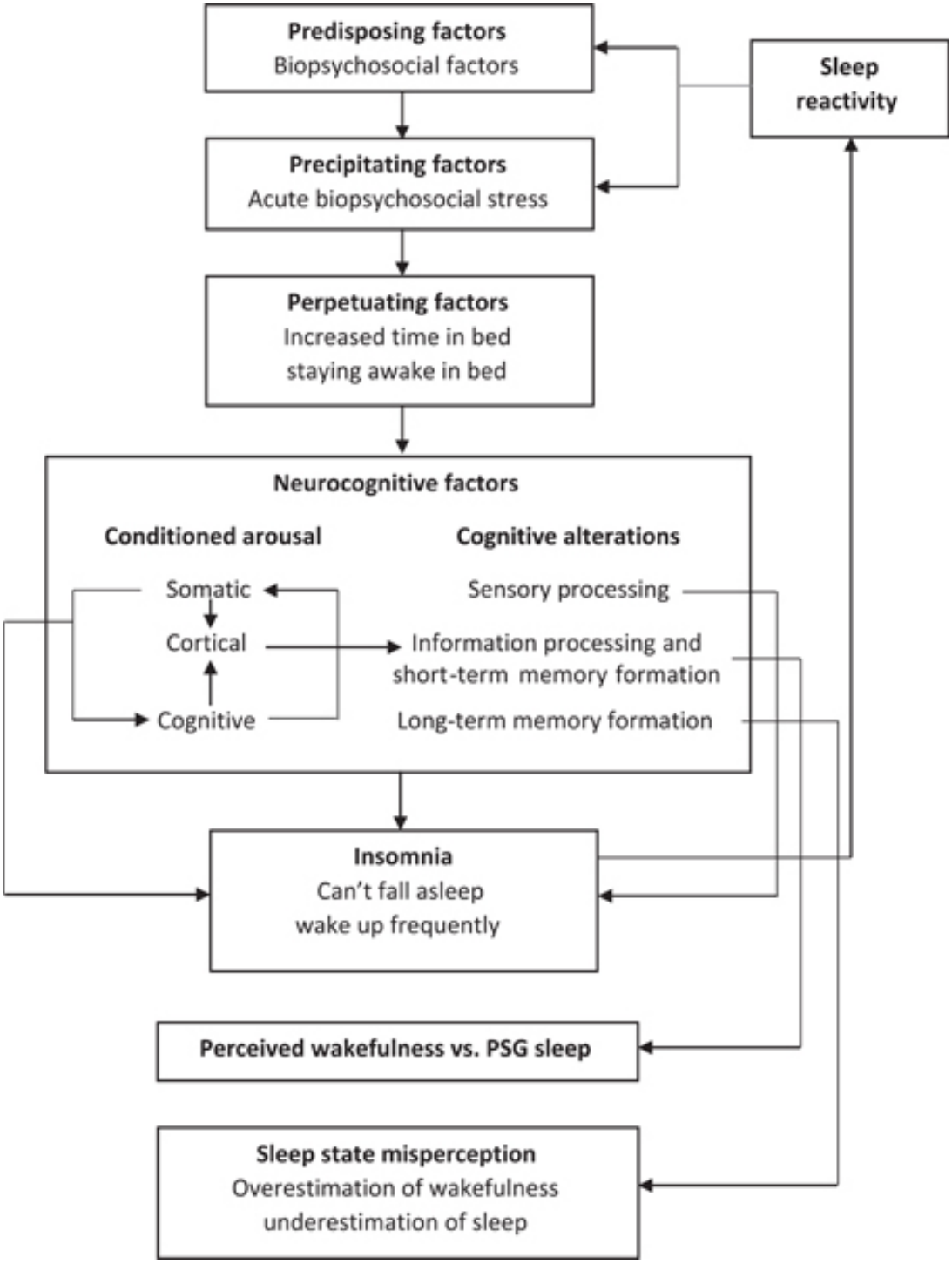


Figure 4. *Schematic representation of the complex interaction between SR, ID and hyperarousal, together with their consequences. PSG = polysomnography (Source: Kalmbach et al. 2018)*

In summary, analysing SR is highly relevant in the context of ID: SR not only enhances our understanding of the mechanisms underlying ID but also has clinical importance, as

it enables the early identification of individuals at risk and opens the way for preventive interventions.

3.2.2. How Stress Impact on the Sleep System

Stress can disrupt sleep through two primary pathways: (1) by directly altering physiological systems involved in sleep regulation, and (2) by influencing cognitive and emotional processes that interfere with the ability to initiate and maintain sleep.

The natural alternation of wake and sleep is regulated by the suprachiasmatic nucleus of the anterior hypothalamus, which also influences sleep phases through its direct projections to the paraventricular nucleus (PVN). The PVN, by acting on the HPA axis, modulates cortisol secretion patterns. Cortisol levels follow a circadian rhythm, with a minimum during the early night and a peak in the morning (Buijs & Kalsbeek, 2001). Exposure to a stressor alters PVN plasticity, reducing inhibitory control and enhancing corticotropin-releasing hormone (CRH) excitability. This sustaining HPA axis hyperactivity and disrupts biological rhythms (Herman et al, 2016). Consequently, increased cortisol secretion interferes the normal regulation of sleep.

Experimental studies have clarified how dysregulated cortisol affects sleep. Artificially increasing cortisol levels allowed us to know how the dysregulation of this hormone impact on sleep. Specifically, elevated cortisol has been associated with increased SL, a higher number of awakenings, and consequently a greater amount of wake after sleep onset (WASO) (Vazquez-Palacios & Velázquez-Moctezuma, 2000; Vazquez-Palacios et al., 2001). At the sleep architecture level, it has also been linked to a reduction in REM%, increased REM latency (Demiralay et al., 2014; Szmyd et al., 2021), and a decrease in slow-wave sleep (SWS) (Born et al., 1991; Hirotsu et al., 2015). Consistent with these evidence, subjects with high SR exhibit a greater number of awakenings and a more frequent transition between stages (Kalmbach et al., 2018a).

Moreover, a recent study by Feng and colleagues investigated the impact of stress on healthy subjects divided in two groups based on their level of SR. In both high and low SR groups, stress impaired sleep, with reduced total sleep time (TST), longer SL, increased REM latency, and reduced REM%, but no changes in stage N3 sleep. However, in the high-SR group stress produced more pronounced effects, including greater WASO, longer SL and REM latency, reduced REM, and lower sleep efficiency (SE%) (Feng et

al., 2023). These findings confirm that stress disrupts sleep in all individuals, but the impact is more severe in those with high SR.

In the previous chapter we have briefly mentioned the fact that SR is strongly related also to psychological processes. Fernández-Mendoza and colleagues, studying healthy individuals across two age group, found that in middle-aged adults, high SR was associated with APS score, neuroticism, perceived stress, and rumination, whereas in young adults it was additionally linked to pre-sleep cognitive and somatic arousal as well as emotion-oriented coping. Notably, highly reactive good sleepers did not significantly differ from clinical insomniacs on measures such as arousability, rumination, pre-sleep arousal, and neuroticism (Fernández-Mendoza et al., 2010).

These findings, together with the physiological evidence, help us build a more complete picture of how SR shapes the sleep system's response to a stressor. From the last study mentioned, SR was associated with markers of cognitive and emotional hyperarousal, suggesting that SR and cognitive–emotional reactivity are distinct yet interrelated processes, linked through a bidirectional relationship. High SR often leads individuals to adopt coping strategies to manage the strong negative impact of stressful events. However, these strategies, such as rumination, are typically ineffective and instead make it harder to fall asleep (Kalmbach et al., 2016b). In turn, these sleep difficulties further reinforce the use of such strategies, setting in motion a cycle that is difficult to break.

Stress-related content can interfere not only with sleep onset but also during dreaming activity, leading to increased awakenings (Nielsen & Levit, 2007). Dreaming activity is highly associated with REM sleep, with about an 80% probability of reporting mental content during REM awakenings in serial awakening paradigms (Martin et al., 2020). It's well established, indeed, that REM sleep plays a crucial role in emotional regulation. During REM sleep, limbic and paralimbic structures, involved in emotional elaboration and memory formation, are highly activated (Braun et al., 1997; Luppi et al., 2017; Nofzinger, 2005). Evidence from the literature support an association between REM% and difficulties in emotional regulation after sleep. In REM restriction/suppression paradigms, authors found a correlation with enhanced emotional reactivity and sustained activation of limbic regions (Rosales-Lagarde et al., 2012) as well as increased negative affect, heightened amygdala responses, and altered amygdala-anterior cingulate cortex

connectivity during social exclusion tasks (Glosemeyer et al. 2020). Another, more ecological, example of this effect has been obtained using a nap paradigm: only participants who obtained REM during the nap showed a recalibration of emotional reactivity, with reduced sensitivity to angry and fearful faces and enhanced ratings of happy expressions at a facial emotion recognition task, whereas those without REM or kept awake did not display such changes (Gujar et al., 2011).

Emotional dysregulation has been observed in ID patients (Galbiati et al., 2020; Palagini et al., 2017) as well as interconnected REM abnormalities (e.g., increase fragmentation, numbers of awakenings, and arousals, named *REM instability*) (Riemann et al., 2012). These alterations likely contribute both to maintaining hyperarousal and to explaining the strong association between ID and risk of depression (Baglioni et al., 2011). In line with this view, Petersen and colleagues reported that experiencing an high-stress periods reduced REM sleep only in individuals with high SR, whereas resilient participants even showed a slight increase in REM, further supporting the notion that REM sleep is centrally involved in the regulation and downscaling of emotional intensity to restore affective balance (Petersen et al., 2013; Vandekerckhove & Cluydts, 2010). We can hypothesise that, in a stress-vulnerable subject, stress exposure leads to a compromised functioning of the physiological process of negative affective resolution. This dysfunction, objectifiable in REM instability, results in emotional dysregulation, which in turn contributes to hyperarousal. The consequence is a worsening and perpetuation of ID symptomatology.

3.3. How Stress Shapes Sleep? A Systematic Review on Sleep Reactivity

Despite the growing theoretical and clinical relevance of SR, the empirical base remains fragmented. As we have just seen, much of the literature relies on self-report measures, as the FIRST. Although valuable, these assessments must be complemented by experimental studies: specifically, paradigms that induce stress and assess sleep before and after, in order to clarify the dynamic and causal nature of the stress–sleep interaction.

This represents the core of the experimental design of this doctoral project. To build the most effective design for inducing stress and evaluating its impact on sleep, a systematic review of the literature was conducted, with the aim of critically examining

research that has used ecological and experimental paradigms to investigate the effects of stress on the sleep system. By focusing on studies that include a measurable stressor, objective and subjective sleep assessment both before and after exposure, our objective is to address core questions: Do stressors alter sleep in predictable ways? Are specific sleep domains or stages more susceptible to disruption? And how do individual differences, in vulnerability, resilience, and stressor type, shape these outcomes?

3.3.1 Eligibility Criteria

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure transparency and methodological rigor (Page et al., 2021).

Studies were selected according to predefined inclusion and exclusion criteria designed to identify research that experimentally or observationally assessed SR in the context of a stressor using pre-post designs. To be included, studies had to: (1) be published in English; (2) involve a within-subject pre/post design, either observational or experimental; (3) assess sleep using at least one electrophysiological/objective measure (e.g., EEG, polysomnography [PSG], or actigraphy [ACT]); and (4) include healthy adult participants without diagnosed psychiatric, neurological, or sleep disorders.

Exclusion criteria were as follows: (1) reviews, meta-analyses, or theoretical articles; (2) grey literature, including case reports, letters, commentaries, conference abstracts, or book chapters; and (3) studies without an available abstract for screening.

3.3.2 Information Sources and Search Strategy

A comprehensive literature search was conducted across four electronic databases: PubMed, Scopus, Embase, and Web of Science, covering all available records up to December 2024. The search strategy was developed to capture studies assessing SR in response to stress using objective sleep measures. The following Boolean search string was used across all databases, with syntax adjusted as necessary:

((sleep reactivity) OR (sleep system sensitivity) OR (vulnerability to stress) OR (vulnerability to stress related insomnia) OR (stress related insomnia) OR (intrusive memor) OR (trauma film paradigm) OR (insomnia)) AND ((electroenceph) OR (polysomnograph*) OR (actigraph*) OR (EEG) OR (PSG))*

All records retrieved were imported into a reference management system for de-duplication and screening. No restrictions were applied to study design at this stage, although inclusion and exclusion criteria were applied during the screening phase.

3.3.3 Study Selection Process

Study selection was carried out in two phases by a team of three independent reviewers, including two PhD students and one post-graduate research trainee, all trained in systematic review methodology. In the first phase, titles and abstracts were screened for relevance according to the predefined eligibility criteria. In the second phase, the full texts of potentially eligible articles were retrieved and assessed for inclusion. To facilitate initial screening and manage duplicate records, the reference management platform Rayyan was used (Ouzzani et al., 2016). Any disagreements regarding study eligibility were resolved through discussion among the reviewers until consensus was reached. Studies that met all inclusion criteria were then retained for data extraction and quality assessment.

Exclusion criteria included:

- Abstract not available: Studies without an accessible abstract were excluded because they could not be reliably screened for relevance against the eligibility criteria.
- Grey literature: Unpublished or non-peer-reviewed sources (e.g., case reports, letters, commentaries, conference abstracts, dissertations, book chapters) were excluded to ensure methodological rigor and comparability of evidence.
- Language: Studies published in languages other than English were excluded.
- Meta-analyses / reviews: Secondary sources such as systematic reviews, meta-analyses, and theoretical or narrative reviews were excluded because the focus of this work was on original empirical data.

- Outcome: Studies that did not include sleep as an outcome variable, or that relied exclusively on subjective measures without objective assessment (i.e., PSG, EEG, actigraphy), were excluded.
- Participants: Studies involving clinical populations or non-adult samples, as well as animal models, were excluded in order to focus on healthy adult participants.
- Study design: Studies that did not employ a within-subject pre–post design to assess the impact of a stressor on sleep were excluded, as they did not allow for direct evaluation of stress-induced changes in sleep.

The search until December 2024 provided 31567 studies (Embase: 8310; Pubmed: 7440; Scopus: 10060; Web of Science: 5757). Using Ryyan semi-automatic system to detach duplicates 20871 results have been removed, for a total of 10696 to be screened. In the first screening we have excluded 10595 studies, and in the second one 92, for a total of nine studies included in the systematic review. Figure 5 shows the selection procedure in detail.

3.3.4 Data Collection Process

Data extraction was carried out in parallel by all three reviewers using a standardized approach to ensure accuracy and completeness. After independently extracting information, the reviewers compared results and resolved discrepancies through consensus discussion. For each included study, the following data items were extracted: sample size, gender distribution, mean age, description of the healthy adult population, and any inclusion or exclusion criteria applied (Table 1). Study design was categorized as experimental or observational. Detailed information was collected on the type of stressor employed, the timing of stressor delivery in relation to the sleep assessment, and whether and how the effectiveness of the stressor was verified independently of sleep outcomes. Finally, sleep assessment methods were recorded, including objective measures (e.g., EEG, PSG, ACT) and subjective self-report tools, along with specific pre- and post-stressor sleep evaluation timepoints.

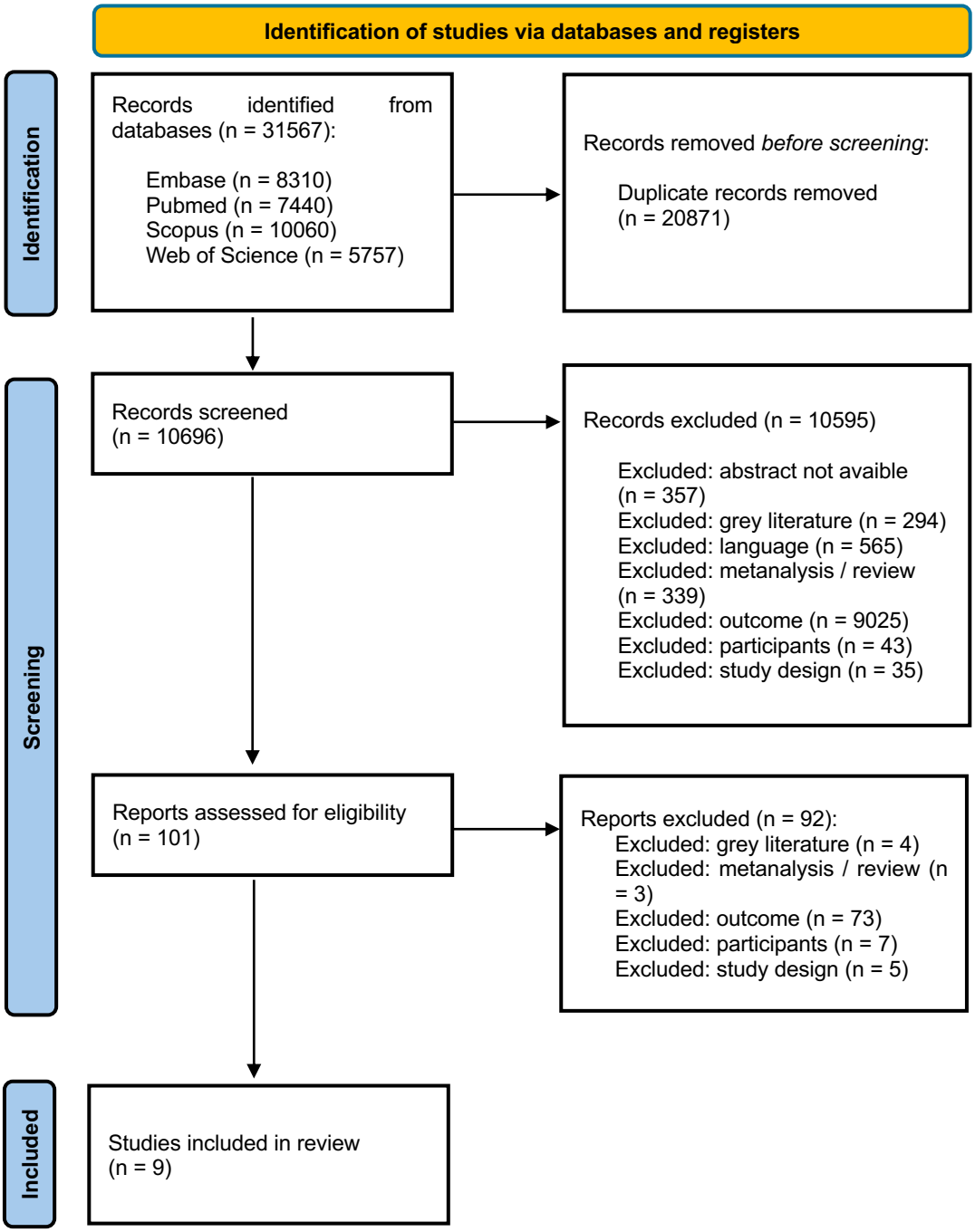


Figure 5. *Study selection flow diagram, using PRISMA 2020 flowchart (Page et al., 2021).*

Study	Study Design	N	Age (m ± st.dev)	Sex (M/F)	Population	Exclusion Criteria
Torsvall & Åkerstedt, 1988	Observational	5	25.0 – 42.0*	5/0	Ship engineers	None
Bader et al., 2010	Experimental	39	24.6 ± 5.2	0/39	University students	None
Vandekerckhove et al., 2011	Experimental	13	32.4 ± 12.2	6/7	Healthy adults	Sleep disturbances; psychiatric disorders; medication usage affecting sleep; psychotherapy; pregnancy; shift-working; high alcohol or caffeine intake; smoking
Petersen et al., 2013	Observational	28	41.0 ± 9.0	7/21	Teachers	ID symptoms or any other sleep disorder; depression or stress related disorders
Wuyts et al., 2012	Experimental	16	23.9 ± 0.8	10/6	Healthy adults	Medical condition affecting sleep; psychologist students; smoke, alcohol or substance abuse; PSQI > 5; SL baseline night > 30min
Talamini et al., 2013	Experimental	32	20.1 ± 1.5	9/23	Healthy adults	Prior history of sleep disorders; neurological or psychiatric disorders; habitual sleep length < 7h; knowing the stressing film
de Zambotti et al., 2015	Experimental	18	48.5 ± 2.3	0/18	Perimenopausal women	Use of hormone therapy; severe medical conditions; sleep medication or antidepressant therapy; sleep disorders
Gehrman et al., 2016	Experimental	19	36.3 ± 8.4	NA	Healthy adults	Neurological or psychiatric disorders; shift working; sleep disorders; tobacco usage; medication usage affecting sleep
Esquivel-Mendoza et al., 2023	Observational	18	38.8 ± 11.8	7/11	Healthy adults	High risk or presence of sleep disorders; presence of a medical condition affecting glucose; history of cognitive or neurological disorders; shift working; experiencing jet leg in the two previous months; alcohol or substance abuse; high caffeine intake; being on diet; medication usage affecting sleep; severe medical conditions; tobacco usage

Table 1. *Studies characteristics.*
N = sample size; m = mean years; st.dev = standard deviation; M/F = male-female proportion; NA = not available; PSQI = Pittsburgh Sleep Quality Index; min = minutes; h = hours.
**only the age range was reported.*

3.3.5 Risk of Bias and Quality Assessment

Risk of bias was independently assessed for all included studies (n = 9) by two reviewers using a custom 12-item checklist developed specifically for this review. This tool was derived and adapted from the NIH Quality Assessment Tool for Before-After (Pre-Post) Studies With No Control Group, tailored to reflect core methodological features relevant to the SR literature (National Heart, Lung, and Blood Institute, 2025). Each criterion consisted of two guiding questions evaluating study design, sample characteristics, stressor validity, sleep assessment reliability, and statistical approach. Responses were coded as Yes, No, or Other (CD = Cannot Determine; NR = Not Reported; NA = Not Applicable).

Reviewers assessed all studies independently and resolved discrepancies through discussion until full consensus was achieved. Inter-rater agreement was not quantified numerically, but procedural reliability was ensured through item-by-item comparison and reconciliation.

To facilitate interpretation, a categorical rating system was applied to summarize overall study quality:

- Studies scoring 11–12 out of 12 were rated as “Good” quality (≥91%),
- Scores of 8–10 were rated as “Fair” quality (66–83%),
- Scores of 7 or lower were rated as “Poor” quality (≤58%).

Of the nine included studies, five were classified as Good quality, and four as Fair quality. No study was classified as Poor quality. The most common limitation across studies was the absence of formal sample size justification or power analysis, and in some naturalistic studies, lack of independent stressor verification. A full summary of quality ratings by criterion and study is provided in Table 2.

Study	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	C11	C12	Tot	%	Quality
Torsvall & Åkerstedt, 1988	yes	no	no	no	yes	yes	yes	yes	yes	yes	no	yes	8	66.7	Fair
Bader et al., 2010	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	yes	yes	11	91.7	Good
Vandekerckhove et al., 2011	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	yes	yes	11	91.7	Good
Petersen et al., 2013	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	no	yes	10	83.3	Fair
Wuyts et al., 2012	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	yes	yes	11	91.7	Good
Talamini et al., 2013	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	yes	yes	11	91.7	Good
de Zambotti et al., 2015	yes	yes	yes	no	yes	yes	yes	yes	yes	yes	yes	yes	11	91.7	Good
Gehrman et al., 2016	yes	yes	no	no	yes	no	yes	yes	yes	yes	yes	yes	9	75.0	Fair
Esquivel-Mendoza et al., 2023	yes	yes	yes	no	no	no	yes	yes	yes	yes	yes	yes	9	75.0	Fair

Table 2. *Studies quality assessment results.*
Tot = Total score: yes, the criteria was satisfied = 1, no, the criteria wasn't satisfied = 0;
C1 = Criteria 1. Did the study clearly define its aim to assess sleep changes following a stressor?
C2 = Criteria 2. Did the authors define inclusion/exclusion criteria?
C3 = Criteria 3. Was the sample appropriate to generalize to healthy adults without clinical sleep disorders?
C4 = Criteria 4. Did the study provide a rationale for the number of participants included?
C5 = Criteria 5. Was the type, intensity, and timing of the stressor in relation to sleep clearly reported and standardized?
C6 = Criteria 6. Did the study confirm that the stressor induced stress, beyond just assuming it?
C7 = Criteria 7. Were standardized tools (e.g., PSG, ACT, validated questionnaires) used to measure sleep?
C8 = Criteria 8. Did the timing of measurement align with the expected window of sleep reactivity?
C9 = Criteria 9. Was the same device/questionnaire/protocol applied consistently before and after the stressor?
C10 = Criteria 10. . Was participant dropout documented, and were reasons provided?
C11 = Criteria 11. Did the study account for individual differences that might influence sleep reactivity?
C12 = Criteria 12. Did the study use appropriate paired/comparative statistics for pre-post analysis?

3.3.6 Data Synthesis

Given the methodological heterogeneity across studies in terms of stressor type, sleep assessment measures, and sample characteristics, a narrative synthesis approach was adopted. Findings were summarized according to stressor paradigm, changes in objective and subjective sleep outcomes, and potential moderating factors. No meta-analysis was conducted due to insufficient accordance and harmonization of outcomes.

3.3.7 Studies Characteristics

A total of nine studies met inclusion criteria, all employing a within-subject pre-post design to examine the impact of a stressor on subsequent sleep. The studies spanned both experimental (n = 6) and naturalistic/observational (n = 3) designs. Sample sizes ranged from 5 to 40 participants, with a combined total of approximately 230 individuals, predominantly healthy young or middle-aged adults. One study focused on a clinical ID group (Gehrman et al., 2016), but we will consider only the results related to the healthy control group and another on perimenopausal women (de Zambotti et al., 2015), while the rest recruited healthy good sleepers.

Stressors included varied widely and were grouped into two major categories:

1. Psychosocial and emotional stressors, including adapted Trier Social Stress Test (de Zambotti et al., 2015; Gehrman et al., 2016), failure feedback tasks (Vandekerckhove et al., 2011), and emotionally distressing film clips (Bader et al., 2010; Talamini et al., 2013);
2. Real-world stressors, such as academic workload during high-stress periods (Petersen et al., 2013) and the COVID-19 pandemic context (Esquivel-Mendoza et al., 2023) and on-call paradigms, including real or simulated night-time availability for work-related tasks (Torsvall & Åkerstedt, 1988; Wuyts et al., 2012).

All studies included objective sleep measures (PSG, EEG, or actigraphy), and six out of ten articles also reported subjective sleep quality via Sleep Diaries or questionnaires.

3.3.8 Type of Stressors Used

The included studies employed a range of stressor paradigms. These paradigms differed in ecological validity, timing relative to sleep, and level of experimental control.

Psychosocial and emotional stressors:

Five studies (Bader et al., 2010; Gehrman et al., 2016; Talamini et al., 2013; Vandekerckhove et al., 2011; de Zambotti et al., 2015) used laboratory-based emotional or psychosocial challenges. These included:

- Emotional film clips shown before bedtime (Bader et al., 2010; Talamini et al., 2013). Bader and colleagues (2010) presented five 5-minute documentary videos in a row, portraying different people telling their experiences with very stressful events, and compared this condition to a night without video viewing. Talamini and colleagues (2013) used a clip from the movie The Passion of the Christ (Mel Gibson, 2004) as the stressful stimulus compared to a clip from March of the Penguins (Luc Jacquet, 2005) as the neutral stimulus.
- A failure-based cognitive task. Participants were told that the test would measure their intelligence. It consisted of six computerized cognitive subtasks (e.g., a visual-spatial task, memory tasks, and an unsolvable semantic reasoning task). After each subtask, participants received on-screen feedback showing extremely low scores, regardless of their actual performance. During the procedure, the experimenter made irritated comments about the participants' behaviour, and at the end of the session they were told that the physiological data collected were "useless," reinforcing the perception of personal failure (Vandekerckhove et al., 2011).
- An adapted Trier Social Stress Test (TSST) with social evaluation. In this paradigm, participants were instructed to prepare a short presentation to deliver the morning after the PSG recording in front of a formal evaluation committee, with both video and audio recording. However, they were not informed of the presentation topic until the following morning. After the presentation, the committee evaluated and questioned them, followed by a five-minute arithmetic task (de Zambotti et al., 2015).
- A threat of electric shock that induce a state of anticipation of a negative event. At the beginning participants received one mild shock and were told they might

receive up to three shocks during their sleep, but no further shocks were delivered (Gehrman et al., 2016).

These stressors were delivered within two hours prior to sleep onset and were followed by PSG or actigraphy. All these studies verified the effectiveness of stress induction using subjective ratings or physiological measures.

Daily-life stressors:

Four studies investigated in an observational way the effects of daily-life stressors on sleep (Esquivel-Mendoza et al., 2023; Petersen et al., 2013; Torsvall & Åkerstedt, 1988; Wuyts et al., 2012). Two of them adopted an ecological observational paradigm, one tested the effects of heightened, compared to regular, academic workload (Petersen et al., 2013) and other studies focused on the impact of COVID-19 pandemic (Esquivel-Mendoza et al., 2023). Such paradigms offered high ecological validity, reflecting stressors encountered in daily-life. However, they generally lacked experimental control or direct manipulation of the stressor. Instead, stress was inferred based on contextual factors and verified using tools such as self-report diaries, workload assessments, or mood questionnaires. Although these designs limited causal inference, they provided valuable insight into how real-life stress experiences may influence sleep parameters.

The other two studies used on-call paradigm in which duty instructions served as a stressor, simulating anticipatory arousal during sleep. Torsvall and Åkerstedt observed sleep on real ships with participants subject to potential night alarms (Torsvall & Åkerstedt, 1988), whereas Wuyts et al. implemented a simulated on-call condition in the lab with no actual awakening events (Wuyts et al., 2012). In both cases, sleep was assessed via PSG, and stress was operationalized as the expectation of being awakened. In this way, the studies attempted to mediate between examining a realistic phenomenon and employing a controlled, circumscribed paradigm that allowed for confound control.

3.3.9 Effects on Objective Sleep Parameters

Across the included studies, objective sleep was primarily assessed using PSG, with two studies employing ACT (Bader et al., 2010; Esquivel-Mendoza et al., 2023) as a less invasive alternative. Notably, Torsvall and Åkerstedt did not use a full PSG setup but instead recorded EEG, electrooculography (EOG), and electrocardiogram (ECG) without

electromyography (EMG), limiting full sleep staging capabilities (Torsvall & Åkerstedt, 1988). Most PSG/EEG-based studies adopted a 1-night pre vs. 1-night post design, excluding any screening or habituation nights from analysis (Torsvall & Åkerstedt, 1988; Petersen et al., 2013; Wuyts et al., 2012; Talamini et al., 2013; de Zambotti et al., 2015; Gehrman et al., 2016). An exception was Vandekerckhove and colleagues, who used two pre- and two post-stressor nights, including a screening night (Vandekerckhove et al., 2011).

Among the ACT studies, protocols varied: Bader and colleagues compared three nights of baseline to a single stressor night, while Esquivel-Mendoza and colleagues conducted a longer-term comparison spanning seven nights pre- and post-stressor (Bader et al., 2010; Esquivel-Mendoza et al., 2023).

Key objective sleep parameters included SL, WASO, SE%, SWS and REM%, spectral EEG features, and autonomic indices such as overnight HR. A summary of the main objective sleep outcomes by study is provided in Table 3.

SE% and WASO were among the most consistently altered parameters. Significant reductions in SE% and increases in WASO were found following stress exposure in multiple studies and these effects were especially marked in laboratory-induced stress and on-call conditions (Vandekerckhove et al., 2011; Wuyts et al., 2012; Esquivel-Mendoza et al., 2023). Petersen and colleagues also reported a significant SE% reduction during high-stress periods but only in the subgroup of individuals with high SR (Petersen et al., 2013). In one other study a numerical trend in the same direction, though without statistical significance has been observed (Gehrman et al., 2016).

Stress was also associated with alterations in sleep initiation, as reflected in changes in sleep latency (SL). A significant increase in SL was observed in the experimental study by Wuyts and colleagues (2012), whereas other studies reported increases in SL that did not reach statistical significance (Torsvall and Åkerstedt, 1988; Petersen et al., 2013; Gehrman et al., 2016). Notably, Petersen's data suggested that the direction and significance of SL changes varied according to individual SR levels, highlighting interindividual differences in predisposing factors. Specifically, participants with high sleep reactivity showed a non-significant increase in SL during the high-stress period, whereas those with low sleep reactivity exhibited a non-significant decrease (Petersen et al., 2013). Overall, these findings indicate that stress-related effects on sleep initiation are

variable and may be moderated by individual vulnerability factors rather than representing a uniform effect.

Regarding TST, results were mixed: two studies reported significant reductions in TST following failure feedback and on-call duty, respectively (Vandekerckhove et al., 2011; Torsvall and Åkerstedt, 1988). Other studies found TST reductions that did not reach statistical significance (Wuyts et al., 2012; Gehrman et al., 2016).

Stress effects on sleep architecture, particularly SWS-related variables, varied between studies. Talamini and collegues found an unexpected increase in SWS% following emotional film exposure (Talamini et al., 2013), whereas Torsvall and Åkerstedt observed a decrease in SWS minutes during an on-call night (Torsvall and Åkerstedt, 1988). Vandekerckhove and colleagues reported a non-significant trend toward reduced %SWS and significantly delayed latency to SWS after stress exposure, again suggesting variability in homeostatic sleep response depending on the nature and intensity of the stressor (Vandekerckhove et al., 2011).

REM-related variables were more consistently impacted. Reductions in REM% were found consistently in three studies, indicating suppression of REM sleep across both emotional and occupational stress paradigms (Talamini et al., 2013; Vandekerckhove et al., 2011; and Torsvall and Åkerstedt 1988). Another study found significantly increased REM latency in healthy participants exposed to a laboratory stressor (De Zambotti et al., 2015), while Petersen and colleagues reported increased REM duration in high-reactivity individuals during a high academic stress period, again highlighting potential trait-like differences in stress vulnerability (Petersen et al., 2013).

Physiological and spectral data, although less frequently reported, further supported alterations in sleep quality under stress. Torsvall and Åkerstedt observed significant reductions in total EEG power density and per-minute power, suggesting lighter and more fragmented sleep (Torsvall and Åkerstedt 1988). On the other hand, De Zambotti and colleagues found no spectral differences in the healthy sleepers group but did observe significantly increased HR across stress nights, reflecting sustained autonomic arousal even in the absence of macrostructural sleep disruption (De Zambotti et al., 2015).

ACT studies generally lacked the sensitivity to detect changes in TST or other architecture parameters. Gehrman and colleagues found no significant differences in SOL, TST, SE%, or WASO in the healthy control group (Gehrman et al., 2016), while

Bader and colleagues, detected no significant changes in SE% (Bader et al.; 2010) which was also the only objective sleep parameter reported in their study. These null results may reflect methodological issues (e.g., reduced sensitivity of ACT) or sample-specific resilience to the applied stressors.

Taken together, the findings suggest that stress most reliably impacts sleep continuity and initiation (e.g., SE%, WASO, SOL), with REM-related variables also frequently affected, in line with the central role postulated in the previous chapters. Changes in SWS and EEG spectral features appeared more variable across studies, potentially reflecting differences in stressor type, measurement sensitivity, and individual SR.

Study	**Objective Sleep Measure**	**TSTmin** (m ± st.dev)		**SE%** (m ± st.dev)		**SLmin** (m ± st.dev)		**WASOmin** (m ± st.dev)		**REM%** (m ± st.dev)	
		pre	**post**	**pre**	**post**	**pre**	**post**	**pre**	**post**	**pre**	**post**
Torsvall & Åkerstedt, 1988	EEG-EOG-ECG	445.0 ± 31.3	352 ± 69.3	NA	NA	9.0 ± 8.9	10.0 ± 11.2	NA	NA	23.8 ± 0.2	21.0 ± 0.9
Bader et al., 2010	ACT	NA	NA	91.0 ± 5.0	90.0 ± 5.0	NA	NA	NA	NA	NA	NA
Vandekerckhove et al., 2011	PSG	***457.4 ± 32.6***	***422.9 ± 72.0***	***95.7 ± 4.7***	***88.8 ± 14.2***	8.8 ± 6.4	12.8 ± 14.1	***13.1 ± 6.5***	***44.4 ± 65.7***	***24.1 ± 4.9***	***18.5 ± 6.2***
Petersen et al., 2013_lSR	PSG	381.8 ± 11.3	377.4 ± 10.7	***92.5 ± 1.2***	***91.4 ± 1.6***	8.2 ± 1.4	6.0 ± 1.0	31.9 ± 5.4	36.7 ± 7.5	***23.9 ± 1.6***	***25.7 ± 1.5***
Petersen et al., 2013_hSR	PSG	441.5 ± 14.7	392.2 ± 25.1	***92.3 ± 1.8***	***88.7 ± 3.3***	8.3 ± 2.3	9.6 ± 3.3	37.6 ± 9.3	52.3 ± 9.6	***20.9 ± 2.0***	***23.5 ± 2.7***
Wuyts et al., 2012	PSG	***434.5 ± 30.3***	***422.3 ± 43.2***	***93.6 ± 4.5***	***91.3 ± 7.2***	17.0 ± 3.2	24.0 ± 6.7	***8.0 ± 2.5***	***17.2 ± 3.6***	NA	NA
Talamini et al., 2013	PSG	486.7 ± 5.1	486.4 ± 6.44	91.8 ± 0.1	90.9 ± 1.0	11.9 ± 2.5	16.4 ± 3.3	32.2 ± 21.7	34.3 ± 0.1	***19.2 ± 0.7***	***18.7 ± 0.6***
de Zambotti et al., 2015	PSG	378.5 ± 50.5	388.9 ± 54.3	87.9 ± 6.2	86.3 ± 8.8	11.2 ± 8.4	14.0 ± 11.3	68.6 ± 27	47.2 ± 34.3	19.4 ± 4.0	18.5 ± 3.1
Gehrman et al., 2016	PSG	393.4 ± 60.4	369.8 ± 79.2	85.3 ± 8.7	82.0 ± 14.8	26.2 ± 24.1	38.3 ± 45.2	66.8 ± 40	80.7 ± 54.2	21.1 ± 4.8	19.9 ± 4.2
Esquivel-Mendoza et al., 2023	ACT	423.0 ± 30.0	436.0 ± 52.0	***88.8 ± 2.4***	***84.7 ± 4.1***	NA	NA	NA	NA	NA	NA

Table 3. *Objective sleep parameters across studies.*
Notably, Petersen et al., 2013 divided their sample into healthy subjects with low (lSR) and high (hSR) levels of sleep reactivity, which are reported in separate rows.
TST = Total Sleep Time; SE% = Sleep Efficiency; SL = Sleep Latency; WASO = Wake After Sleep Onset; REM% = Rapid Eye Movement
EEG = electroencephalography; EOG = electrooculography; ECG = electrocardiogram; ACT = actigraphy; PSG = polysomnography; m = mean years; st.dev = standard deviation; min = minutes; NA = not available.
*Results reported in **bold italics** indicate that the pre–post stress comparison was significant.*

3.3.10 Effects on Subjective Sleep and Sleep Quality

Subjective sleep outcomes were reported in six of the nine included studies, using a range of self-report tools including sleep diaries, standardized questionnaires and custom rating scales (Torsvall & Åkerstedt, 1988; Bader et al., 2010; Petersen et al., 2013; Wuyts et al., 2012; Talamini et al., 2013; Esquivel-Mendoza et al., 2023). These measures captured participants' perceived sleep quality, restfulness, sleep onset difficulties, and post-sleep fatigue or recovery. A summary of subjective sleep measures and outcomes by study is provided in Table 4.

Torsvall and Åkerstedt observed clear subjective effects in their on-call paradigm. They obtained a sleep quality index combining four questions, and also measured subjective sleepiness during the day after the on-call paradigm (three times). Participants reported significantly lower sleep quality and increased next-day sleepiness on the on-call night compared to baseline. These findings were consistent with the physiological alterations observed in the same study, including reductions in total sleep time and sleep architecture quality (Torsvall & Åkerstedt, 1988).

Bader and colleagues, who used Görtelmeyer's Schlaffragebogen (SF-A) questionnaire (Görtelmeyer, 1986), found that participants exposed to a distressing film reported a reduction in perceived recovery and sleep quality score that did not reach statistical significance. However, when stressful memory reactivation was included as a covariate, the pre-post comparison on the subjective measure reached significance, indicating the importance of verifying the effectiveness of the stressor (Bader et al., 2010).

Petersen and colleagues, using the Karolinska Sleep Diary (Kaida et al., 2006), found that in both high and low SR groups, the high-stress period was associated with significantly higher ratings of stress at bedtime and greater difficulty feeling awake and not in sleep debt. However, they did not find significant differences in subjective sleep quality or daytime sleepiness following the PSG recording (Petersen et al., 2013).

Wuyts and colleagues administered a sleep diary and found consistent subjective disturbances under the on-call condition. Participants reported significantly greater WASO (both in minutes and percentage) and two near-significant trends: shorter TST and lower SE% compared to the control night. Interestingly, subjective SL was also higher, although this difference did not approach significance. These results highlight a close

correspondence between perceived and objectively measured sleep fragmentation in this real-world simulation. Other interesting results come from the administration of the Karolinska Sleepiness Scale (KSS), a questionnaire measures the subjective level of sleepiness referring to the last 5 minutes. During the baseline night, KSS scores followed their natural course: they decreased significantly in the evening compared to arrival and increased again in the morning compared to the evening before. However, under the on-call condition, no differences were found between evening and morning sleepiness, and morning KSS scores were significantly lower than at arrival (Wuyts et al., 2012).

Talamini and colleagues, using a Dutch sleep quality scale, did not find significant changes in sleep quality scores after the emotional film exposure. However, the distribution of scores following the emotional condition was bimodal, suggesting that individual differences, like trait SR, may have moderated the subjective impact of the stressor (Talamini et al., 2013).

Finally, Esquivel-Mendoza and colleagues, using the Patient-Reported Outcomes Measurement Information System (PROMIS) Sleep Disturbance questionnaire (Yu et al., 2011), reported significantly worse scores during the COVID-19 stress period relative to baseline, reflecting both increased sleep complaints and delayed sleep end times. These findings were notable given the relatively subtle changes observed in ACT recordings, underscoring the relevance of subjective appraisal in stress-related sleep disturbances (Esquivel-Mendoza et al., 2023).

Overall, subjective sleep outcomes were found to be sensitive to stress exposure in most studies, often corroborating objective alterations, particularly for sleep continuity metrics. In some cases, however, subjective deterioration appeared more pronounced than physiological disruption For instance, Torsvall & Åkerstedt and Bader did not observe any significant physiological changes, yet reported notable subjective disturbances (Torsvall & Åkerstedt, 1988; Bader et al., 2010), suggesting that cognitive-emotional processing of stress may amplify perceived sleep disturbance even in the absence of marked changes in sleep architecture.

Study	Subjective Sleep Measure	Results
Torsvall & Åkerstedt, 1988	Four-items sleep quality questionnaire and a single item sleepiness scale	On-call night compared to baseline: < Sleep quality > Sleepiness the day after
Bader et al., 2010	SF-A questionnaire	Including stressful memory reactivation as a covariate made the pre–post change in subjective sleep measures significant: decrease in both perceived quality and recovery
Vandekerckhove et al., 2011	None	
Petersen et al., 2013	Karolinska Sleep Diary	High stress period (in both groups) was associated with: > Stress at bedtime < Awakening index (reduced alertness, increase difficulty feeling rested)
Wuyts et al., 2012	Sleep Diary and KSS	On-call night compared to baseline: Significant > WASO + 2 trends: < TST < SE% Sleepier (KSS) at awakening compare to arrival
Talamini et al., 2013	Dutch sleep quality scale	No impact of the stressor on subjective sleep quality. Nevertheless, the scale followed a bimodal distribution the morning after the stressor
de Zambotti et al., 2015	None	
Gehrman et al., 2016	None	
Esquivel-Mendoza et al., 2023	PROMIS Sleep Disturbance questionnaire	Increase sleep disturbance in the high stress period

Table 4. *Subjective sleep results across studies.*
SF = Görtelmeyer's Schlaffragebogen questionnaire; KSS = Karolinska Sleepiness Scale; WASO = Wake After Sleep Onset; TST = Total Sleep Time; SE% = Sleep Efficiency; PROMIS = Patient-Reported Outcome Measurement Information System.

3.3.11 Moderating Factors

Some studies investigated individual differences that moderate how stress affects sleep, highlighting the role of trait vulnerability, cognitive-emotional reactivity, and population-specific characteristics.

Trait SR itself was the most commonly explored moderator. Petersen and colleagues, as anticipated, categorized participants based on their sensitivity to stress using a modified version of the FIRST. Only individuals with high SR showed significant stress-related reductions in SWS and alterations in REM latency during high academic workload periods (Petersen et al., 2013). Similarly, Talamini and colleagues observed that sleep quality responses to pre-sleep emotional stimuli followed a bimodal distribution: participants classified as low sleep quality responders showed decreased sleep quality

after the emotional film compared with the neutral film, whereas high sleep quality responders exhibited a slight improvement (Talamini et al., 2013).

Physiological and hormonal status was also relevant. In de Zambotti et al. (2015), healthy perimenopausal women without ID symptoms still exhibited increased sleep onset latency and reduced parasympathetic activity (HRV) following stress, suggesting that this life stage may heighten vulnerability to stress-related sleep disruption even in the absence of clinical ID (de Zambotti et al., 2015).

Demographic or biological moderators such as chronotype and age were assessed by Esquivel-Mendoza and colleagues, who found that younger participants and those with evening-type tendencies exhibited greater changes in sleep timing and rest-activity rhythms during the pandemic (Esquivel-Mendoza et al., 2023). These findings, while exploratory, suggest that circadian characteristics may shape how individuals adapt to chronic environmental stressors.

Notably, Gehrman and colleagues included both individuals with ID and healthy controls, but only the control group was considered for the present review. In this subgroup, no significant moderating effects emerged, possibly due to the absence of a robust stress response in the manipulation (Gehrman et al., 2016).

In sum, findings suggest that stable individual differences, such as trait SR, hormonal status, or circadian profile, can modulate vulnerability to stress-related sleep disturbance supporting the view of SR as a trait-like, endophenotypic marker.

3.3.12 Stressor Efficacy: External Validity of Stress Induction

To meaningfully interpret sleep changes following stress exposure, it is crucial to establish that the employed stressors effectively induced a stress response. While all studies inferred stress from their designs, only a subset incorporated independent measures, subjective or physiological, to confirm stressor efficacy. All the measure and outcomes are summarized in Table 5.

Among studies using emotional film stimuli, two studies reported significant increases in negative affect following the stressor. Bader and colleagues used Likert scales assessing mood and psychophysiological arousal, showing marked deterioration in both (Bader et al., 2010). Talamini employed the Profile Of Mood Scale (POMS) (McNair et al., 1971) and a valence visual analog scale (vVAS) (Watson et al., 1988), with results

indicating a clear post-film rise in negative mood (in particular the response to the first film was different compare to the second in terms of depression, anger and tension/anxiety), the day after the video viewing (Talamini et al., 2013). These findings support the reliability of emotional film clips in inducing affective arousal prior to sleep, consistent with research on the general stress-inducing capacity of the Trauma Film Paradigm (TFP) (Holmes & Bourne, 2008).

Psychosocial challenges, such as failure tasks or public speaking scenarios, were also consistently validated. Vandekerckhove and colleagues used the Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988) to assess emotional reactivity and found increased negative affect immediately after the task and even the following morning, confirming its emotional salience (Vandekerckhove et al., 2011). De Zambotti and colleagues provided the most robust evidence, combining subjective ratings, including a stress visual analog scale and an evaluation of perceived tension, with physiological markers such as pre sleep salivary samples to measure cortisol and ECG recordings. They found significantly increased scores of perceived tension, cortisol levels, HR, and reduced parasympathetic activity (lower HF-HRV) during the TSST night, strongly supporting effective stress induction (de Zambotti et al., 2015). In contrast, Gehrman and colleagues observed only mild elevations in cortisol and anxiety, using the state scale of the State-Trait Anxiety Inventory (STAI) (Spielberger et al., 1983), without reporting statistical significance, limiting confidence in the stressor's impact (Gehrman et al., 2016).

Naturalistic stress paradigms varied in how thoroughly stress exposure was verified. Petersen and colleagues collected extensive daily and weekly self-reports, showing significantly elevated perceived stress, reduced cognitive control, worse health ratings, and lower next-day anticipation during high workload periods. However, cortisol levels did not differ significantly between high- and low-stress weeks (Petersen et al., 2013). Esquivel-Mendoza and colleagues also exploited a real-world context (COVID-19), but did not report cortisol results nor include any non-sleep measures of stress, making the efficacy of the stressor uncertain (Esquivel-Mendoza et al., 2023). Nevertheless, it can reasonably be assumed that the context was effective in inducing stress, as the physiological and psychological effects of the COVID-19 pandemic have been widely documented (Baliyan et al., 2021; Manchia et al., 2022; Taylor et al., 2022).

Finally, studies using on-call conditions showed mixed evidence. One study employed a broad set of subjective tools (Activation-Deactivation Adjective Check List [AD-ACL], POMS, and vVAS) (Thayer, 1986). Although some trends indicated higher tension and arousal in the on-call condition, only the AD-ACL Arousal subscale showed significant change (Wuyts et al., 2012). In contrast, Torsvall & Åkerstedt did not include any independent stress verification beyond sleep data, limiting confirmatory value despite the ecological realism of the setting (Torsvall & Åkerstedt et al., 1988).

Study	Stressor	Type	Time of delivering	Stressor Effects other than sleep disturbances
Torsvall & Åkerstedt, 1988	On-call duty paradigm	Daily life experimental stressor	Entire night	None
Bader et al., 2010	Stress-inducing video	Experimental emotional stressor	Between 21:00-22:00, after subjects went home to sleep	Pre-post comparison of scales measuring emotional state and arousal
Vandekerckhove et al., 2011	Failure induction	Experimental psychosocial stressor	Within 60 minutes before bedtime (23:00)	PANAS questionnaire
Petersen et al., 2013	Work-related stress	Daily life natural stressor	Stress period	Salivary cortisol levels and self-report questionnaire about perceived stress
Wuyts et al., 2012	On-call paradigm	Daily life experimental stressor	25 minutes before bedtime	AD-ACL, POMS and vVAS questionnaires
Talamini et al., 2013	Stress-inducing video	Experimental emotional stressor	Within 120 minutes before bedtime (23:00-00:00)	POMS and valence VAS the day after the video viewing and the PSG night
de Zambotti et al., 2015	Adaptation of the Trier Social Stress Test	Experimental psychosocial stressor	30 minutes before bedtime	Stress VAS, self-report tension scale, salivary cortisol levels, ECG
Gehrman et al., 2016	Experimental thread of electric shock	Experimental emotional stressor	40 minutes before bedtime	Salivary cortisol levels and STAI questionnaire
Esquivel-Mendoza et al., 2023	COVID-19	Daily life natural stressor	Stress period	None

Table 5. *Overview of stressor types, timing, and related measures of their impact across studies. PSG = Polysomnography; PANAS = Positive and Negative Affect Schedule; AD-ACL = Activation-Deactivation Adjective Check List; POMS = Profile Of Mood Scale; VAS = visual analog scale; ECG = electrocardiogram; STAI = State-Trait Anxiety Inventory.*

3.3.13 Conclusions

This systematic review shows that many of the included studies have successfully induce measurable sleep disturbances manipulating stress. Despite differences in design and methodology, a recurring picture emerges: stress disrupts sleep continuity, most notably by increasing SL, reducing SE%, and elevating WASO. Importantly, consistent alterations in REM sleep were also observed. Given the central role of REM sleep in emotional-stress processing discussed in the previous chapter, these findings strongly reinforce the view that stress-related sleep disturbances can compromise overall functioning by impairing emotional regulation abilities.

At the same time, two main concerns affect the strength of current evidence. First, only a handful of studies to date have employed a rigorous pre–post within-subject design combined with objective sleep measures, what we think should be the gold standard for clarifying causal relationships between stress-introduction and sleep disturbances. Second, the studies identified were highly heterogeneous, differing in stress paradigms, sleep metrics, and participant populations, and only a minority directly verified the effectiveness of the stressor itself, independent of its impact on sleep. This raises important questions about the comparability and interpretability of existing findings.

For these reasons, in the present doctoral project we chose to employ a widely validated stress-induction paradigm, ensuring robust manipulation of stress. Crucially, we not only applied this instrument but also specifically assessed and validated its effects on sleep, thereby addressing a central gap highlighted by this review and advancing the methodological rigor of research on stress–sleep interactions.

4. Why Consider the Role of Attention?

4.1. Attention as a Cognitive Function: a Brief Introduction

Attention is one of the most complex and widely studied cognitive functions. Entire volumes have been written in an effort to define its boundaries, describe its mechanisms, and explore its implications. The concept of attention has been discussed since as early as the 4th century B.C., when Aristotle distinguished between simply experiencing a sensation and the act of "directing the mind" toward something. From then on, even if not always explicitly, many philosophers continued to reflect on how we focus and select among mental contents. Thinkers such as Descartes, Locke, and Kant each offered their own interpretation of what it means to attend.

By the late 19th and early 20th centuries, with the birth of scientific psychology, attention began to be investigated experimentally. Figures such as Wilhelm Wundt and William James tried to describe and measure it as a mental process. Later, the rise of neuroscience allowed researchers to look beneath behaviour, identifying the brain networks that support different attentional processes (Wayne and Brett, 2015). For this reason, in this chapter, without aiming to be exhaustive, my goal is simply to provide a working definition of attention that will serve the purposes of this work.

William James, one of the founders of experimental psychology, defined attention as a process by which the mind selects and consciously focuses on a single element, external or internal, while disregarding the rest (James, 1980). Although it was later demonstrated that not all attentional processes require conscious or voluntary participation (Koch & Tsuchiya, 2007) and many alternatives or expanded definitions have been developed since then, James's definition remains valid in its essence. Yet a question arises spontaneously: thank to which mechanisms are we able to filter incoming information, both internal and external, while selecting only the most relevant?

One of the most influential models of attention in the one proposed by Broadbent, who focused on the role of sensory processing. According to his model, everything that reaches our senses enters a sensory register ("sensory buffer"), which has an unlimited capacity but lasts only briefly. During this short period, our attentional system act as a filter ("early selection"), analysing the perceptual proprieties of inputs and selecting those that are relevant. These selected inputs proceed further processing, while all the other information in the sensory buffer is discarded (Broadbent et al., 1958).

A major criticism of Broadbent's model is represented by, what is called, the *cocktail party effect*: even unattended auditory inputs can still be perceived if they are particularly salient. A classic example is our ability to notice our own name being spoken by someone across the room while we are engaged in another conversation (Cherry, 1953).

So a revised version of this model was proposed by Treisman in 1969, following Cherry's example and other evidence showing that non-attended sensory inputs can still affect cognitive functioning (Corteen & Wood, 1972; Lewis, 1970). Treisman postulated that even in the early stages of sensory information processing, a primitive level of elaboration is already present, and that unattended inputs are not deleted but merely attenuated (Treisman, 1969).

A significant step forward in attention research, particularly meaningful for the present work, has been done by Posner and Petersen, starting in 1990 and culminating more than 20 years later in 2012. Their model conceptualized attention as comprising three distinct but interacting functions, each supported by different neural systems:

5. Alerting Network: Responsible for vigilance and readiness to respond to meaningful stimuli. The key hub of the alerting network is the locus coeruleus (LC) with his noradrenergic projection, particularly to dorsolateral prefrontal cortex (DLPFC). Some authors have described an "adaptive gain" model, according to which the LC modulates our efficiency in attentive task execution: the alternation between phasic and tonic LC activity modulates the alternation of sustain attention and flexibility to explore. Specifically, task engagement and focused attention are correlated with the phasic activity of this area (Aston-Jones & Coen, 2005; Posner & Petersen, 1990; Posner & Petersen, 2012).
6. Orienting Network: This process enables us to selectively focus our attention on one or more target stimuli, prioritizing to sensory information according to modality or spatial position. Posner and Petersen distinguished between endogenous orienting, a top-down process, in which attention is directed intentionally according to one's goals; and exogenous orienting, a bottom-up process, in which attention is captured automatically by salient stimuli (Posner & Petersen, 1990). Although both processes have the same outcom, they rely on different neuronal systems. Dorsal frontalparietal regions, such as the intrapariental sulcus and the frontal eye fields, are involved in the endogenous

orienting, while more ventral regions, including the temporoparietal junction and the ventral frontal cortex support exogenous orienting (Corbetta & Shulman, 2002; Heilman et al., 1985). The activity of these regions, which determines orienting performance, is regulated by the cholinergic system. Evidence from animal models studies has shown that artificially interrupting cholinergic activity in the intraparietal cortex reduce orienting abilities (Davidson & Marrocco, 2000; Voytko et al., 1994). Consistently, experimentally increasing acetylcholine levels in human modulate attention orienting performance, particularly in parietal area and in frontal areas involved in visual-spatial orienting (Bentley et al., 2003; Furey et al., 2000).

7. Executive Control Network: This system allows to focus the attention according to internal goals by regulating and resolving cognitive conflicts, maintaining goal-directed behaviour in response a complex or new situations, and inhibiting automatic or inappropriate response. It represents a top-down regulative processes sustained by the activity of high-order regions, such as the Anterior Cingulate Cortex (ACC) and the Prefrontal Cortex (PFC), both crucial to detect and solve conflicts (Pardo et al., 1990; Liu et al., 2004; Fan, 2014). The activity of these regions, especially the ACC, is strongly influenced by the dopaminergic system. For instance, Fosella and colleagues found a positive correlation between two dopaminergic-related genes polymorphisms (one slowing dopamine degradation process, the other increasing dopamine receptor sensitivity) and improved performance in attentional task requiring conflict resolution (Fosella et al., 2002).

Attention should not be regarded as a unitary cognitive function, but rather as a set of distinct mechanisms supported by different neuronal networks that together shape overall attentional performance (see Figure 6).

Since attention plays a fundamental role in processes ranging from perception to higher-order cognition and behaviour (Gazzaniga et al., 2019), understanding its influence requires viewing it as a multidimensional cognitive domain, where specific attentional systems may selectively contribute to certain processes but not to others.

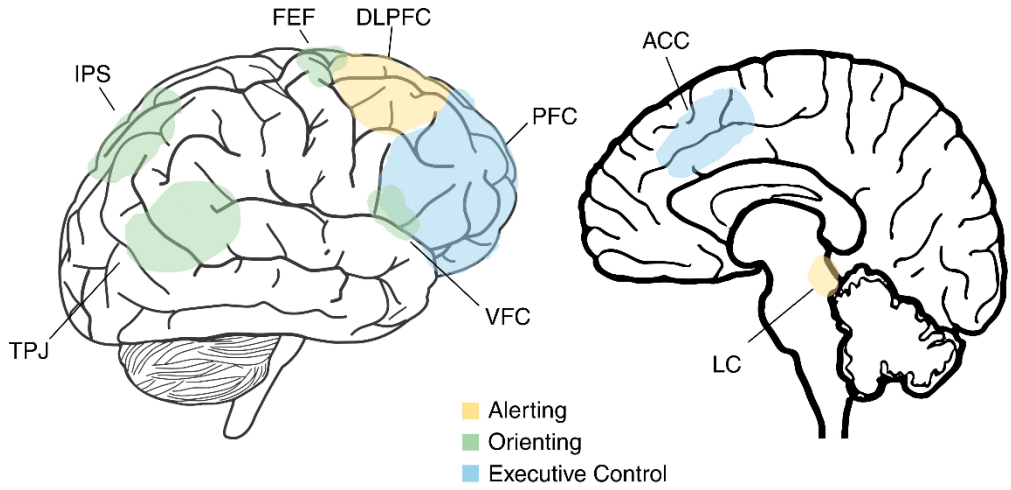


Figure 6. *Representation of the major brain regions involved in the three attentional domains: alerting (yellow), orienting (green), and executive control (blue).*
TPJ = Temporal-Parietal Junction; IPS = Intraparietal Sulcus; FEF = Frontal Eye Fields; DLPFC =Dorsolateral Prefrontal Cortex; PFC = Prefrontal Cortex; VFC = Ventral Frontal Cortex; ACC = Anterior Cingulate Cortex; LC = Locus Coeruleus.

4.2. The Role of Attention in Insomnia Disorder: The Attention-Intention-Effort (AIE) Model and Beyond

Is it meaningful to introduce the construct of attention, as anticipated in the introduction of the present work, because it may help to better understand the role of cognitive domain in ID, providing a more complete view of the mechanisms undergoing the disorder. It is well established in the literature that CBT-I represents the most effective treatment for ID (Hertenstein et al., 2022; Jernelöv et al., 2022; Morin & Espie, 2007), therefore, acting at a cognitive level could have a significant impact on the disease course. For this reason, one of the priorities of sleep medicine research should be to further investigate this aspect in order to design increasingly targeted therapeutic approaches, making ID therapy progressively more efficient.

In this framework, attention constitutes a great candidate among cognitive domains, as its relationship with ID has already been demonstrated. Specifically, Colin Espie, in 2006, by introducing the Attentional-Intention-Effort (AIE) Model, analysed the pivotal role of attention component in ID. His hypothesis has its roots in the behavioural psychology field, particularly in the concept of stimulus control, according to which a given behaviour is more likely to occur following the presence of a specific stimulus, called *discriminative*

stimulus, while, in its absence, the behaviour is less likely to occur (Dismoor, 1995; Skinner, 1938). Bootzin applied this concept to ID: normal sleep is associated with several discriminative stimuli (i.e. the bed and the bedroom environment, nighttime, darkness, etc.) and ID develops when this association is disrupted because the individual has learned new association between these discriminative stimuli and wakefulness (Bootzin, 1972). Espie integrated this view of sleep as an operant conditioning process with the hyperarousal hypothesis within his Psychobiological Inhibition Model, which posits that the interruption of the normal automatic occurrence of sleep in ID results from a failure to reduce the arousal state, typical of wakefulness (Espie, 2002). The arousal observed in ID is therefore not merely the outcome of a state of hyperactivation, but rather the consequence of an inability to inhibit wakefulness.

The AIE Model was proposed to explain how this inhibition process became disrupted. As suggested by its name, and illustrated in Figure 7, the model proposed that the interruption results from the interaction of three distinct factors.

The first is Attention. As a direct consequence of the distress caused by experiencing sleep difficulties, individuals tend to allocate their attention on sleep-related cues, both external (e.g., environment), and internal (e.g., monitoring level of tiredness) (Espie et al., 2006). The presence of this attentional bias has been consistently demonstrated in various paradigms implemented by Espie's group. For instance, ID patients show significantly shorter reaction times to sleep-related words compare to neutral words and compare both to healthy controls and individuals with other sleep difficulties (i.e., Delayed Sleep Phase Syndrome) (MacMahon et al., 2006). In other paradigms implying an emotional version of the Stroop task, ID patients display greater interference (longer reaction times in naming the colour of the written word) when the words presented were sleep-related (Spiegalhalder et al., 2008; Spiegalhalder et al., 2010). Woods and colleagues used a modified Posner paradigm to examine attentional disengagement from one of the main sleep-related cues, a clock, showing that individuals with primary ID displayed delayed disengagement from a clock stimulus compared with good sleepers (Wood et al., 2009). From these results it can be inferred that ID patients tend to focus on sleep-related cues at an explicit (monitoring processes) and implicit level. This mechanism interferes with the automatic nature of sleep, since focusing attention on sleep requires consciousness, and that, by definition, inhibits sleep.

The conscious, sleep-focused state that catheterizes ID patients easily leads to the development an Intention to sleep. As already noted, sleep is a naturally automatic process that cannot be voluntarily induced. Attempting to self-induce sleep leads to behaviours that have the opposite effect. Espie drew inspiration from Wegner's Ironic Process Theory: when we try to deliberately suppress a mental content, we obtain a rebound effect that makes this content even more salient. His classic example is: "Try not to think of a white bear", and the more one tries, the more the thought of the bear intrudes (Wegner, 1994). Espie postulated that a similar mechanism occurs with sleep: the more an ID patient tries to control the process of falling asleep, the more they monitor themselves, and the more they remain awake (Espie et al., 2006). Interestingly, long before Espie theory, Ascher and Turner (1979) asks to ID patients either to try to remain awake, to practice progressive relaxation, or to follow a behavioural protocol based on Bootzin guidelines, those with paradoxical instruction to stay awake showed significantly greater reductions in sleep-onset latency and reported less sleep-related anxiety than participants in the other groups (Ascher & Turner, 1979).

The third component of the model is the spontaneous consequence of the previous ones as well: the ID patient has his attentions on sleep and *wants* to sleep, therefore invests cognitive and behavioural Effort to achieve it. This effort manifests indirectly through many of the perpetuating behaviours discussed in the first chapter (e.g., increase the time spent in bed, limiting nigh time social activities, etc.) and directly through performative relaxation: continuously checking one's body and mind, trying to suppress intrusive thoughts, or implementing various strategies such as counting, breathing exercises, or meditation, etc. All of these efforts, however, maintain mental activation, especially in the prefrontal cortex, which is crucial for monitoring, thereby increasing arousal and making it harder to transition from wakefulness to sleep (Espie et al., 2006). In a previous review, Harvey already underlined how increased sleep effort and sleep-related performance anxiety were significantly associated with longer SL, higher physiological and cognitive arousal before sleep, and poorer subjective sleep quality, illustrating how the very attempt to control or optimise sleep paradoxically maintains the state of wakefulness (Harvey, 2002). Supporting this perspective in a study implying, between other sleep measures, the Glasgow Sleep Effort Scale (that measure the tendency to try to

fall asleep) authors found that greater sleep effort scores were associated ID severity (Broomfield & Espie, 2005; Hertenstein et al., 2015).

The model is not without criticism, such as the difficulty in clearly distinguishing and measuring its three components separately. Nevertheless, it provides a valuable conceptual framework for understanding how ID is perpetuated. Moreover, it offers indication regarding which cognitive and behavioural components could be targeted therapeutically to break the vicious cycle of ID more effectively. The strongest evidence so far, likely because it is the easiest component to measure, concerns the role of attention, one of the main focuses of the present research as well. However, no studies to date have explored which specific domains of this multifaced cognitive function are implicated in ID and how they contribute to its progression.

While Espie's AIE model primarily describes an attentional bias toward sleep-related cues, more recent theoretical frameworks have emphasized the importance of the attentional process itself rather than its specific content. In this sense, Wells' metacognitive model proposes that psychopathology is maintained by a maladaptive pattern of cognitive-attentional processing, termed the Cognitive Attentional Syndrome (CAS), characterized by persistent self-focused attention, monitoring, and attempts to control internal states (Wells, 1995; Wells & Matthews, 1996; Wells, 2009). This model is developed in the anxiety disorder context, but it can be easily adapted to ID. This is because, within this framework, a maladaptive function of attention is not determined by its content (i.e., sleep-related cues, in our case), but by how attention is allocated and regulated. Individuals characterized by the CAS show a reduced ability to disengage from ongoing monitoring processes. Such monitoring may target various internal states, that in case of ID can ranging from arousal and fatigue to negative or intrusive thoughts and thereby sustains a heightened level of cognitive activation that interferes with the natural de-arousal mechanisms required for sleep initiation and maintenance.

Accordingly, the aim of the present work is to investigate which specific attentional components are most critical in the maintenance of ID and to clarify how they might be targeted to improve treatment efficacy.

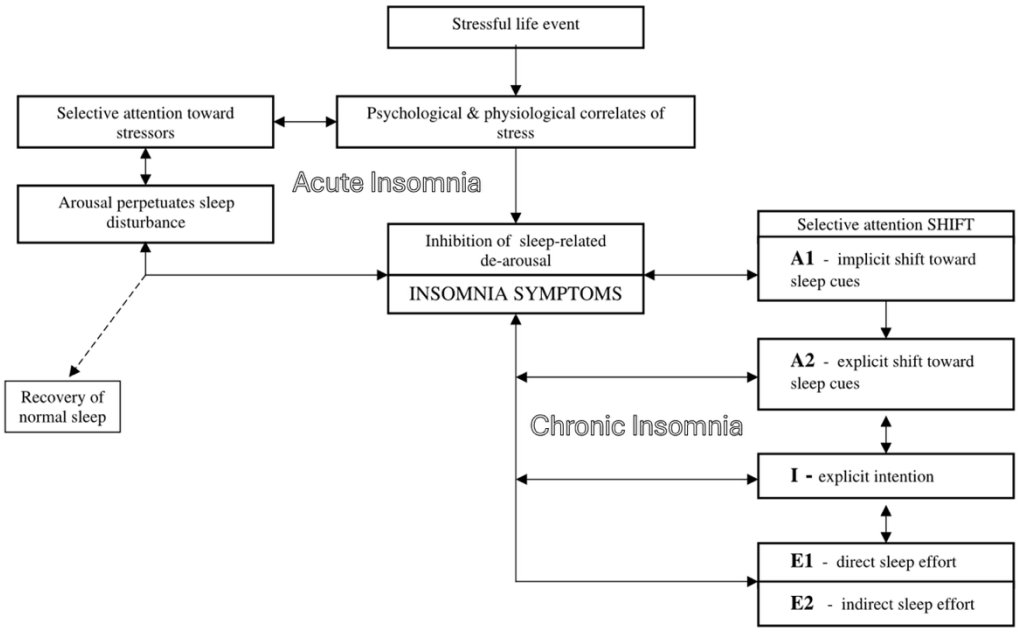


Figure 7. *Schematic representation of the transition from acute ID to chronic ID, based on the Attention–Intention–Effort model (Source: Espie, 2006; License number: 6124960925573).*

5. Experiment 1: Experimental Validation of Stress-Inducing Video Stimuli

The main objective of this work is to investigate the role of attentional abilities in modulating the sleep system's response to stress. To address this aim, sleep reactivity was experimentally manipulated through a standardized stress-induction procedure. Therefore, the first challenge for this work was to identify the most appropriate instruments to induce stress.

From the systematic review of the literature, it emerged that there is no agreement within the scientific community on what constitutes the most appropriate and effective way to manipulate stress. We observed that different studies employ a wide variety of approaches, which makes it difficult to compare their findings. Our goal was to identify a solid and ecologically valid stress manipulation that could be applied across different populations.

Two of the articles included in the review used negative video to induce stress within the framework of the Trauma Film Paradigm (TFP) (Bader et al., 2010; Talamini et al., 2013). The TFP was developed between the 1960s and 1970, initially by Lazarus and later refined by Horowitz, as a more ecological alternative to traditional laboratory methods based on static emotional stimuli for studying psychophysiological reactions to stress and trauma (Lazarus & Alfert, 1964; Lazarus & Opton, 1964; Lazarus et al., 1965; Horowitz, 1969; Horowitz, 1975; Horowitz & Becker, 1971; Horowitz et al., 1972). These videos have the advantage of being able to elicit psychological and physiological distress similar to that experienced during an actual traumatic event, while not exposing participants to actual danger. Indeed, the induced distress is both less intense and more transient than that resulting from real trauma (Holmes & Bourne, 2008; James at al., 2016). Based on these considerations we adopted this approach as we considered it as a good compromise between achieving an ecological paradigm and a sufficiently ethical one.

While examining the studies by Talamini and Bader, it emerged that none of the videos they used have been previously validated, and their efficacy was verified only posteriorly. This limitation largely stems from the lack of a standardized database of film stimuli comparable to the emotional images sets (e.g. Internation Affective Picture System, Nencki Affective Picture System, Geneva Affective Picture Database, etc.) (Lang et al., 1999; Marchewka et al, 2014; Dan-Glauser & Scherer, 2011). This is problematic

because, even when the efficacy of a video is demonstrated, there is no guarantee that the negative affect induced by one video is equivalent in intensity or quality to that elicited by another (Open Science Collaboration, 2015). Consequently, even when two studies employ the TFP, comparing their results remains challenging.

For this reason, and in order to ensure maximum control over our paradigm, we decide to use a previously validated set of TFP videos developed by Arnaudova and Hagenaars in 2017, and (1) replicate their findings on the efficacy of these videos in inducing stress, (2) expand the set by adding three additional negative videos, and (3) test the effects of these videos on subjective sleep (Arnaudova & Hagenaars, 2017). We decided to start from a previously validated set for three main reasons: first, to ensure that at least some of the videos had a high likelihood of inducing stress; second, to extend the available material with new, comparable clips; and third, because in psychological science the importance of replicating data is often overlooked; indeed, a 2024 study found that from 2010 to 2021, among the 100 highest-impact psychology journals, only 0.2% of all published articles were direct replications (Clarke et al., 2024).

Based on these considerations, the aim of the first experiment was twofold: on the one hand, to assess the ability of the videos, both those previously validated and the new ones we introduce, to elicit a subjective and physiological stress response; on the other hand, to explore how these videos affect the participant's subjective sleep, as sleep will be the target of the next and main experiment of my thesis.

5.1. Methods

5.1.1. Participants

Participants were recruited on voluntary basis from the general adult population through posters, online advertisements, and word of mouth. A total of 65 individuals completed the online screening questionnaire; however, only 40 participants (20/20 male/female [M/F] age: 30.3 ± 11.9 years; range: 20.0 - 62.0) met the inclusion criteria and took part into the study. Inclusion criteria required participants to be adults between 18 and 65 years of age, fluent in Italian, and with normal or corrected-to-normal vision and hearing.

The screening questionnaire was administrated using Survey Monkey platform.

Participants were excluded if they met any of the following conditions (assessed using closed-ended questions):

- Suffering from severe or degenerative neurological, cognitive, or psychiatric disorders;
- Having a current of diagnosis for a Sleep Disorder;
- Having experienced a traumatic event related to one of the video themes (physical violence, sexual violence, traffic accident), in order to avoid potential bias from participants who might be particularly vulnerable to these themes due to their personal experiences;
- Suffering from hemophobia (fear of blood) or trypanophobia (fear of needles);
- Presence of substance abuse.

Moreover, after signing the informed consent participant completed a battery of questionnaires that included the Beck Depression Inventory (BDI-II score < 14) (Beck et al., 1996; Solaro et al., 2016), the STAI (STAI trait and state; score ≤ 40) (Spielberger, 1989; Pedrabissi & Santinello, 1989) and the Insomnia Severity Index (ISI). This to ensure that at baseline all the subjects have a good sleep quality (ISI score < 14) (Morin et al., 2011; Castronovo et al., 2016). These measures were used to assess the presence of depressive and anxious symptoms and to ensure that all included participants were below the clinical thresholds, as well as to verify that they had good sleep quality at baseline.

5.1.2. Experimental Protocol

The study was reviewed and approved by the internal Ethics Committee of Sigmund Freud University. All procedures complied with institutional ethical standards and with the Declaration of Helsinki.

If participants met the inclusion criteria and consented to participate in the two weeks protocol, they were first instructed by telephone on how to complete an online Sleep Diary within 10 minutes after awakening, for the entire duration of the study.

After the first seven days of diary completion, participants came to the laboratory for the video administration session, which lasted approximately one hour and was always scheduled on weekdays between 9:00 a.m. and 1:00 p.m. to minimize circadian variability

in psycho-physiological responses. During the video administration, participants underwent a Photoplethysmography (PPG) and Electrodermal Activity (EDA) recording using the BIOPAC System (Biopac System Inc., 2014, Santa Barbara, CA).

After this session participants continued filling in the Sleep Diary for another seven days and completed an additional online Intrusion Diary. At the end of the second week, subjects completed one last questionnaire, the Impact of Event Scale Revised (IES-R) in a version adapted to the videos, based on Arnaudova and Hagenaars (Weiss & Marmar, 1997; Pietrantonio et al., 2003; Arnaudova & Hagenaars, 2017). The IES-R scale has been ideated to assess PTSD symptoms and is divided into two subscales: 15 items assess intrusive symptoms, and 15 items assess avoidance symptoms (Weiss & Marmar, 1997).

The timeline of the experimental protocol is illustrated in Figure 8.

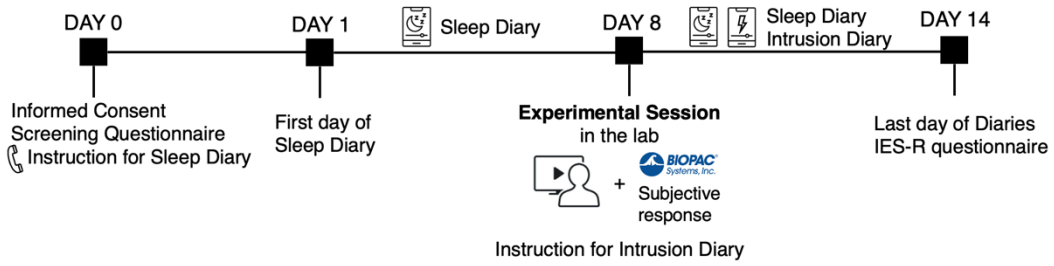


Figure 8. *Overview of the two-week experimental protocol.*
IES-R = Impact of Event Scale Revised.

5.1.3. Video Administration

The entire laboratory session lasted approximately 40 minutes, although participants were told that it would take about one hour to allow for possible unforeseen delays. The physiological setup with the initial questionnaires took approximately 10 minutes, followed by about 30 minutes of video administration. Before the beginning of the session, participants were informed that, due to the potentially distressing nature of the videos, they could pause or terminate the experiment at any time. Additionally, they were informed that a licensed clinical psychologist was available after the session for anyone wishing to discuss their experience.

During the laboratory session, before the video administration began, all participants completed the BDI-II (Beck et al., 1996; Solaro et al., 2016) and the STAI (Spielberger,

1989; Pedrabissi & Santinello, 1989) questionnaires to assess their psychological state prior to the video viewing.

After that, the Biopac MP160 hardware system (Biopac System Inc., Santa Barbara, CA) was set up to record EDA and PPG. The PPG transducer was fasted around the wrist of the non-dominant hand to allow participants to interact freely with the computer during the session. EDA was measured using two Ag/AgCl electrodes (EL507) attached to the distal phalanges of the middle and ring fingers of the same hand; while PPG was recorded via a transducer placed on the index finger of the same hand and it was secured firmly to ensure optimal signal quality. Calibration was performed twice to ensure accurate signal detection and signal quality was checked. Participants were asked to take a deep breath to verify the stability of both the EDA and HR traces before proceeding. Participants were instructed to remain as still as possible throughout the entire recording session. If they felt the need to move, they were asked to do so only during the brief intervals between videos. Moreover, throughout the recording, the experimenter monitored the participant's behaviour and inserted flags into AcqKnowledge (version 5.0) software signal trace whenever a visible movement occurred, so that these segments could later be identified and excluded during signal preprocessing. All physiological signals were continuously recorded throughout the video presentation at a sampling rate of 1000 Hz (Boucsein et al., 2012; Benedek & Kaernbach, 2010; BIOPAC Systems, Inc., 2014). This setup allowed for continuous monitoring of the immediate autonomic physiological responses to the videos' presentation.

The video presentation took place on a computer equipped with a 15.6-inch monitor, positioned approximately 60 cm from the participant, who was seated comfortably in front of the screen. The experiment was conducted in a dark, sound-attenuated room to minimize external distractions and ensure consistent viewing conditions. Room temperature and humidity were kept as constant as possible throughout the session to prevent environmental factors from influencing physiological measurements. The experimenter sat behind the participant at a workstation equipped with a computer connected to two synchronized monitors: the one in front of the participant, and the other showing the real-time skin conductance activity and cardiac signal via AcqKnowledge (version 5.0) software. This setup allowed the experimenter to continuously monitor signal quality and insert event markers to flag any movement artifacts. Each time the

participant initiated the playback of a video, a trigger signal was automatically sent to the physiological recording system, marking the onset of the stimulus on the trace.

After the BIOPAC setup, all participants first watched a mood-stabilizing video ("*Planet Earth: From Pole to Pole", Fothergill, 2007*), a neutral, naturalistic documentary 3 minutes segment depicting polar bears in their environment. Afterward, participants completed a Mood Questionnaire developed by Arnaudova and Hagenaars to ensure that the baseline emotional state of each participant was neutral before the presentation of the experimental videos (Arnaudova & Hagenaars, 2017).

Then, the actual video session began: all the 12 target videos were presented in randomized order (no participant viewed the videos in the same order as another), and the randomization algorithm was implemented in R (version 4.2.2) (R Core Team, 2022). After each video, subjective immediate responses were collected using a Film Response Questionnaire (FRQ) developed by Arnaudova and Hagenaars. This instrument evaluates perceived valence, degree of physical activation (arousal), sense of immobility, perceived distress, disgust, embarrassment, involvement, and attention, as well as whether participants had seen the video before and whether they averted their gaze from the screen during the presentation (Arnaudova & Hagenaars, 2017). All dimensions were rated on a 9-point Likert scale ranging from 1 (not at all) to 9 (extremely), with the exception of valence, which was assessed on a bipolar scale ranging from −4 (extremely negative) to +4 (extremely positive), with 0 indicating a neutral valence.

Both the Mood Questionnaire and the FRQ were translated from English by the research team, and the english versions are reported in Appendix B.

Arnaudova & Hagenaars included four video themes: physical, relational, traffic and food. For each theme, three film clips where selected, each representing a different category: neutral, positive and negative. In the present study, we added three additional negative videos, one for each theme except for the food theme, which we decided to exclude. This decision was made for two main reasons: first, to ensure that the entire procedure did not exceed one hour and thus avoid participant fatigue and distraction; and second, because in the original experiment the food theme was included specifically to test the traumatic potential of disgust, which was not a focus of the current study (Arnaudova & Hagenaars, 2017). We obtained the original video clips from Arnaudova and Hagenaars (2017). Some of these clips were recreated frame by frame to improve

their resolution, using iMovie 10.3.2 (Apple Inc., 2021), the same software employed to edit the three new videos we added. Table 6 reports all the videos, organized by theme and emotional category, along with a brief description of their content.

Theme	Category		Description	Length (m:ss)	Source	Year	Director
Physical	Neutral	PN	A basketball match	1:54	"Coach Carter"	2005	Thomas Carter
	Positive	PP	A compilation of football players celebrating goals in funny and creative ways	1:17	Real footage of professional football matches		
	Negative 1	PT2	A scene in which a man during an assault is brutally hit in the face with a fire extinguisher	2:30	"Irreversiblé"	2002	Gaspard Noé
	Negative 2	PT2	A beating scene in which a man is hit and humiliated by his cellmates	2:31	"Stoic"	2009	Uwe Boll
Relational	Neutral	RN	A man and a woman walking while talking by the sea	1:18	"Mr Jones	1993	
	Positive	RP	A young couple makes love on a bed	2:00	"Come early morning"	2006	
	Negative 1	RT1	A man raping a woman in an underpass	2:09	"Irreversiblé"	2002	Gaspard Noé
	Negative 2	RT2	Three men raping a woman in a forest	2:12	"I spit on your grave"	1978	Meir Zarchi
Traffic	Neutral	TN	A woman driving a car	1:07	"Happy-go-luck"	2008	Mike Leigh
	Positive	TP	A comic scene showing a taxi chase where the taxi suddenly grows wings and starts flying	1:27	"Taxxi 2"	2000	Gérard Krawczyk
	Negative 1	TT2	Three girls having a violent car accident due to phone use while driving	2:44	Texting w driving		Television advertisement
	Negative 2	TT2	Real footage of Formula 1 driver Romain Grosjean's car catching fire during a race because of an accident	1:56	"Grosjean's insane fireboat crash"	2019	Formula 1 documentary: "Drive to Survive"

Table 6. *List of the 12 videos used in the study, with their corresponding acronyms as used in the text, accompanied by a brief description, the duration of the extracted clip, the title and release year of the source film/video, and the director's name when available. PN = Physical Neutral Video; PP = Physical Positive Video; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); m:ss = minutes : seconds.*

5.1.4. Sleep and Intrusion Measures

Intrusions are defined as traumatic event-related involuntary mental contents, which can consist of a perceptual re-experiencing of the event or of related cognitive thoughts and emotions. They occur involuntarily and are typically accompanied by distress and negative affects (Iyadurai et al., 2018). Intrusions represent one of the hallmark symptoms of PTSD, so can be consider an indicator of the traumatic impact of an event (Clark & Mackay, 2015). Arnaudova and Hagenaars (2017) already took this aspect into consideration to evaluate the efficacy of their video within the TFP. For this reason, we included the same intrusion measure they used.

On the other hand, Arnaudova and Hagenaars (2017) did not investigated the role of stress on sleep (Lo Martire et al., 2020); therefore, no sleep measures were included in their original paradigm. Since the main focus of the present research is sleep, and as those videos represent surrogates of real traumatic experiences, we aimed to preliminarily verify their impact on sleep.

Therefore, to measure the delayed impact of the video, two self-report online diaries (delivered throughout Google Form Platform) were included in the protocol:

1. Sleep Diary: A self-report measure used to monitor subjective sleep quality (e.g. perceived sleep depth, restorativeness, and restlessness) and perceived sleep parameters (e.g. estimated SL, WASO, TST, etc.). Participants were instructed to complete the diary every morning within 10 minutes after awakening. They were asked to refer only to their perception without considering external time indicators (e.g. alarm clock, phone) or other's impressions. Participants were also asked to maintain as regular sleep schedule as possible for the entire duration of the protocol. To enhance compliance, a daily completion reminder was sent to each subject just before their usual awakening time.
2. Intrusion Diary: A self-report measure to monitor the occurrence of video-related intrusive memories. Participants were instructed to complete the diary every time they experienced an intrusion for an entire week after video viewing. They were asked to briefly describe the intrusion and to evaluate its characteristics on Lickert scales (e.g. degree of perceived control, level of emotional distress, vividness etc.). Because Arnaudova and Hagenaars (2017)

reported limited compliance with diary completion, we decided to follow their procedure and include a daily self-report of the total number of intrusions experienced each day, collected via smartphone.

The complete set of questions included in both the Sleep Diary and the Intrusion Diary is reported in Appendix A.

5.1.5. Physiological Data Analysis

Having recorded the EDA and PPG signals continuously throughout the entire video-viewing session, we used the automatically inserted triggers on the signal trace to identify the segments of interest for analysis.

The PPG-derived heart signal was band-pass filtered between 0.5 and 8 Hz (4th-order Butterworth, zero-phase) to remove low-frequency drift and high-frequency noise. Heartbeat peaks were automatically detected using the *findpeaks function* in MATLAB (2021b) (MathWorks, Inc., 2021, Natick, MA, USA), with a minimum interbeat interval of 0.4–1.5 s (corresponding to 40–150 beats per minutes [bpm]). Interbeat intervals (IBIs) were computed as the time difference between consecutive peaks, and instantaneous HR was calculated as HR = 60 / IBI (bpm). The resulted HR signal was visually inspected, and spurious peaks were removed and linearly interpolated. The resulting HR time series was then smoothed using a 5-second moving average window to reduce residual noise and movement artifacts.

For each video, the mean HR was calculated over the entire clip duration, regardless of its length. A baseline HR value was computed for each participant as the mean HR during the mood stabilizing video. We chose to extract HR data to allow direct comparison with the physiological results reported by Arnaudova and Hagenaars (2017) and to avoid using heart rate variability (HRV), thereby enabling the analysis of entire video durations as done in their study.

In addition to HR, EDA signals were analysed to capture sympathetic arousal responses associated with specific emotionally salient moments of the videos. Unlike heart rate, which reflects both sympathetic and parasympathetic influences and varies relatively continuously throughout the entire stimulus, EDA provides a more direct index of transient sympathetic activation (Dawson et al., 2007). Therefore, to ensure

comparability across clips of different lengths, a common 60-second analysis window was selected for all videos, with the 30-second time point approximately aligned with the emotional peak or pivotal moment of the clip[4]. This procedure allowed us to focus on the period of maximal affective engagement while maintaining a standardized temporal frame for physiological analysis.

EDA preprocessing and analysis were carried out using *Ledalab* (version 3.4.6), a MATLAB-based toolbox specifically designed for EDA analysis (Benedek & Kaernbach, 2010a; Benedek & Kaernbach, 2010b). Raw signals were imported into Ledalab and downsampled to 10 Hz to reduce data size and computational load while preserving the temporal resolution necessary for detecting phasic responses. Prior to analysis, the signal underwent a series of preprocessing steps to ensure data quality: adaptive smoothing and a low-pass filter (1st-order Butterworth) were applied to remove high-frequency noise and slow drifts, then the 60-second segment for each video was visually inspected to identify and correct possible artifacts (e.g., abrupt signal jumps or motion-related disturbances) using the *Artifact correction tool*. For each participant, the exact onset and offset times of the analysed video segments were determined from the event.

The preprocessed signals were then analyzed using Ledalab's *Continuous Decomposition Analysis* (CDA), which separates the EDA signal into its tonic (skin conductance level, SCL) and phasic (skin conductance responses, SCRs) components (see Figure 9). This approach allows for a more physiologically valid estimation of underlying sympathetic activity by optimizing model parameters (τ_1 and τ_2)[5] iteratively before applying the decomposition (Benedek & Kaernbach, 2010a; Benedek & Kaernbach, 2010b).

From the CDA output, we extracted four indices representing complementary aspects of sympathetic activation:

1. CDA.AmpSum: the sum of amplitudes of significant Skin Conductance Responses (SCR) within the segment, indexing overall phasic response strength;

[4] The peak of emotional activity for the positive and negative videos was determined based on the principal experimenter's personal perception of the most emotional salient moment of the clip, whereas for the neutral videos, given their monotonous nature, it was set to correspond to the temporal midpoint of the scene.

[5] τ_1 and τ_2 correspond to the time constants of the canonical SCR function, describing the rise and recovery phases of the sympathetic activation, respectively.

2. CDA.SCR: the mean value of the phasic driver signal, reflecting the average intensity of phasic activity;
3. CDA.ISCR: the integrated area under the phasic driver curve (SCR × window duration), indicating the cumulative magnitude of phasic activation;
4. CDA.Tonic: the mean tonic activity level (baseline skin conductance), representing the underlying autonomic arousal tone.

Together, these indices provided a comprehensive quantification of both the tonic and phasic components of electrodermal activity, allowing for a detailed assessment of sympathetic arousal dynamics during the selected video segments.

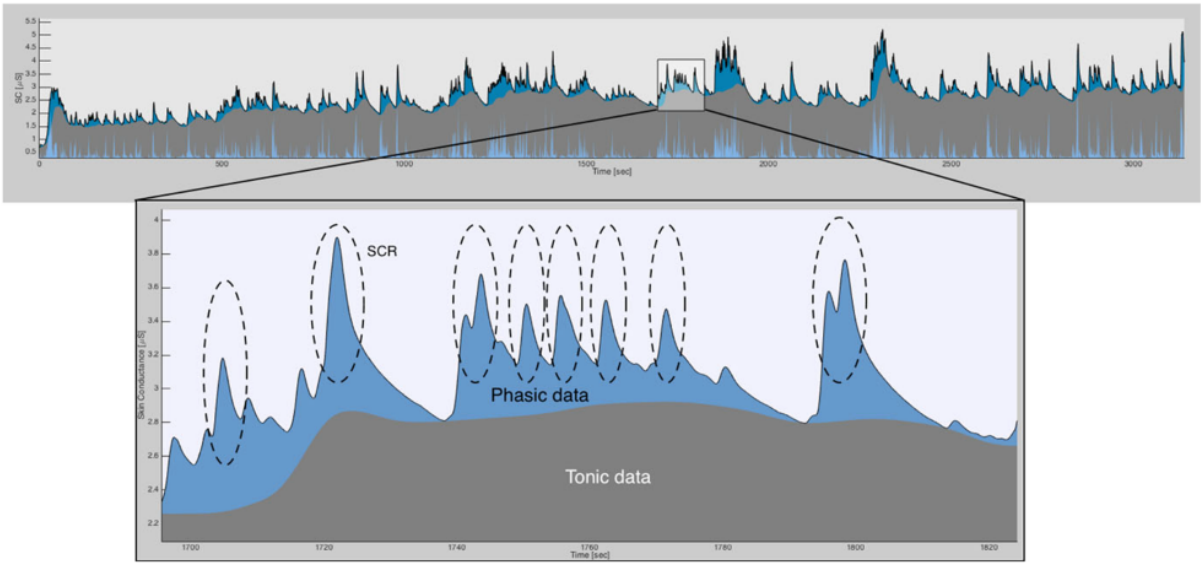


Figure 9. *Example of Continuous Decomposition Analysis (CDA) results on Electrodermal Activity (EDA). The grey line depicts the tonic component, whereas the blue line shows the phasic activity peaks. Dashed circles mark the detected Skin Conductance Responses (SCRs) (Source: Barral, 2016; License number: 6130920558090).*

5.1.6. Statistical Analysis

For the statistical analysis we used two different software: Jeffreys's Amazing Statistics Program (JASP, Version 0.18.2.0; 2024) and MATLAB (2021b) (MathWorks, Inc., 2021, Natick, MA, USA). The level of statistical significance was set at $p < 0.05$ for all tests.

Immediate physiological and psychological responses to the videos were analysed using repeated-measures analyses of variance (rmANOVAs) to compare films within each

thematic category. In addition, a separate rmANOVA was conducted to compare all the negative videos in order to identify the most effective negative stimulus. When the assumption of sphericity was violated, Greenhouse–Geisser corrections were applied. Significant rmANOVA effects were followed by Bonferroni-corrected post-hoc comparisons. For physiological measures that did not meet normality assumptions, non-parametric Friedman tests were used, followed by Conover's pairwise post-hoc comparisons with Bonferroni correction for multiple testing.

Regarding the analyses of the Sleep Diary indices, we performed rmANOVA or non-parametric alternatives to assess differences in sleep quality between the periods preceding and following the experimental session. Specifically, we examined two temporal windows: (1) the entire week before versus the entire week after the experiment, and (2) the two days before versus the two days after the experimental session. This allowed us to evaluate both short-term and slightly longer-term effects of the experimental manipulation on sleep quality.

For delayed responses, correlation analyses were carried out using Spearman's rank correlation coefficient to explore the relationships between immediate psychological and physiological reactions and subsequent developments of both intrusive memories and IES-R scores. The number of intrusions reported by participants during the first two days of the Intrusion Diary was compared with those reported during the last two days using Wilcoxon signed-rank tests, as the data were not normally distributed.

Normality was assessed using the Shapiro–Wilk test and sphericity using Mauchly's test. When the assumptions of normality or sphericity were violated, appropriate corrections or non-parametric statistical tests were applied.

Finally, age was included as a covariate, and analyses were stratified by gender to account for potential sex-related differences.

5.2. Results

5.2.1. Descriptive Statistics

Descriptive statistics are reported in Table 7. The final sample included 40 participants, balanced by sex (20/20 M/F). The average age was 30.3 ± 11.9 years, with a range: 20-

62. We purposefully included participants of across a wide range of age, in order to enhance the generalizability of the findings. Before the beginning of the video administration session, participants reported low levels of state anxiety (STAI-state: 33.0 ± 6.6; range: 21-37) and trait anxiety (STAI-trait: 31.00 ± 8.8; range: 20-39) at the and low levels of depressive symptoms (BDI-II: 5.8 ± 5.7; range: 2-11), all below clinical thresholds. No significant sex differences in baseline STAI or BDI-II scores were observed (independent-samples t-tests, *all ps* > 0.05), nor were significant associations found between age and baseline STAI or BDI-II scores (Pearson correlations, *all ps* > 0.05).

The mood-stabilizing video proved effective, as participants reported low levels of anxiety (1.8 ± 1.5), fear (1.6 ± 1.5), vulnerability (1.8 ± 1.6), sadness (2.0 ± 1.5), anger (1.7 ± 1.4) and immobility (2.2 ± 1.9), together with medium levels of happiness (5.9 ± 1.4) and high levels of perceived control (7.2 ± 1.7). These results suggest that, before the video viewing, all participants were in a neutral to positive emotional state and not affected by negative emotions.

	n (%)	Mean ± SD	Range (min-max)
Age		30.3 ± 11.9 years	20-62
Sex (M/F)	20/20 (50%/50%)		
STAI-S		33.0 ± 6.6	21-37
STAI-T		31.00 ± 8.8	20-39
BDI-II		5.8 ± 5.7	2-11
Mood Questionnaire			
Anxiety		1.8 ± 1.5	1-5
Happiness		5.9 ± 1.4	4-8
Fear		1.6 ± 1.5	1-3
Control		7.2 ± 1.7	5-9
Vulnerability		1.8 ± 1.6	1-4
Sadness		2.0 ± 1.5	1-5
Anger		1.7 ± 1.4	1-5
Immobility		2.2 ± 1.9	1-3

Table 7. *Descriptive statistics.*
STAI-S = State-Trait Anxiety Inventory - state scale; STAI-T = State-Trait Anxiety Inventory - trait scale; BDI-II = Beck Depression Indicator; M/F = male/female; n = number of subjects; SD = standard deviation; min = minimum; max = maximum

5.2.2. Subjective Immediate Response

To evaluate participants' immediate subjective responses to the videos, we compare subjects' response to the Film Response Questionnaire (comprising the following variables: valence, arousal, immobility, distress, disgust, embarrassment, involvement and attention) using Friedman's tests, as the variables were non-normally distributed. These were followed by Conover's pairwise post-hoc comparisons with Bonferroni correction for multiple testing. Age and anxiety (STAI) were included as covariates but showed no significant effects and stratified analyses by gender revealed only minor modulations of the main results.

Importantly, no significant differences were found across videos in terms of attention, which was assessed subjectively through self-reported ratings in the FRQ. Therefore, any differences observed in emotional or physiological measures cannot be attributed to variations in attentional engagement.

Additionally, the number of participants who reported moving their gaze from the screen did not differ significantly across videos. The analyses were further stratified for both dichotomous questionnaire variables (previous exposure to the video or its excerpts, and gaze aversion during viewing), and no significant effects or interactions were observed for either factor. Therefore, being familiar with the video seems to not reduce its stressing effect.

Results related to within-theme comparisons between categories are reported below (detailed statistical outputs are reported in Figures 10–16 and presented in Table 8 in Appendix C):

- Physical Theme: Friedman's tests indicated significant differences across emotional categories for all questionnaire dimensions. Negative films (PT1, PT2) were rated as significantly more *unpleasant* than neutral (PN) or positive (PP) videos ($Fr(3) = 103.4$, $p < 0.001$), elicited higher *arousal* (PT1, PT2 > PN/PP; $Fr(3) = 80.7$, $p < 0.001$) and induced significantly stronger *immobility* (PT1, PT2 > PN/PP; $Fr(3) = 68.1$, $p < 0.001$), *distress* (PT1, PT2 > PN/PP; $Fr(3) = 97.0$, $p < 0.001$) and *disgust* (PT1, PT2 > PN/PP; $Fr(3) = 110.5$, p e < 0.001). *Embarrassment* showed modest increases (PT1 > PN/PP; PT2 > PN; Fr(3) 33.7, $p < 0.001$). *Involvement* was higher for PT1/PT2 compared to PN ($Fr(3) = 27.7$, $p < 0.001$) but not to PP. Gender stratifications confirmed the same pattern.

PP did not differ significantly from PN in most of the measured dimensions, including *valence*, *arousal*, *immobility*, *distress*, *disgust,* and *embarrassment* (*all ps* > 0.05), the only exception was *involvement*, which was higher in PP compared to PN (Fr(3) = 27.7, $p < 0.001$).

- Relational Theme: Friedman's tests revealed significant differences across emotional categories. Negative films (RT1, RT2) were rated as significantly more *unpleasant* than neutral (RN) and positive (RP) videos (Fr(3) = 102.6, $p < 0.001$), and elicited higher *arousal* (RT1 > RN; RT2 > RN/RP; Fr(3) = 54.2, $p < 0.001$), *immobility* (RT1, RT2 > RN/RP; Fr(3) = 80.6, $p < 0.001$), *distress* (RT1, RT2 > RN/RP; Fr(3) = 92.2, $p < 0.001$), *disgust* (RT1, RT2 > RN/RP; Fr(3) = 111.0, $p < 0.001$), *embarrassment* (RT1, RT2 > RN/RP; Fr(3) = 57.8, $p < 0.001$) and *involvement* (RT1, RT2 > RN/RP; Fr(3) = 27.2, $p < 0.001$).

  Gender effects were generally small and non-significant, except for *immobility*, which was significantly higher for RT1 and RT2 compared to RN and RP in both males and females, but the effect was more pronounced in females (Fr(3) = 46.1, $p < .001$) than in males (Fr(3) = 36.1, $p < .001$). To further explore this, Mann–Whitney U tests were conducted between males and females for each relational video. No significant differences were observed, except for the RT2 condition, where females reported significantly higher *immobility* scores than males (U = 155.0, N = 38, $p = .015$; Female: 5.2 ± 2.7, Male: 3.0 ± 2.1).

  RP did not differ significantly from RN in most dimensions, including *valence*, *immobility*, *distress*, *disgust, involvement* and *embarrassment* (*all ps* > 0.05), with the exception of *arousal*, which was higher in RP compared to RN (Fr(3) = 54.2, $p < 0.001$).

- Traffic Theme: Friedman's tests indicated significant differences across films. Negative videos (TT1, TT2) were rated as more *unpleasant* than neutral (TN) and or positive (TP) clips (Fr(3) = 102.6, $p < 0.001$), with higher *arousal* (TT1, TT2 > TN/TP; Fr(3) = 74.8, $p < 0.001$), *immobility* (TT1, TT2 > TN/TP; Fr(3) = 56.1, $p < 0.001$), *distress* (TT1, TT2 > TN/TP; Fr(3) = 80.3, $p < 0.001$) and *involvement* (TT1, TT2 > TN/TP ; Fr(3) = 70.4, $p < 0.001$). *Disgust* levels were generally low for this theme, with only TT1 reaching significance (TT1 > TN/TP; Fr(3) = 53.6,

$p < 0.001$). *Embarrassment* showed a small effect, involving only TT1 (TT1 > TN; Fr(3) = 12.8, $p = 0.005$).

Stratified analyses revealed no gender effects.

TP did not differ significantly from TN in most dimensions, including *valence*, *immobility*, *distress*, *disgust* and *embarrassment*, except for *involvement* (TP > TN; Fr(3) = 70.4, $p < 0.001$) and *arousal* (TP > TN; Fr(3) = 74.8, $p < 0.001$).

Focusing on between theme negative video (PT1, PT2, RT1, RT2, TT1, TT2) comparison, Friedman's tests revealed significant main effects for all the measured dimensions, as detailed below:

- Valence: The most *unpleasant* films were PT1, RT1, and RT2, which were rated significantly lower in valence than PT2, TT1, and TT2 (Fr(5) = 45.4, $p < 0.001$). Among them, RT2 was rated as significantly more unpleasant than RT1 (Fr(5) = 45.4, $p < 0.001$), while other pairwise comparisons were non-significant. Overall, relational negatives and the first physical video were perceived as the most emotionally aversive stimuli, with RT2 emerging as the most unpleasant of all.
- Arousal: Negative videos were generally comparable in terms of *arousal*, with PT1 and RT2, elicited the highest arousal levels. Specifically, PT1 showed significantly higher arousal compared to almost all other negative films (PT1> PT2/RT1/TT1/TT2; Fr(5) = 32.9, $p < 0.001$), with the exception of RT2. RT2 comparison did not survived to Bonferroni correction, except for PT2 (RT2 > PT2; Fr(5) = 32.9, $p < 0.001$).
- Immobility: Ratings were higher for PT1 and RT1, though most differences between negative films did not remain significant after correction. After Bonferroni correction both RT1 and PT1 resulted higher in *immobility* compared to TT2 and PT2 (Fr(5) = 22.9, $p < 0.001$).
- Distress: RT2 produced the highest *distress* ratings, differing significantly from all the traffic and the second physical videos (RT2 > PT2/TT1/TT2; Fr(5) = 20.4, $p < 0.001$).
- Disgust: *Disgust* levels were very high for PT1, PT2, RT1, and RT2, and markedly lower for the traffic negatives (TT1, TT2). In particular, RT2 elicited significantly higher disgust compared to the videos in the other themes (RT2 > PT1/PT2/

TT1/TT2; Fr(5) = 158.2, $p < 0.001$). RT1 was also significantly higher than PT2 and both traffic films (RT1 > PT2/ TT1/TT2; Fr(5) = 158.2, $p < 0.001$). Both physical videos resulted significantly higher in disgust than the the traffic videos (PT1, PT2 > TT1/TT2; Fr(5) = 158.2, $p < 0.001$).

- Embarrassment: The highest *embarrassment* levels were observed for the relational negatives (RT1, RT2), followed by PT1. The traffic videos consistently elicited low embarrassment scores. Both RT1 and RT2 showed significantly higher level of embarrassment compared to all the other videos (RT1,RT2 > PT1/PT2/ TT1/TT2; Fr(5) = 92.1, $p < 0.001$). PT1 also elicited significantly higher level of embarrassment compared to the traffic videos (PT1, PT2 > TT1/TT2; Fr(5) = 92.1, $p < 0.001$).
- Involvement: Differences in *involvement* among negative films were generally small. After Bonferroni correction, only TT2 > PT2 remained significant (Fr(5) = 13.0, $p = 0.024$).

Stratified analyses revealed no gender effects. Detailed statistical outputs are shown in panel D of Figures 10–16 and reported in Table 9 in Appendix C.

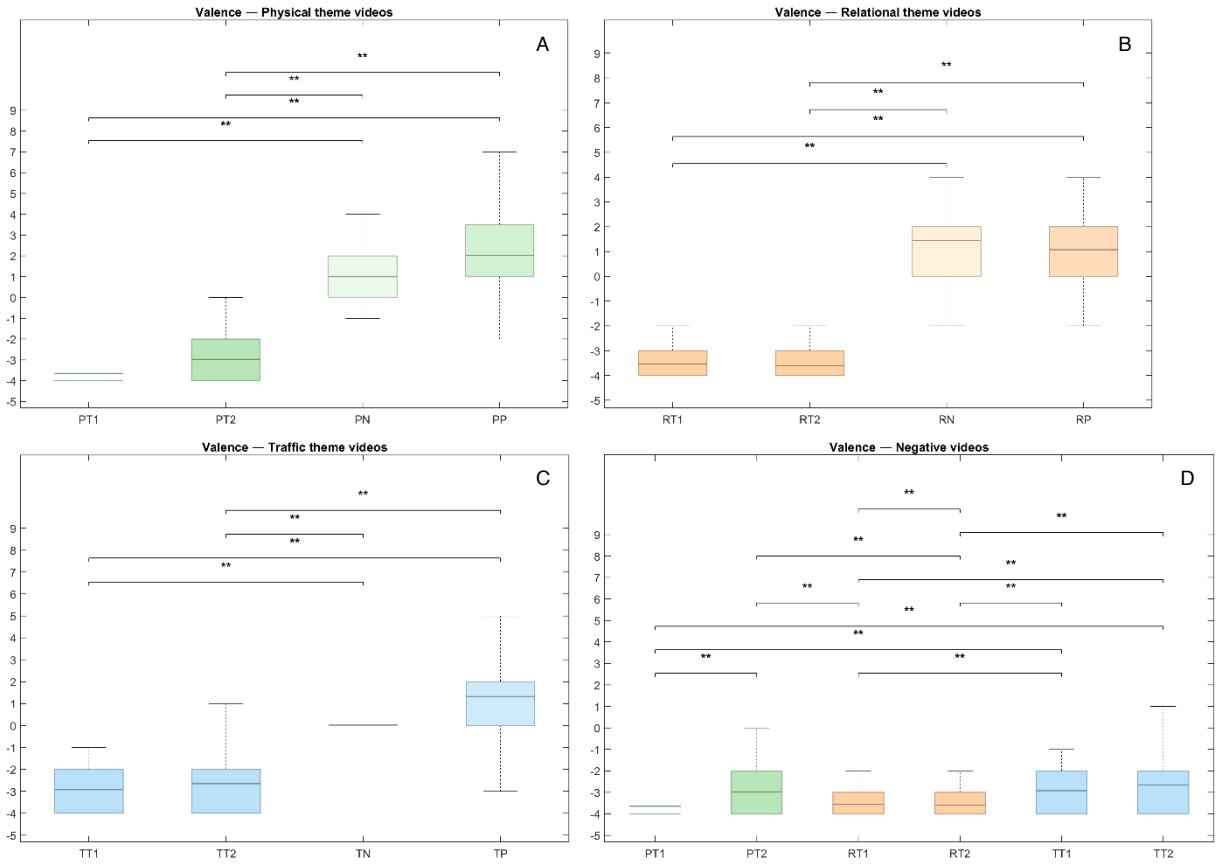
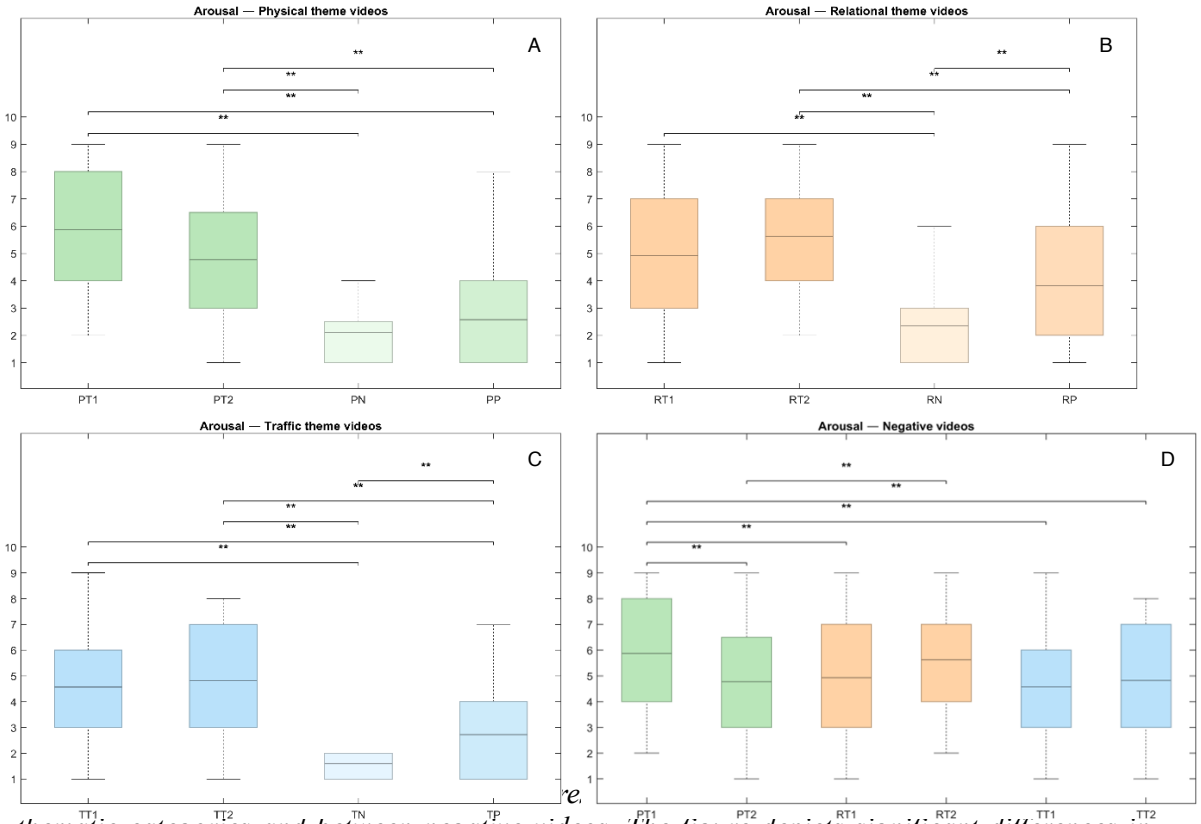


Figure 10. *Visual comparison of valence scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective valence responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison of valence scores among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** $p < 0.001$.*



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thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective arousal responses following video exposure.

Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.

*** p < 0.001.*

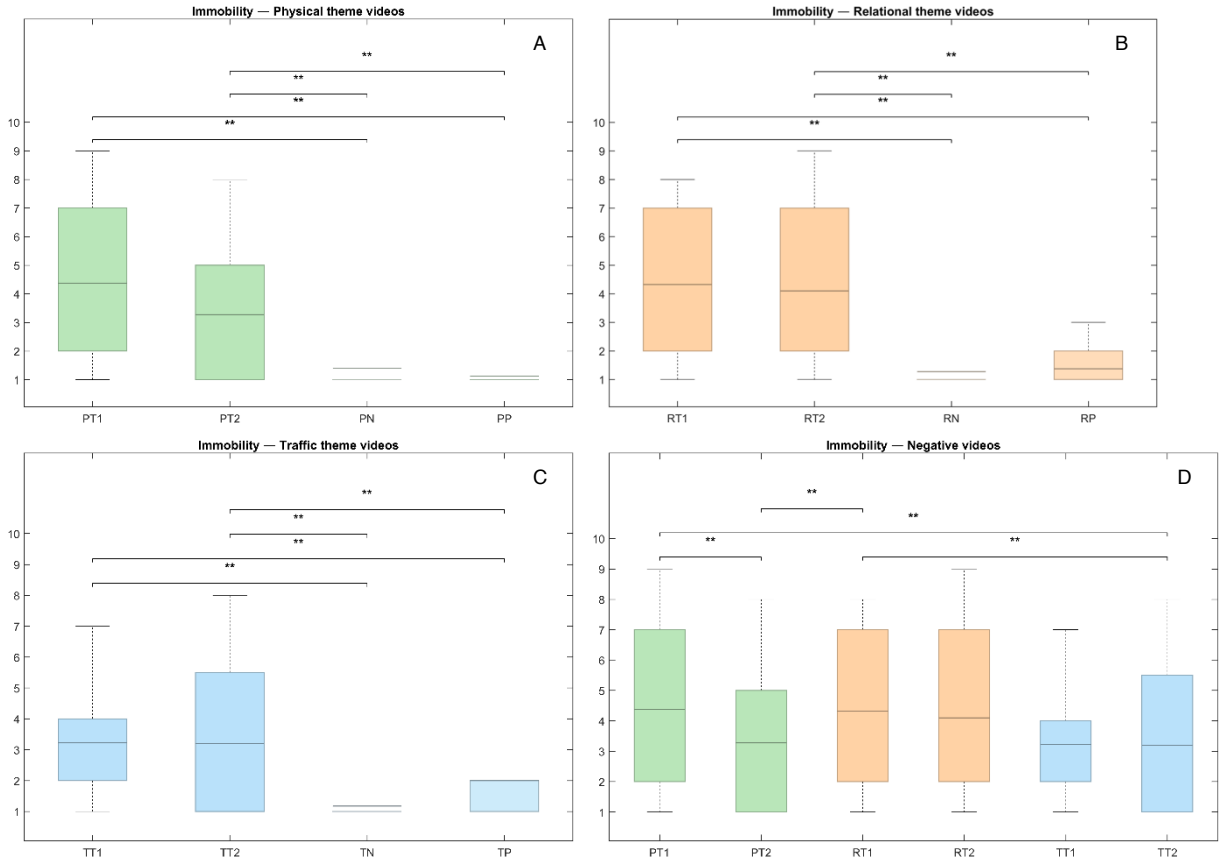

Figure 12. *Visual comparison of immobility scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective immobility responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** $p < 0.001$.*

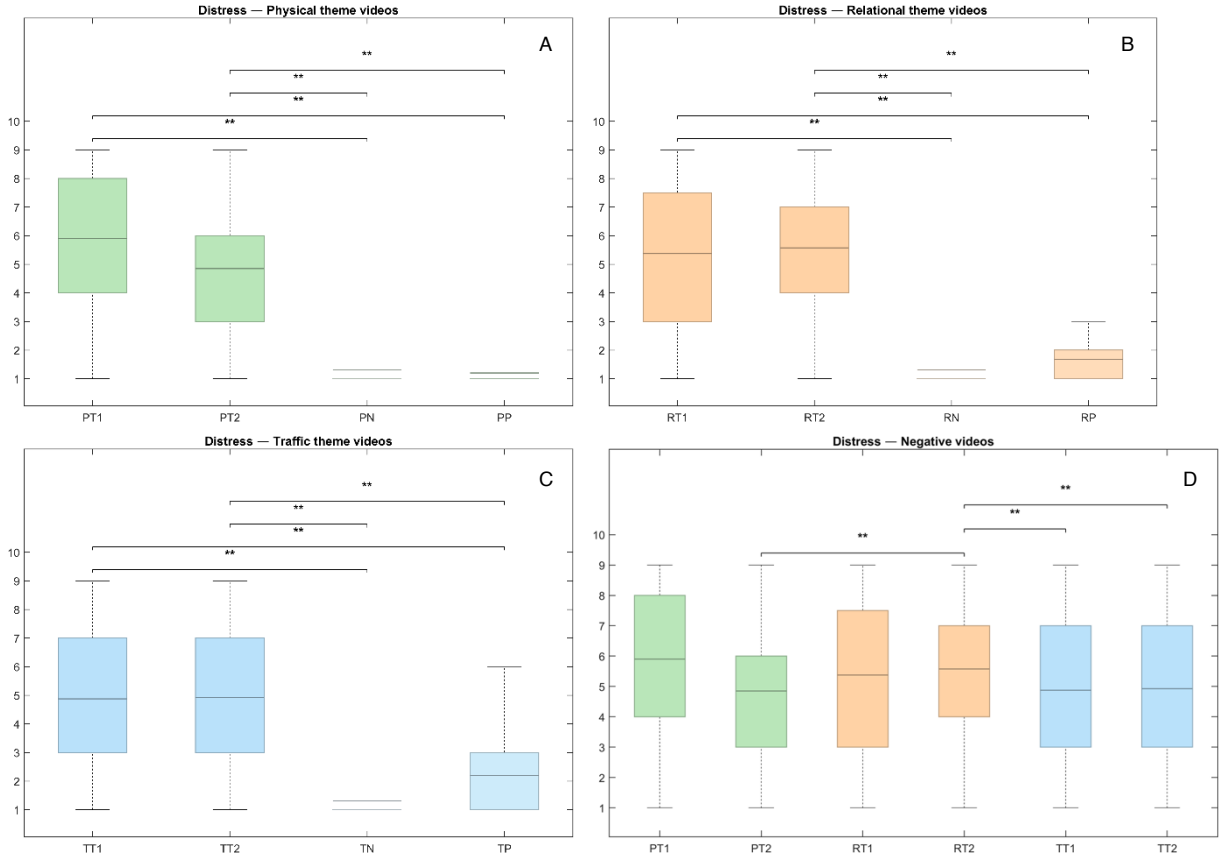

Figure 13. *Visual comparison of distress scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective distress responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** $p < 0.001$.*

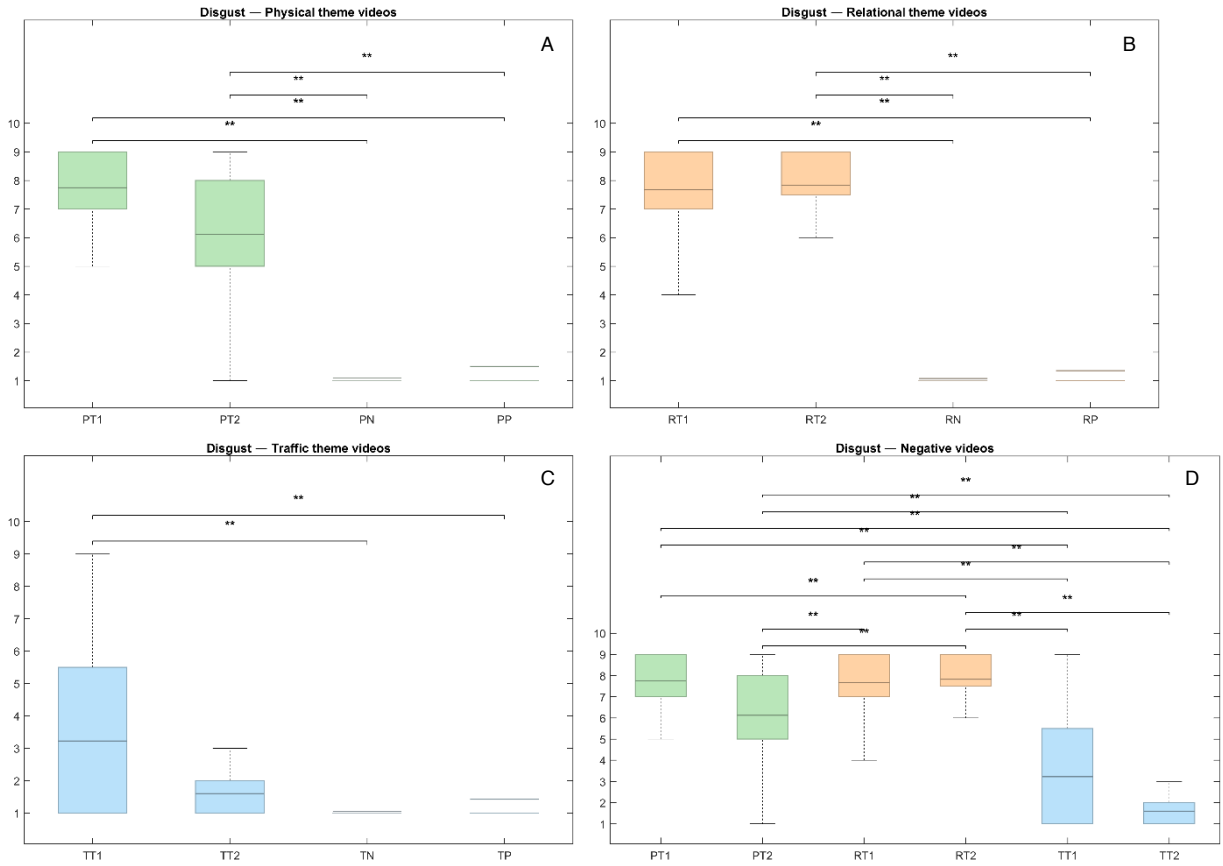

Figure 14. *Visual comparison of disgust scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective disgust responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** $p < 0.001$.*

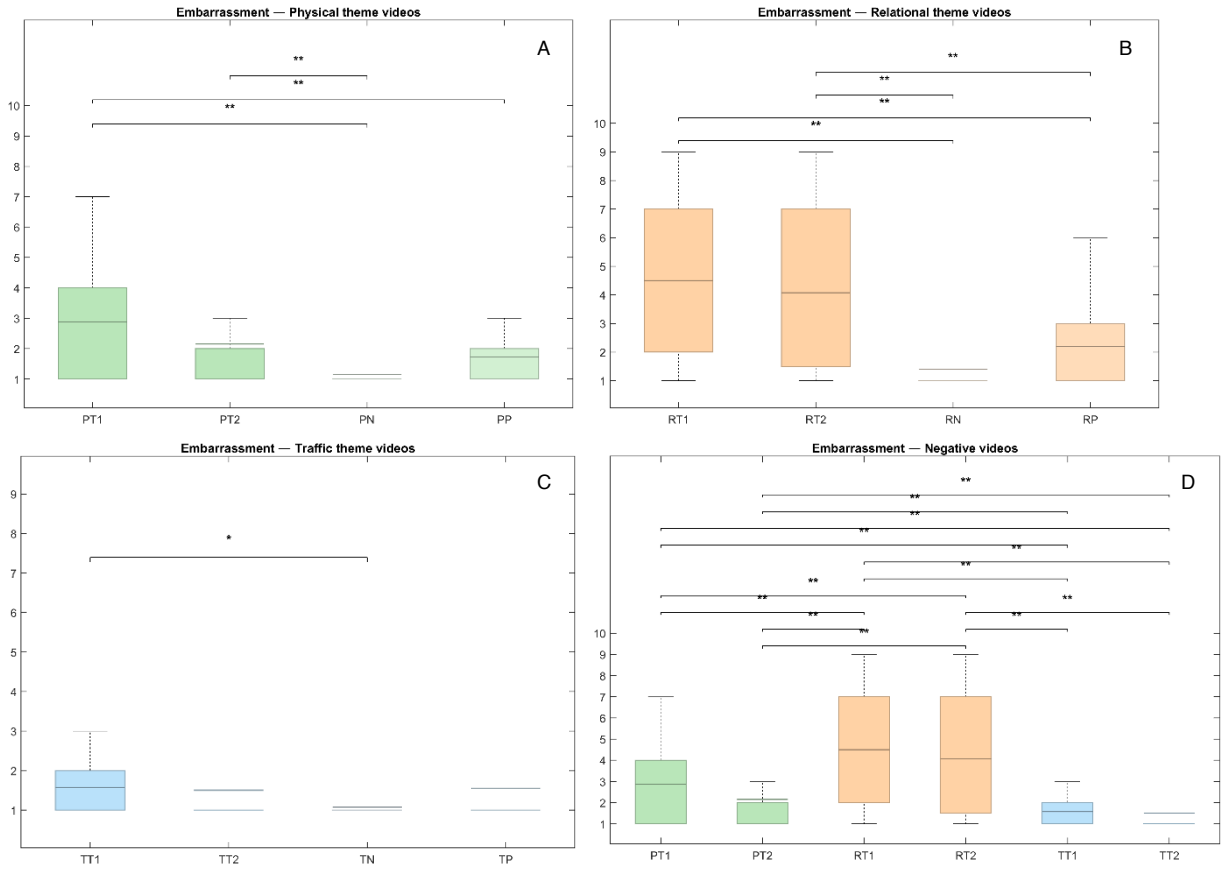

Figure 15. *Visual comparison of embarrassment scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective embarrassment responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
** $p < 0.05$; ** $p < 0.001$.*

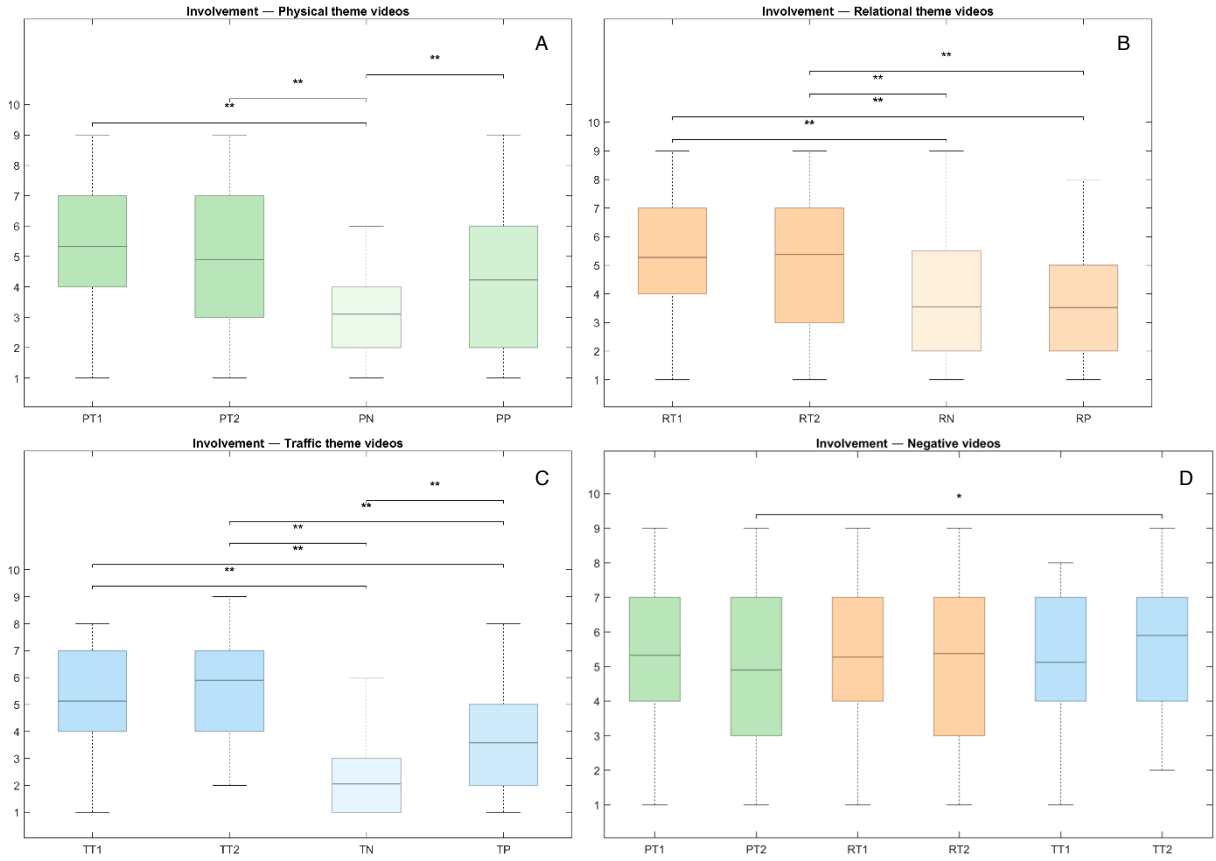

Figure 16. *Visual comparison of involvement scores from the Film Response Questionnaire across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate subjective involvement responses following video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
** $p < 0.05$; ** $p < 0.001$.*

5.2.3. Objective Immediate Response

Similarly to what we have done for the subjective response to evaluate participants' immediate subjective responses to the videos, we compare segments of PPG and EDA signals corresponding to different video administration first within each theme between video categories, and after focusing on negative videos only. In order to do that, as all the data followed a normal distribution, we used rmANOVA followed by post-hoc comparison with Bonferroni correction for multiple testing. Age and state and trait

anxiety (STAI) were included as covariates but showed no significant effects and no significant differences emerged from stratified analyses by gender.

5.2.3.1 PPG results

PPG analyses were conducted on the entire duration of the video. We extracted an HR index to measure autonomic activity. Importantly, we first checked the HR at baseline for all participants, and during the viewing of the mood-stabilizing video the HR was 78.41 ± 12.6 bpm, indicating a resting and emotionally neutral physiological state across the sample before target video administration. A visual representation of the comparisons is shown in Figure 17, while the detailed statistical outputs are presented in Table 10 in Appendix C.

Results related to within-theme comparisons between categories are reported below:

- Physical Theme: A significant main effect of video category on HR was found, ($F(3, 117) = 44.7$, $p < 0.001$) with mean HR was significantly lower during PT1 (72.8 ± 3.1) and PT2 (74.1 ± 3.2) compared to PN (78.6 ± 3.1) and PP (78.8 ± 4.4). No significant difference emerged between PN and PP.
- Relational Theme: A similar to the previous one, but stronger, pattern emerged, with a robust main effect of video category on HR ($F(3, 117) = 102.7$, $p < 0.001$). HR decreased markedly during both RT1 (70.9 ± 2.8) and RT2 (72.5 ± 2.9) compared RN (78.5 ± 2.9) and RP (79.0 ± 4.7). Moreover, RT1 elicited a significantly larger HR deceleration than RT2. No difference was found between RN and RP.
- Traffic Theme: From the rmANOVA analysis a significant main effect of the video category on HR was observed ($F(3, 117) = 17.3$, $p < 0.001$) with HR significantly lower during TT1 (75.3 ± 3.7) and TT2 (75.8 ± 3.5) compared to TN (78.7 ± 2.1) and TP (79.1 ± 5.8). Once again, no significant difference was found between TN and TP.

After the within-theme analyses, we investigated whether there were differences in HR responses between all the negative videos, as we did for the subjective response. A rmANOVA between the negative videos of all themes revealed a significant main effect emerged on HR ($F(5, 195) = 16.0$, $p < 0.001$). Post-hoc Bonferroni comparisons showed

that RT1 elicited a significantly stronger HR decrease compared to all other videos (RT1 < RT2/PT1/PT2/TT1/TT2; $F(5, 195) = 16.0$, $p < 0.001$). Additionally, RT2 and both the negative physical videos showed a significantly stronger decrease compared to both the traffic videos (RT2, PT1, PT2 < TT1/TT2; $F(5, 195) = 16.0$, $p < 0.001$).

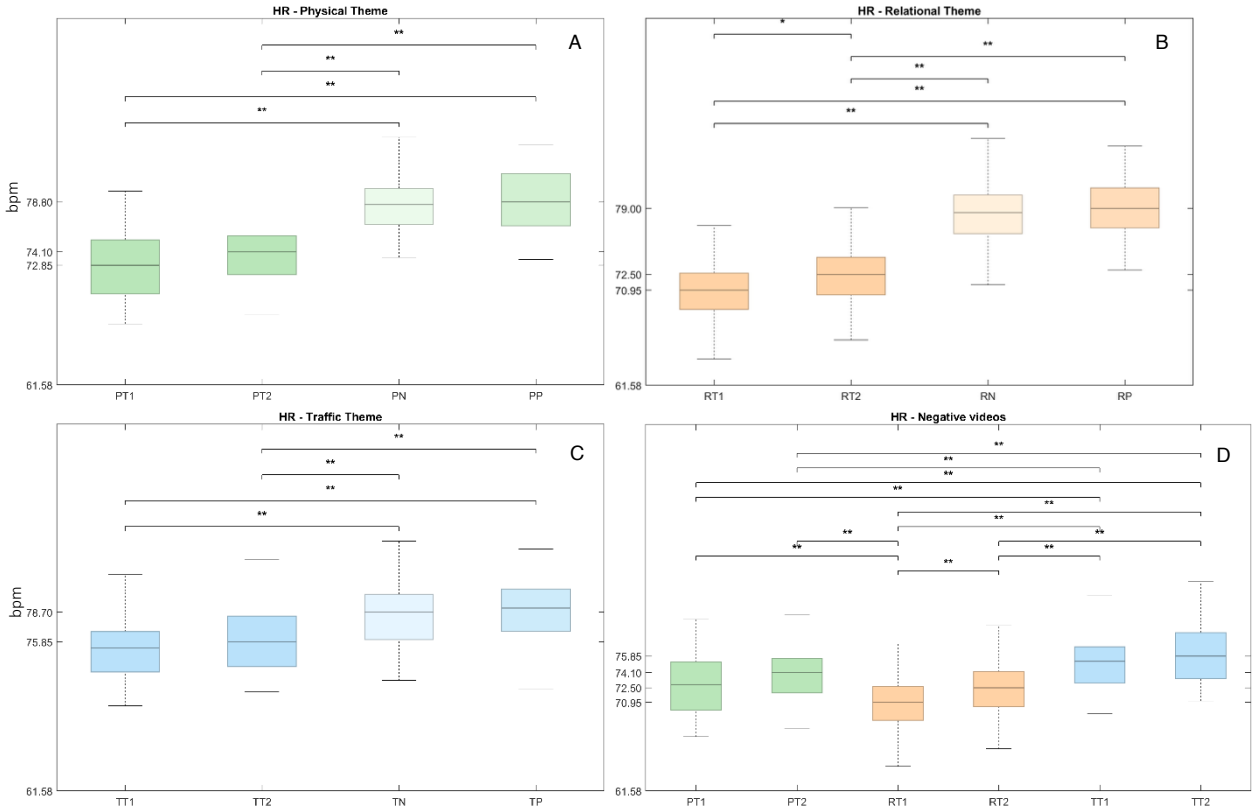

Figure 17. *Visual comparison of Heart Rate (HR) means values across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate physiological response during video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video; HR = Heart Rate; bpm = beats per minute.
*** $p < 0.001$.*

5.2.3.2 EDA results

To compare EDA signal, we selected a common 60-second analysis window for all videos, with the 30-second time point approximately aligned with the emotional peak or pivotal moment of the clip.

From the CDA output, we extracted four indices of sympathetic activation: CDA.AmpSum, CDA.SCR, CDA.ISCR and one index of tonic activity, CDA.Tonic. Notably from the rmANOVA analysis no significant difference emerge in terms of tonic activity (CDA.Tonic).

Notably, data from three participants were excluded from this analysis due to poor signal quality, and in one case, due to a failure in EDA recording.

At baseline, during a 60-second window of viewing of the mood-stabilizing video, the mean values of the indices were as follows: CDA.AmpSum = 0.35 ± 0.19 μS, CDA.SCR = 0.07 ± 0.04 μS, CDA.ISCR = 2.02 ± 0.87 μS·s, and CDA.Tonic = 4.89 ± 1.48 μS.

Results related to within-theme comparisons between categories are reported below (see Figures 18, 19, 20 and Table 11 in Appendix C):

- Physical Theme: From rmANOVA a significant main effect of video category on CDA.AmpSum emerged ($F(3,108) = 77.9$, $p < 0.001$), with PT1 and PT2 showing significantly higher values (0.54 ± 0.32; 0.45 ± 0.25) compared to PN (0.33 ± 0.19) and PP (0.34 ± 0.02), which did not differ from each other. Considering CDA.SCR, the effect of video category was also significant ($F(3,108) = 96.85$, $p < 0.001$): PT1 (0.12 ± 0.05) and PT2 (0.10 ± 0.05) elicited higher responses than PN (0.08 ± 0.03) and PP (0.07 ± 0.03), which were statistically comparable. A similar pattern was observed for CDA.ISCR ($F(3,108) = 133.5$, $p < 0.001$), with PT1 and PT2 (3.56 ± 1.54; 2.95 ± 1.29) significantly higher than PN (2.04 ± 0.90) and PP (2.02 ± 0.84), which did not differ.
- Relational Theme: For CDA.AmpSum, a robust main effect of video category was observed ($F(3,108) = 104.3$, $p < 0.001$), with RT1 (0.56 ± 0.30) and RT2 (0.53 ± 0.27) showing higher values than RN (0.36 ± 0.20) and RP (0.33 ± 0.17), which did not differ from each other. Regarding CDA.SCR, the effect was also significant ($F(3,108) = 89.3$, $p = 0.003$) and RT1 (0.13 ± 0.05) elicited higher responses than RN (0.08 ± 0.03) and RP (0.08 ± 0.03), which were

comparable both to each other and to RT2 (0.11 ± 0.04). For CDA.ISCR, the main effect was again significant ($F(3,108) = 134.9$, $p < 0.001$), with RT1 and RT2 (3.56 ± 1.54; 2.97 ± 1.28) significantly higher than RN (2.03 ± 0.84) and RP (2.04 ± 1.83), which did not differ from each other.

- Traffic Theme: For CDA.AmpSum, the rmANOVA revealed a significant main effect of video category ($F(3,108) = 14.3$, $p = 0.007$). TT1 (0.42 ± 0.24) showed higher values than TN (0.33 ± 0.19) and TP (0.26 ± 0.15), while TT2 (0.34 ± 0.20) did not differ from TN but was higher than TP. Additionally, TP was significantly lower than RN. For CDA.SCR, no significant differences between categories were found ($F(3,108) = 47.4$, $p = 0.067$). CDA.ISCR showed a significant main effect ($F(3,108) = 28.1$, $p = 0.018$), mirroring the AmpSum pattern: TT1 (2.69 ± 1.14) was higher than TN (2.01 ± 0.87) and TP (1.51 ± 0.70); TT2 (2.01 ± 0.84) did not differ from TN but was higher than TP; which did not differ from each other.

We also compared CDA responses across all negative videos to examine whether differences emerged between themes (see Table 12 and panel D of Figures 18, 19, 20).

For CDA.AmpSum, a significant main effect of video category was found ($F(5,180) = 69.2$, $p = 0.011$). Post-hoc Bonferroni comparisons showed that PT1, RT1, and RT2 elicited comparable and significantly higher amplitudes compared to PT2, TT1, and TT2, with PT2 in turn higher than TT2.

For CDA.SCR, the analysis revealed a significant main effect ($F(5,180) = 87.7$, $p = 0.037$). RT1 and PT1 showed significantly greater SCR amplitudes than all other videos, which did not differ from each other, except for RT2, whose value was not significantly higher than any of the remaining conditions.

For CDA.ISCR, a significant main effect of video category was also observed ($F(5,180) = 115.5$, $p < 0.001$). RT1 and PT1 elicited the largest responses, significantly exceeding all other videos. Additionally, PT2, RT2, and TT1 showed higher ISCR values compared to TT2.

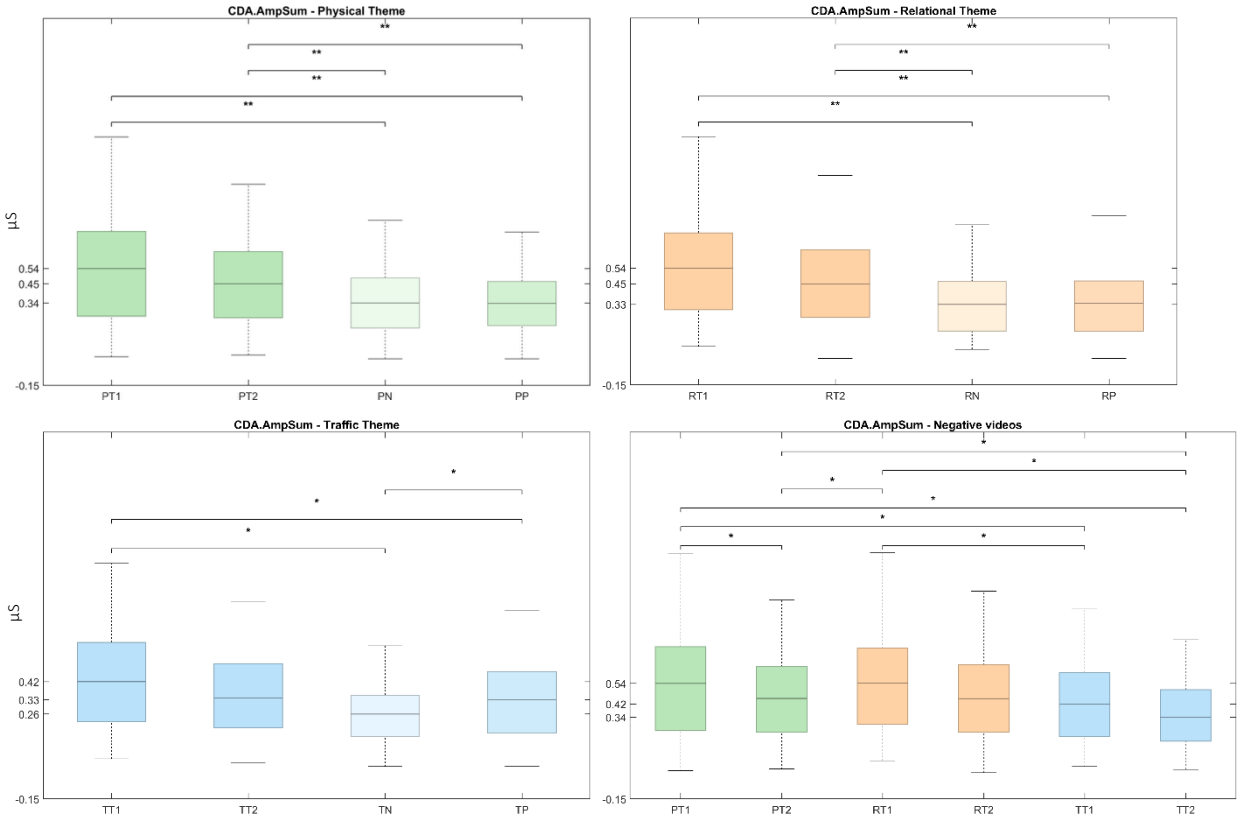

Figure 18. *Visual comparison of CDS.AmpSum means values across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate physiological response during video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video; CDA = Conductance Data Analysis; AmpSum = amplitude sum of SCRs; SCR = Skin Conductance Response; µS = microsiemens.
** $p < 0.05$; ** $p < 0.001$.*

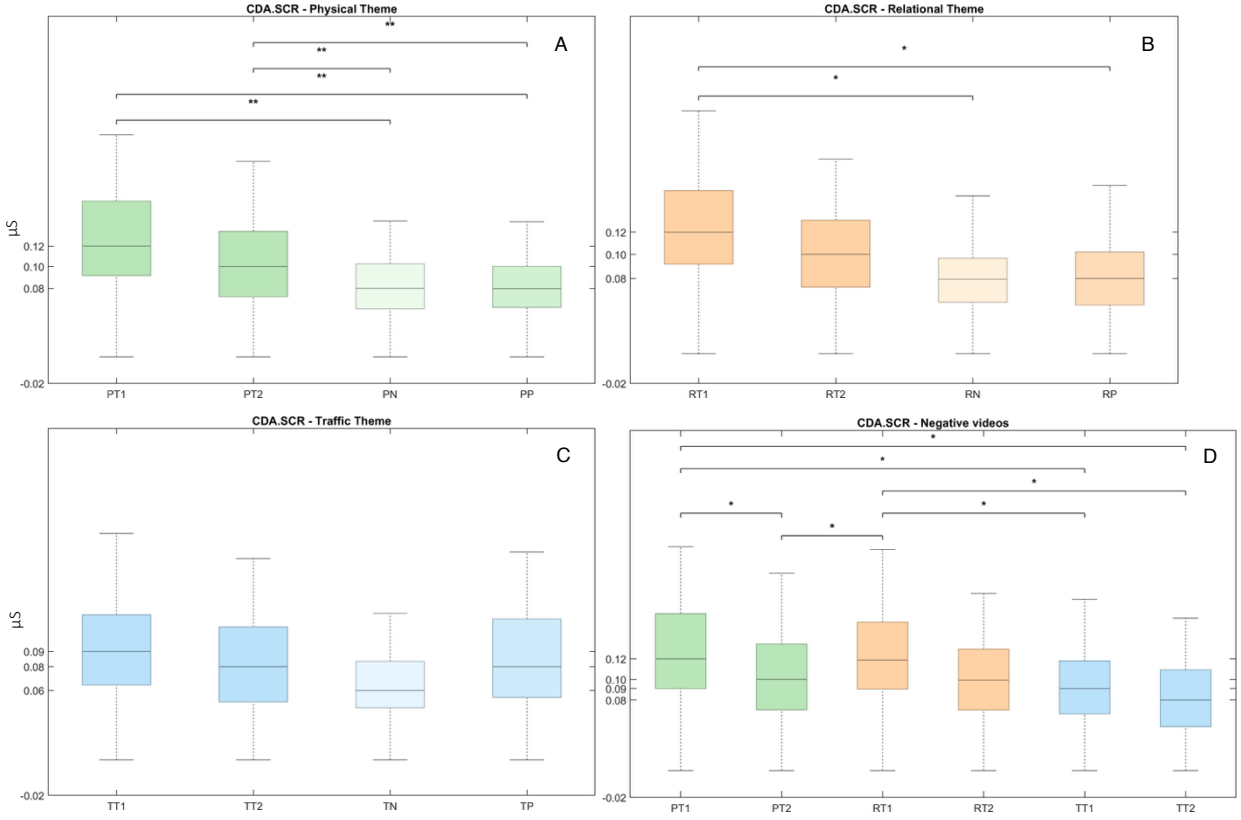

Figure 19. *Visual comparison of CDS.SCR means values across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate physiological response during video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video; CDA = Conductance Data Analysis; SCR = Skin Conductance Response; μS = microsiemens.
** $p < 0.05$; ** $p < 0.001$.*

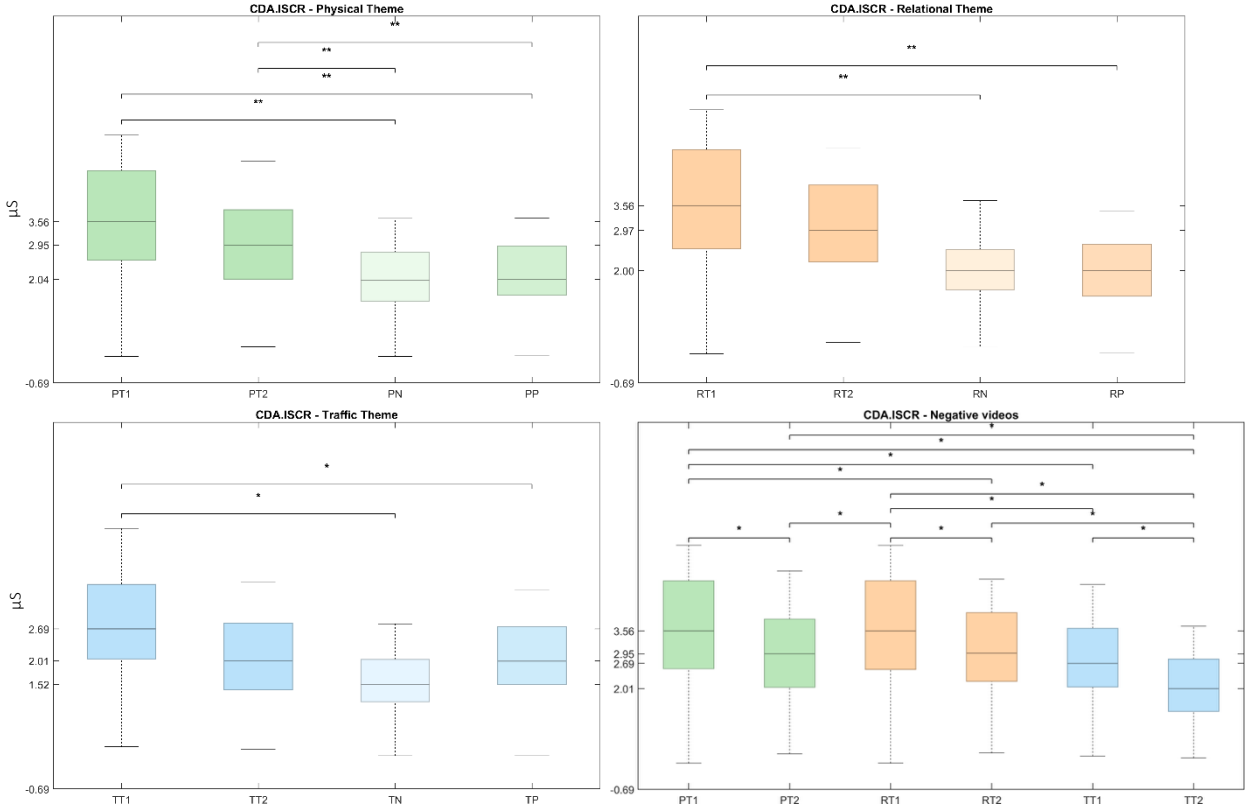

Figure 20. *Visual comparison of CDS.ISCR means values across thematic categories and between negative videos. The figure depicts significant differences in participants' immediate physiological response during video exposure.*
Three boxplots illustrate comparisons within each thematic group: [A] comparison between physical theme videos (PT1, PT2, PN, PP), [B] comparison between relational theme videos (RT1, RT2, RN, RP), [C] comparison between traffic theme videos (TT1, TT2, TN, TP). Panel [D] presents the comparison among all negative videos across themes. PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video; CDA = Conductance Data Analysis; ISCR = Integrated Skin Conductance Response; µS = microsiemens.
** $p < 0.05$; ** $p < 0.001$.*

5.2.4. Subjective delayed response: Sleep Diaries

Participants completed a Sleep Diary for the entire duration of the protocol, namely for seven days before and seven days after the experimental session. To evaluate the impact of the videos on subjective sleep, two different time windows were considered:

1. The entire week before and the entire week after the session;
2. The two days immediately preceding the experimental session and the two days following it (acute phase).

The second time window was chosen because the TFP paradigm is designed to mimic a traumatic experience, typically producing not only a milder level of distress but also a short-lived effect. We therefore aimed to verify whether this transient impact could also be observed in subjective sleep measures.

Sleep diary data were analysed using rmANOVA or, when normality assumptions were violated, the Friedman test. Age was included as a covariate, and the analyses were stratified by gender. However, neither age nor gender produced significant effects or interactions, and results are therefore reported for the overall sample. Results are summarized in Table 13.

Sleep Index	Derivation Method	Week Pre (M ± SD)	Week Post (M ± SD)	Stat	2 Days Pre (M ± SD)	2 Days Post (M ± SD)	Stat
SL_min	Estimated by the subject	7.9 ± 4.9	9.6 ± 7.4	2.4†	6.9 ± 4.9	13.3 ± 7.7	37.0**†
TIB_min	Lights out time – (Woke up time + Time in bed after awakening)	493.8 ± 45.0	503.6 ± 57.4	1.6	490.4 ± 58.9	508.0 ± 71.7	2.5
sTST_min	Estimated by the subject	448.1 ± 37.44	443.9 ± 43.8	0.5	451.5 ± 53.7	439.1 ± 59.8	7.6
sSE%	sTST/TIB	91.1 ± 5.9	88.9 ± 6.8	8.2*	92.6 ± 7.2	85.2 ± 8.0	24.6**†
cTST_min	Woke up time – (Lights out time + SL + WASO)	439.4 ± 37.9	435.2 ± 45.3	2.5	443.5 ± 52.1	431.8 ± 61.2	4.2
cSE%	cTST/TIB	89.2 ± 5.8	87.8 ± 6.5	4.1	90.7 ± 5.9	84.7 ± 7.3	27.9**†
NAWK	Estimated by the subject	0.9 ± 0.8	0.8 ± 0.8	0.7†	0.8 ± 0.8	0.9 ± 1.0	0.4†
WASO_min	Estimated by the subject	11.2 ± 10.4	19.7 ± 21.1	6.5*†	8.7 ± 12.7	22.2 ± 17.8	27.0**†
Dreams	Reported by the subject	1.1 ± 1.0	1.1 ± 0.9	0.2†	2.7 ± 2.1	3.4 ± 2.1	6.3*†
Deepness	Reported by the subject	3.9 ± 0.8	3.7 ± 0.8	1.6	3.4 ± 0.7	3.7 ± 0.8	13.7*†
Quietness	Reported by the subject	2.1 ± 0.8	2.2 ± 0.9	1.0	2.2 ± 0.7	2.2 ± 0.8	0.2†
Restorativeness	Reported by the subject	3.5 ± 1.0	3.2 ± 0.8	13.8	3.4 ± 0.7	3.2 ± 0.8	4.3*†

Table 13. *Comparison between sleep quality indices from the Sleep Diary collected during the week before and the week after the experimental session (left panel), and during the two days before and the two days after the session (right panel). Data were analysed using rmANOVA or, when normality assumptions were violated, the Friedman test. Derivation mode indicates how each index was obtained: most parameters were directly reported or estimated by participants, whereas others were calculated from their estimates. "Lights out time" refers to the moment participants decided to stop any activities in order to fall asleep.*
Stat = Statistic; SL_min = Sleep Latency (minutes); TIB_min = Time in Bed (minutes); sTST_min = Subjective Total Sleep Time (minutes); sSE% = Subjective Sleep Efficiency; cTST_min = Calculated Total Sleep Time (minutes); cSE% = Calculated Sleep Efficiency; NAWK = Number of Awakenings; WASO_min = Wake After Sleep Onset (minutes); Dreams = Number of Dreams; Deepness = Perceived Sleep Depth; Quietness = Perceived Sleep Quietness; Restorativeness = Perceived Sleep Restorativeness; M = mean; SD = standard deviation.
† Friedman test was conducted
** $p < 0.05$; ** $p < 0.001$.*

As expected, when comparing the entire week before with the week after the experimental session, only a few comparisons reached the significance threshold. Specifically, we observed a significant decrease in subjective SE% (sSE%) from the pre- to the post-week ($F(1,39) = 8.2$, $p = 0.007$) and a significant increase in WASO ($Fr(1) = 6.5$, $p = 0.011$). Two additional comparisons approached statistical significance, both suggesting a worsening in perceived sleep quality: a trend toward longer SL during the post-week ($Fr(1) = 2.4$, $p = 0.067$) and an increase in calculated SE% (cSE%) following video viewing ($F(1,39) = 4.1$, $p = 0.051$). Significant comparisons are illustrated in Figure 21.

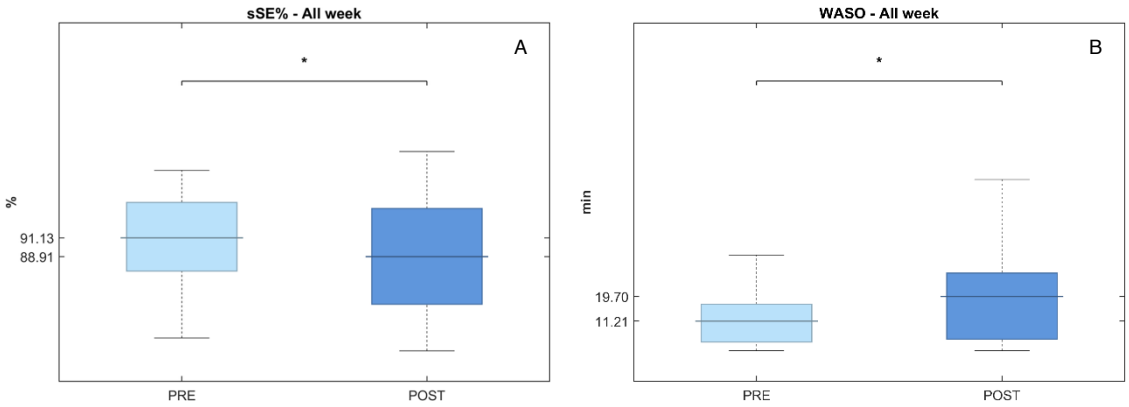

Figure 21. *Visual comparison of subjective sleep indices extracted from the Sleep Diary, considering the entire week before and after the experimental session. The figure depicts only the comparisons showing significant differences in participants' delayed psychological responses to video exposure.*
[A] Boxplots illustrating the significant comparison for subjective sleep efficiency (sSE%). [B] Boxplots illustrating the significant comparison for Wake After Sleep Onset (WASO) express in minutes. sSE% = subjective Sleep Efficiency; WASO = Wake After Sleep Onset; min = minutes.
** $p < 0.05$.*

On the other hand, when focusing on the period surrounding video administration (two days before vs. two days after), a much more consistent picture of decreased subjective sleep quality emerged. We observed a significant increase in SL after video exposure ($Fr(1) = 37.0$, $p < 0.001$), a significant decrease in both subjective and calculated SE% (sSE%: $Fr(1) = 24.6$, $p < 0.001$; cSE%: $Fr(1) = 27.9$, $p < 0.001$), and a significant increase in WASO ($Fr(1) = 27.0$, $p < 0.001$). In addition, a significant increase in the number of dreams have been observed during the two days after the session ($Fr(1) = 6.3$, $p = 0.012$). Finally, the subjective sense of sleep satisfaction significantly decreased in the post-video

period, in terms of perceived deepness and restorativeness of sleep (both evaluated on a six-point Likert scale) (Deepness: Fr(1) = 13.7, $p = 0.002$; Restorativeness: Fr(1) = 4.3, $p = 0.008$).

No significant changes were detected for all other indices in the two time windows, including time in bed (TIB), subjective and calculated TST, number of awakenings (NAWK), or perceived quietness (*all ps* > 0.05). All the significant results are illustrated in Figure 22.

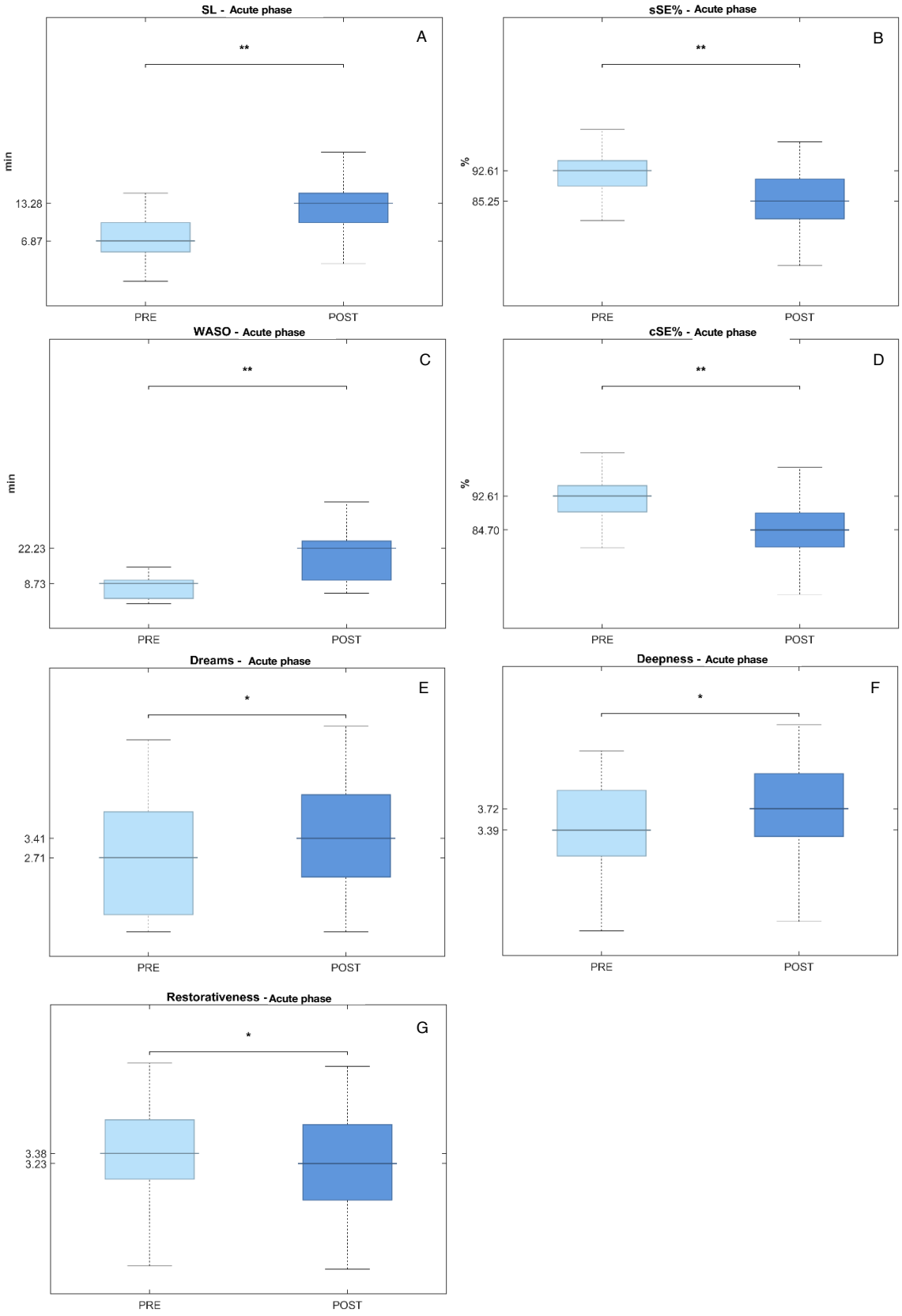

Figure 22. *Visual comparison of subjective sleep indices extracted from the Sleep Diary, considering only the acute phase of the experimental session (two days before vs. two days after). The figure depicts only the comparisons showing significant differences in participants' delayed psychological responses to video exposure.*

*[A] Boxplots illustrating the significant comparison for subjective sleep latency (SL) express in minutes. [B] Boxplots illustrating the significant comparison for subjective sleep efficiency (sSE%). [C] Boxplots illustrating the significant comparison for Wake After Sleep Onset (WASO) express in minutes. [D] Boxplots illustrating the significant comparison for calculated sleep efficiency (cSE%). [E] Boxplots illustrating the significant comparison for dream rate. [F] Boxplots illustrating the significant comparison for deepness score. [G] Boxplots illustrating the significant comparison for restorativeness score. SL = Sleep Latency; sSE% = subjective Sleep Efficiency; WASO = Wake After Sleep Onset; cSE% = calculated Sleep Efficiency; min = minutes. * $p < 0.05$; ** $p < 0.001$.*

5.2.5. Subjective delayed response: Intrusion Diary and IES-R

Lastly, we considered the post-traumatic symptom measures (Intrusion Diary and IES-R questionnaire) to further investigate the impact of the (hopefully) stressful videos and the temporal extent of this impact. We also examined the presence of a relationship between the delayed response and the immediate psychological and physiological responses to the videos. Notably, correlations between EDA and both the Intrusion Diary and the IES-R were not reported in the tables or discussed in the text, as none of them reached statistical significance.

5.2.5.1 Intrusion Diary

Participants reported a total number of 126 intrusions during the week following the experimental session. Unfortunately, as expected, compliance with the daily phone check-ins was relatively low, therefore, this number refers to the overall number of intrusions reported each day via phone to the main experimenter. In contrast, in the Intrusion Diary participants recorded a total of 61 intrusions (see Table 14). Most of these were related to PT1 and both the relational videos (PT1: 14/61; RT1: 15/61; RT2: 14/61), while the least frequently reported was PT2 (3/61). Both traffic videos showed medium reporting rates (TT1: 8/61; TT2: 7/61).

Based on the phone-reported data, participants experienced an average of 3.1 ± 3.6 intrusions during the week after the experimental session. To examine whether the effect of the videos rapidly diminished over time, similarly to what we observed for the impact on subjective sleep, we compared the number of intrusions reported during the first two days after video exposure with those reported during the last two days of the week using a Wilcoxon signed-rank test, as the data were not normally distributed. A significant

decrease was observed from the first two days to the last two ($Z = 3.7$, $p < 0.001$) consistent with the patterned found in sleep measures.

Associated rating of vividness, discomfort and control showed medium-high average scores (assessed on a seven-point Likert), indicating that most intrusion were characterized by moderate to high perceptual vividness (5.1 ± 1.2) and accompanied by a medium-high level of stress (4.1 ± 1.5) and they perceived a medium-high level of control (5.2 ± 1.2) in manipulating their intrusion. Conversely, spontaneity showed a medium-low mean value (3.4 ± 1.2), suggesting that most of the intrusions were not completely spontaneous but often triggered by external cues. Comparisons of these qualitative rating between the first and the last two days of the week revealed no significant differences, although the decreased in vividness approached the significance in the paired sample t test ($t = -2.0$, $p = 0.059$).

		All week (M ± SD)	First 2 Days (M ± SD)	Last 2 Days (M ± SD)	Statistic
Number of intrusions		3.1 ± 3.6	1.9 ± 2.1	0.8 ± 1.2	302.5**†
Vividness		5.1 ± 1.2	5.1 ± 1.1	4.6 ± 1.7	-2.0
Discomfort		4.1 ± 1.5	4.2 ± 1.8	3.6 ± 1.7	7.0†
Control		5.2 ± 1.2	5.2 ± 1.2	4.9 ± 1.4	13.0†
Spontaneity		3.4 ± 1.2	3.5 ± 1.2	2.8 ± 1.5	12.0†
Intrusion related to specific videos		**Tot (n)**	**First 2 Days (n)**	**Last 2 Days (n)**	
Physical	PT1	14	8	3	
	PT2	3	3	0	
Relational	RT1	15	9	3	
	RT2	14	10	1	
Traffic	TT1	8	5	3	
	TT2	7	6	1	

***Table 14**. Descriptive statistics of Intrusion Diary data and comparison between the number of intrusions reported during the first two days of the diary and those reported during the last two days. Data were analysed using paired-samples t-tests or, when normality assumptions were violated, the Wilcoxon signed-rank test (upper part of the table). Data reported in the table refers to the 61 intrusions reported in the Intrusion Diary, with the exception of the data about the "Number of intrusions" that refers to the total number of intrusions reported in general. The lower part of the table reports the frequency distribution of intrusions specifically related to each video.*

M = mean; SD = standard deviation; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); Tot = Total number of intrusions experienced after the experimental session; n = Number of intrusion.

† Wilcoxon signed-rank test was conducted

*** $p < 0.001$.*

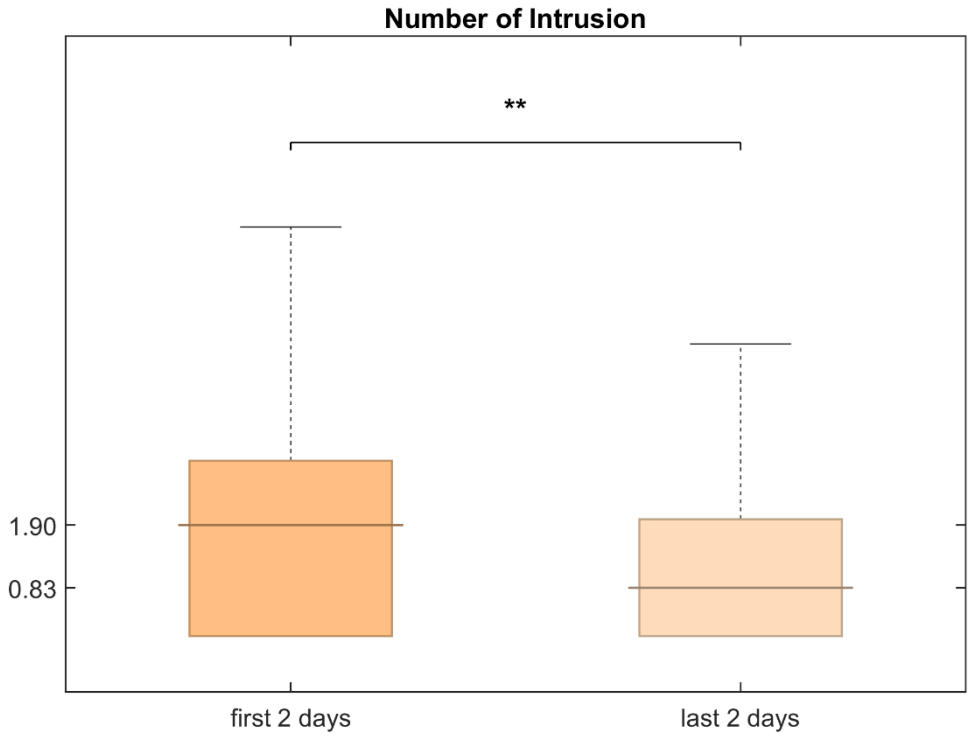

Figure 23. *Visual comparison of the number of intrusions reported in the Intrusion Diary during the first two days after the video session and the last two days. The figure displays only the comparison showing a significant difference in participants' delayed psychological responses to video exposure.*
*** p < 0.001.*

To explore the relationship between the immediate psychological and physiological response (PPG data) to the videos and the delayed impact measured through intrusions, Spearman's non-parametric rho (ρ) test was computed, as the data were not normally distributed. Analyses were also stratified by gender, but no significant differences emerged between male and female participants; therefore, results are reported for the overall sample. Correlation results are reported in Table 15.

Theme		Intrusions	Valence (rho)	Arousal (rho)	Immob (rho)	Distress (rho)	Disgust (rho)	Embarrass (rho)	Involv (rho)	HR (rho)
Physical	PT1	**Specific**	0.016	0.216	0.159	0.212	0.162	0.084	0.215	0.090
		2 days	-0.275	0.205	0.232	0.238	0.215	0.109	0.360*	0.204
		Tot	-0.253	0.273	0.187	0.233	0.170	0.102	0.397*	0.361*
	PT2	**Specific**	-0.255	0.391*	0.391*	0.332*	0.385*	-0.092	0.067	0.014
		2 days	-0.280	0.233	0.360*	0.141	0.113	0.172	0.309	0.378*
		Tot	-0.355*	0.255	0.381*	0.159	0.106	0.142	0.350*	0.267
Relational	RT1	**Specific**	-0.125	0.062	0.214	0.340*	0.377*	0.170	0.304	0.393*
		2 days	-0.157	0.344*	0.331*	0.302	0.399*	-0.034	0.372*	0.321*
		Tot	-0.210	0.397*	0.343*	0.345*	0.446*	-0.060	0.355*	0.401*
	RT2	**Specific**	0.023	0.178	0.407*	0.287	0.017	0.260	0.245	0.199
		2 days	-0.250	0.250	0.359*	0.248	0.172	0.009	0.363*	0.375*
		Tot	-0.256	0.241	0.405*	0.221	0.181	0.059	0.419*	0.381*
Traffic	TT1	**Specific**	-0.108	0.125	0.047	0.159	0.033	0.032	0.168	0.145
		2 days	-0.009	-0.078	-0.118	0.039	-0.143	0.193	0.144	0.127
		Tot	-0.104	0.106	0.035	0.139	-0.089	0.212	0.281	0.201
	TT2	**Specific**	-0.326*	0.197	0.350*	0.187	0.108	0.067	0.086	0.178
		2 days	-0.142	-0.055	0.042	-0.002	0.009	-0.087	0.165	0.154
		Tot	-0.223	-0.026	0.138	-0.008	0.025	-0.059	0.211	0.196

Table 15*. Correlation between immediate psychological reactions, Heart Rate (HR) and intrusions using the Spearman's non-parametric rho test.*
PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); Specific = intrusions specifically related to that video; 2 days = Total number of intrusions experienced in the first 2 days following the experimental session; TOT = Total number of intrusions experienced after the experimental session; Immob = Immobility; Embarrass = Embarrassment; Involv = Involvement; HR = Heart Rate.
** $p < 0.05$.*

Overall, the pattern of correlations revealed that intrusions were more strongly associated with the response to videos in the relational and physical themes, whereas response to traffic-related videos showed no consistent associations with the intrusion.

- Physical Theme: The number of intrusions specifically related to PT1 was positively correlated with participant involvement to the video ($\rho_{spearman} = 0.360$, $p = 0.024$) whereas the total number of intrusions correlates with the HR ($\rho_{spearman} = 0.361$, $p = 0.046$) and the involvement score during PT1 viewing ($\rho_{spearman} = 0.397$, $p = 0.011$). The number of intrusions associated with PT2 showed positive correlations with PT2 scores of arousal ($\rho_{spearman} = 0.391$, $p = 0.013$), immobility ($\rho_{spearman} = 0.391$, $p = 0.021$), distress ($\rho_{spearman} = 0.332$, $p = 0.036$), and disgust ($\rho_{spearman} = 0.385$, $p = 0.014$). Additionally, a significant correlation was found between the number of intrusions experienced during the two days following the experimental session and immediate reactions to PT2 in terms of immobility ($\rho_{spearman} = 0.350$, $p = 0.024$) and HR ($\rho_{spearman} = 0.378$, $p = 0.008$). Finally, we

found a significant correlation between the total number of intrusions and subjective response to PT2 in terms of valence ($\rho_{spearman}$ = -0.355, p = 0.025), immobility ($\rho_{spearman}$ = 0.381, p =0.015), and involvement ($\rho_{spearman}$ = 0.350, p = 0.027). Notably, the correlation between valence and the total number of intrusions is negative, as lower valence scores indicate a more unpleasant video-viewing experience.

- Relational Theme: A significant correlation was found between the number of intrusions specifically related to RT1 and immediate reactions to RT1 in terms of distress ($\rho_{spearman}$ = 0.340, p = 0.023), disgust ($\rho_{spearman}$ = 0.377, p = 0.032), and HR ($\rho_{spearman}$ = 0.393, p = 0.002). The number of intrusions experienced during the two days after the experimental session correlates with the immediate response to RT1 for arousal ($\rho_{spearman}$ = 0.344, p = 0.032), immobility ($\rho_{spearman}$ = 0.331, p = 0.040), disgust ($\rho_{spearman}$ = 0.399, p = 0.012), involvement ($\rho_{spearman}$ = 0.372, p = 0.020) and HR ($\rho_{spearman}$ = 0.321, p = 0.032). The total number of intrusions correlates with nearly every immediate response variables to RT1 viewing (among them: arousal: $\rho_{spearman}$ = 0.397, p = 0.011; immobility: $\rho_{spearman}$ = 0.343, p = 0.030; distress: $\rho_{spearman}$ = 0.345, p = 0.029; involvement: $\rho_{spearman}$ = 0.355, p = 0.024), with the exceptions of valence and embarrassment. The strongest correlations were observed with disgust ($\rho_{spearman}$ = 0.446, p = 0.004) and HR ($\rho_{spearman}$ = 0.401, p = 0.006).

  Regarding RT2, the immediate response in terms of immobility is positively correlated with all the intrusions indices (number of intrusions associated with RT2: $\rho_{spearman}$ = 0.407, p = 0.009; number of intrusions during the first two days: $\rho_{spearman}$ = 0.359, p = 0.025; total number of intrusions: $\rho_{spearman}$ = 0.405, p = 0.009). Fnally, involvement and HR correlated with the non-RT2-specific intrusion indices (involvement - number of intrusions during the first two days: $\rho_{spearman}$ = 0.363, p = 0.023; involvement - total number of intrusions: $\rho_{spearman}$ = 0.419, p = 0.007; HR - number of intrusion in the first two days: $\rho_{spearman}$ = 0.375, p = 0.037; HR - total number of intrusion: $\rho_{spearman}$ = 0.381, p = 0.012).
- Traffic Theme: As anticipated, both TT1 and TT2 exhibited only weak or inconsistent associations with the immediate responses. Only the number of intrusions specifically related to TT2 showed modest correlations with TT2 rates

of valence ($\rho_{spearman}$ = -0.326, p = 0.040) and immobility ($\rho_{spearman}$ = 0.350, p = 0.027), which did not extend to the total intrusion counts.

Theme		Intrusions	Valence (rho)	Arousal (rho)	Immob (rho)	Distress (rho)	Disgust (rho)	Embarrass (rho)	Involv (rho)	HR (rho)
Physical	PT1	**Specific**	0.016	0.216	0.159	0.212	0.162	0.084	0.215	0.090
		2 days	-0.275	0.205	0.232	0.238	0.215	0.109	0.360*	0.204
		Tot	-0.253	0.273	0.187	0.233	0.170	0.102	0.397*	0.361*
	PT2	**Specific**	-0.255	0.391*	0.391*	0.332*	0.385*	-0.092	0.067	0.014
		2 days	-0.280	0.233	0.360*	0.141	0.113	0.172	0.309	0.378*
		Tot	-0.355*	0.255	0.381*	0.159	0.106	0.142	0.350*	0.267
Relational	RT1	**Specific**	-0.125	0.062	0.214	0.340*	0.377*	0.170	0.304	0.393*
		2 days	-0.157	0.344*	0.331*	0.302	0.399*	-0.034	0.372*	0.321*
		Tot	-0.210	0.397*	0.343*	0.345*	0.446*	-0.060	0.355*	0.401*
	RT2	**Specific**	0.023	0.178	0.407*	0.287	0.017	0.260	0.245	0.199
		2 days	-0.250	0.250	0.359*	0.248	0.172	0.009	0.363*	0.375*
		Tot	-0.256	0.241	0.405*	0.221	0.181	0.059	0.419*	0.381*
Traffic	TT1	**Specific**	-0.108	0.125	0.047	0.159	0.033	0.032	0.168	0.145
		2 days	-0.009	-0.078	-0.118	0.039	-0.143	0.193	0.144	0.127
		Tot	-0.104	0.106	0.035	0.139	-0.089	0.212	0.281	0.201
	TT2	**Specific**	-0.326*	0.197	0.350*	0.187	0.108	0.067	0.086	0.178
		2 days	-0.142	-0.055	0.042	-0.002	0.009	-0.087	0.165	0.154
		Tot	-0.223	-0.026	0.138	-0.008	0.025	-0.059	0.211	0.196

***Table 16**. Correlation between immediate psychological reactions, Heart Rate (HR) and intrusions using the Spearman's non-parametric rho test.*
PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); Specific = intrusions specifically related to that video; 2 days = Total number of intrusions experienced in the first 2 days following the experimental session; TOT = Total number of intrusions experienced after the experimental session; Immob = Immobility; Embarrass = Embarrassment; Involv = Involvement; HR = Heart Rate.
** $p < 0.05$.*

5.2.5.1 Post-Traumatic Symptoms - IES-R

Finally, we considered IES-R scale to evaluate the traumatic impact of the videos during the week following the experimental session, considering post-traumatic symptoms beyond intrusions. IES-R is a 22-item questionnaire that assesses subjective distress caused by traumatic events. It yields a total score ranging from 0 to 88, with higher scores indicating greater post-traumatic stress symptoms (clinical cut-off: > 33). The scale includes three subscales:

1. Avoidance symptoms scale: measure efforts to evade reminders of the event;
2. Intrusiveness symptoms scale: measure unwanted mental content or dreams related to the event;

3. Hyperarousal symptoms scale: measure the presence of a state of heightened alertness, irritability, and difficulty concentrating.

We used the adapted version of the scale developed by Arnaudova and Hagenaars, which evaluates a period of a week after the vision of the videos.

Not surprisingly, the average scores obtained from this questionnaire were quite low, both for the overall mean score (2.4 ± 1.7) and for the subscales (Avoidance: 0.8 ± 0.6; Intrusiveness: 0.9 ± 0.6; Hyperarousal: 0.7 ± 0.7). None of the subscale showed notably higher involvement compared to the others.

We then explored the relationship between this scale and the immediate psychological and physiological response to video viewing using Spearman's non-parametric rho test. Analysis was stratified again by sex, but no statistical differences emerged. Notably, no significant correlations have been found between IES-R scores and HR response to the videos. The detailed correlation results are reported in Table 17.

Theme			Valence (rho)	Arousal (rho)	Immob (rho)	Distress (rho)	Disgust (rho)	Embarrass (rho)	Involv (rho)	HR (rho)
Physical	PT1	**Avoidance**	-0.299	0.357*	0.315	0.440*	0.339*	0.264	0.307	0.287
		Intrusiveness	-0.158	0.224	0.204	0.231	0.241	0.003	0.093	0.176
		Hyperarousal	-0.281	0.271	0.267	0.309	0.154	0.217	0.385*	0.334
		IES-R	-0.290	0.308	0.309	0.379*	0.261	0.210	0.267	0.341
	PT2	**Avoidance**	-0.167	0.208	0.365*	0.310	0.126	0.182	0.258	0.225
		Intrusiveness	-0.200	0.111	0.082	0.162	-0.059	0.099	-0.021	0.098
		Hyperarousal	-0.194	0.206	0.325	0.227	0.053	0.350*	0.263	0.245
		IES-R	-0.192	0.179	0.312	0.261	0.068	0.252	0.149	0.231
Relational	RT1	**Avoidance**	-0.227	0.373*	0.294	0.367*	0.316	0.173	0.395*	0.286
		Intrusiveness	-0.194	0.166	0.209	0.273	0.168	0.222	0.247	0.299
		Hyperarousal	-0.284	0.256	0.308	0.401*	0.251	0.285	0.353*	0.302
		IES-R	-0.261	0.292	0.299	0.384*	0.270	0.251	0.366*	0.167
	RT2	**Avoidance**	-0.002	0.355*	0.219	0.259	0.154	0.011	0.338*	0.299
		Intrusiveness	-0.108	0.250	0.134	0.135	0.146	0.007	0.199	0.204
		Hyperarousal	-0.009	0.249	0.248	0.213	0.241	0.053	0.337*	0.317
		IES-R	-0.045	0.300	0.242	0.238	0.161	0.051	0.320	0.287
Traffic	TT1	**Avoidance**	0.000	0.184	0.064	0.179	-0.046	0.264	0.230	0.187
		Intrusiveness	0.113	0.078	0.038	0.018	-0.089	-0.022	0.140	0.098
		Hyperarousal	0.019	0.205	0.104	0.118	0.012	0.325	0.270	0.126
		IES-R	0.045	0.174	0.077	0.118	-0.041	0.218	0.238	0.089
	TT2	**Avoidance**	-0.135	0.285	0.163	0.418*	0.026	-0.009	0.391*	0.112
		Intrusiveness	0.175	0.025	-0.083	0.034	-0.137	-0.139	0.165	0.159
		Hyperarousal	-0.087	0.125	0.127	0.150	0.077	0.011	0.332*	0.302
		IES-R	-0.009	0.175	0.104	0.228	0.042	-0.026	0.301	0.297

Table 17*. Correlation between immediate psychological reactions, Hart Rate (HR) and Impact of Event Scale – Revised (IES-R) total score and subscales (avoidance, intrusiveness and hyperarousal) using the Spearman's non-parametric rho test.*
PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); IES-R: Impact of Event Scale – Revised; Immob = Immobility; Embarrass = Embarrassment; Involv = Involvement; HR = Heart Rate.
** $p < 0.05$.*

- Physical Theme: For PT1, the avoidance subscale showed positive correlations with PT1 scores in terms of arousal ($\rho_{spearman} = 0.357$, $p = 0.030$), distress ($\rho_{spearman} = 0.440$, $p = 0.006$), and disgust ($\rho_{spearman} = 0.339$, $p = 0.040$). The hyperarousal subscale correlated positively with PT1 involvement score ($\rho_{spearman} = 0.385$, $p = 0.019$), while the total IES-R score correlated with distress score ($\rho_{spearman} = 0.379$, $p = 0.021$). Avoidance scale score correlated positively with PT2 immobility score ($\rho_{spearman} = 0.365$, $p = 0.026$), and hyperarousal correlated with PT2 embarrassment ($\rho_{spearman} = 0.350$, $p = 0.034$).
- Relational Theme: The avoidance score correlated positively with RT1 immediate response in terms of arousal ($\rho_{spearman} = 0.373$, $p = 0.023$), distress ($\rho_{spearman} = 0.367$, $p = 0.026$), and involvement ($\rho_{spearman} = 0.395$, $p = 0.016$). The hyperarousal subscale and the IES-R score showed significant positive correlations with RT1 distress (hyperarousal: $\rho_{spearman} = 0.401$, $p = 0.014$; IES-R: $\rho_{spearman} = 0.384$, $p = 0.019$) and involvement (hyperarousal: $\rho_{spearman} = 0.353$, $p = 0.032$; IES-R: $\rho_{spearman} = 0.366$, $p = 0.026$).

  For RT2, avoidance correlated positively with his score of arousal ($\rho_{spearman} = 0.355$, $p = 0.031$) and involvement ($\rho_{spearman} = 0.338$, $p = 0.041$), which is also correlated with hyperarousal score ($\rho_{spearman} = 0.337$, $p = 0.041$).
- Traffic Theme: Once again, traffic videos presented the fewest correlations, with TT1 presenting no significant association with any of the IES-R scores. In contrast, avoidance subscale correlated positively with distress ($\rho_{spearman} = 0.418$, $p = 0.010$) and involvement ($\rho_{spearman} = 0.391$, $p = 0.017$) in response to TT2, while hyperarousal correlated positively with TT2 involvement ($\rho_{spearman} = 0.332$, $p = 0.045$).

Overall, the pattern of correlations indicates that avoidance and hyperarousal were the subscales most consistently associated with immediate emotional reactions, whereas intrusiveness did not show any significant correlations. These subscales seem to be particularly associated with physical and relational themes, whereas the traffic videos elicited only weak or inconsistent relationships.

5.3. Discussion

The main aim of the present study was to evaluate the efficacy of a set of videos in replicating a stress-like experience within the context of the TFP. Our objectives were, on one hand, to replicate the findings obtained with a previously validated set of videos by Arnaudova and Hagenaars (2017), and, on the other hand, to expand the video set by adding three new stressful clips and including a subjective measure of the impact of video exposure on sleep. To this end, we replicated an experimental protocol in line with the paradigm used by Arnaudova and Hagenaars, which includes both psychological and physiological immediate responses to the film clips (assessed through PPG and EDA), as well as delayed measures of trauma-like symptoms (i.e., intrusions and PTSD symptoms assessed using the IES-R questionnaire, and subjective sleep measures obtained from the Sleep Diary).

5.3.1 Immediate Response

5.3.1.1 Subjective Psychological Response

In line with previous findings from Arnaudova and Hagenaars (2017), our results confirm that the negative traumatic videos included in the paradigm were capable of eliciting strong stress-like reactions involving a range of affective responses, when compared with both negative and positive videos. Across all the three themes, participants consistently rated negative clips as more unpleasant (negative valence), more activating (perceived arousal), and more distressing and reported higher levels of immobility (the last two both typically associated with traumatic experiences) (Volchan et al., 2011). They also reported greater levels of disgust, embarrassment and involvement.

Interestingly, the negative Traffic videos showed the weakest differentiation from their corresponding neutral and positive counterparts, being rated as less distinct in terms of emotional impact, particularly for disgust and embarrassment. This finding highlights the important role of these two affective components in shaping the stressful impact of the stimuli. Recent research has emphasised that emotions such as disgust and shame (embarrassment can be considered a state response that can negatively develop into shame) play a pivotal role in determining the psychological impact and consequences of stressful and traumatic experiences. Traditionally, trauma models have centered on fear

conditioning; however, evidence demonstrate that these moral emotions contribute to trauma-related distress (Lee & Turner, 2001). For instance, it has been observed that shame measured one month after a stressful event was the only emotion that predict PTSD symptoms entity six months later, even when controlling for fear levels measured immediate after the trauma (Andrews et al., 2000). In other study authors found that shame and guilt, another moral emotion, increase with the number and severity of violent experiences and are associated with more severe negative psychological consequences, specifically in the female sample (Aakvaag et al., 2016). Disgust, too, has been shown to be particularly salient among survivors of sexual assault, who frequently report sensations of mental contamination (defined as a subjective feeling of inner dirtiness not linked to physical contact), with prevalence rate around 60% (Fairbrother & Rachman, 2004; Badour & Feldner, 2012). Moreover, as with shame, disgust predicts both the severity and persistence of PTSD symptoms and is largely independently from fear responses (Badour et al., 2013; Coyle et al., 2014). Thus, the relatively weak responses to Traffic videos may be explained by their reduced activation of these moral emotions. Unlike interpersonal or socially charged scenarios, traffic accidents may evoke fear and shock but lack the interpersonal or moral dimension that triggers first embarrassment (that later will become shame) and disgust.

Consistent with Arnaudova and Hagenaars (2017), no significant differences were observed across videos in terms of attention. We interpreted this as methodological strength, suggesting that all clips successfully maintained participants' attention throughout the session. The significant difference that we found in involvement, however, are particularly meaningful. Although attention and involvement may appear similar at a surface level, they represent distinct constructs. They both describe processes of engagement with the stimulus, but attention is a primarily cognitive process that can be goal-directed or stimulus-driven (Corbetta & Shulman, 2002) reflecting participants' efforts to stay focused as instructed but also the ability of the videos to capture attention. Involvement, on the other hand, is a motivational–affective state that cannot be voluntarily directed but must be induced by the characteristics of the stimulus itself (Celsi & Olson, 1988). Therefore, the finding that negative videos elicited higher involvement than both positive and neutral clips indicate their strong emotional impact. Supporting this, Herzog and McNelly (2024) found, in a paradigm that explore TFP stimuli

characteristics, the level of involvement to 8 different clips (measured with ITC-Sense of Presence Inventory that assess individuals' subjective experience of presence and engagement when interacting with media environment) (Lessiter et al., 2001) that the clip that were rated as more realistic in the involvement scale were also perceived as more threatening, as measured by the Perceived Threat Questionnaire (specifically designed for the study) (Herzog & McNelly, 2024).

Similarly, the lack of effects for previous exposure or gaze aversion reinforces the robustness of the paradigm, familiarity with the clips did not appear to attenuate their affective impact, suggesting that the stress induction relies primarily on the emotional content rather than on novelty or surprise.

Unlike Arnaudova and Hagenaars (2017), we found limited gender differences, with the exception of a stronger freezing response (immobility score), in the female subgroup, to the relational video selected by us (RT2). This partially diverges from previous evidence, as women are generally about twice as likely to develop PTSD following trauma (Gill et al., 2008; Silove et al., 2017), and also tend to exhibit stronger and more negative reactions to emotional stimuli; indeed a lot of articles shows how woman tend to present stronger and more negative responses to IAPS images (Arnone et al., 2011; Bradley et al., 2001; Lithari et al., 2010). The fact that we didn't replicate this result may be due to the relatively small sample sizes for male and female subgroups; thus, significant differences may have emerged only for the most gender-salient stimulus (the video representing a group rape).

Another important difference from Arnaudova and Hagenaars (2017) that we failed to replicate is that, except for involvement and arousal, the positive stimuli were not significantly different from the neutral ones. This is not surprising, as previous research shows that it is harder to experimentally induce positive emotions than negative emotions. In a study from Mar and colleagues, the authors compare five different experimental designs to manipulate positive and negative affect and consistently observed that all the paradigms were more capable to induce negative emotions than positive ones, both from a psychological and a physiological standpoint (Mar et al., 2018). Moreover, in a meta-analysis by Fernández-Aguilar and colleagues specifically investigating studies that employed video paradigms to manipulate emotions, it was found that clips more easily induced negative emotion than positive emotion, and that negative emotions were also

more intense and longer-lasting than positive ones (Fernández-Aguilar et al., 2019). This pattern reflects an evolutionary bias toward threat detection: negative content commonly constitutes a threat, therefore is perceived as more salient compared to positive content, and tends to elicit more automatic responses (Mar et al., 2018).

Accordingly, our failure to replicate Arnaudova and Hagenaars results on positive video could, once again, be due to the smaller sample size, which may have been insufficient to capture these subtler differences, as well as to interpersonal differences; what is considered negative is more universal and less influenced by cultural or age differences (Farb et al., 2013). For the purpose of this experiment, the fact that we did not replicate these results is not highly consequential, as we were mainly interested in ensuring the effects of the negative videos in order to select the best stimulus to induce stress. However, in a replication framework, this limitation should be taken into account, particularly when aiming to employ the video set to induce both negative and positive affective states.

Considering the comparison between all the negative videos of different themes, differences emerged across all the variables representing subjective immediate response, with the exception of attention and involvement. Overall, both relational and the first physical videos were consistently rated as more unpleasant, arousing and induced higher immobility, disgust and embarrassment compared to PT2 and traffic accidents. This supports the notion of reduced affective salience for the traffic category. Possible explanations include the fact that car accident are considered a more common experience, which in some cases may not have dramatic consequences; the fact that, as discussed above, traffic accident tend to involve moral emotions to a lesser extent which are pivotal in determining the traumatic effect of an event; or the nature of the materials, since for all the other negative videos we selected clips from movies, whereas for the traffic category we used an advertisement and a documentary, and with this type of video it may be harder to empathize. However, this last hypothesis is not fully consistent with the evidence that all the negative videos were comparable in terms of involvement, with the only exception of TT2, which, paradoxically, was rated as more engaging than PT2.

RT2 were the only video that elicited significantly higher levels of distress than all the others, and this score could be influenced, or even determined, by the fact that this video

was able to induce other, more specific affects. In general, the reason why both relational videos resulted as more effective compared to the others could once again be due to the role of moral emotions (disgust and embarrassment) in determining traumatic effects, as these emotions are usually related to social violations (Tangney et al., 2007).

The immediate response alone it's not sufficient to determine the most affective stimulus in the context of the TFP related to the purpose of this work, but the variability observed so far highlights that not all traumatic contents evoke the same emotional pattern or intensity and this is something to take into account when you are building you experimental paradigm depending on specific research objectives.

5.3.1.2 Objective Physiological Response

Consistent with the subjective pattern described above, the physiological data revealed significant modulation of autonomic activity in response to the negative stimuli compared to positive and negative one, confirming the presence of an effect beyond the participants' subjective experience. HR and EDA indices provided converging evidence of the heightened emotional and physiological engagement elicited by the traumatic videos, but with partially divergent temporal and functional profiles.

In line with previous findings by Arnaudova and Hagenaars (2017), all negative clips, across all themes, elicited a significant mean HR deceleration relative to their neutral and positive counterparts. Also here, this pattern was particularly marked for the relational theme, followed by the physical and, to a lesser extent, the traffic theme. HR deceleration is a well-established index of freezing-like defensive responses in response to negative stimuli. For instance, in a study that used negative pictures of mutilation (from IAPS dataset) compared to neutral or positive images, they observed a significant decrease in HR in response to negative images compared to positive and neutral pictures (Azevedo et al., 2005). Hagenaars and colleagues in 2014 showed an analogue finding in an experiment with neutral, negative and positive videos: only negative videos elicited a significant HR deceleration compared to the neutral and positive ones (Hagenaars et al., 2014). This reaction is well known as *freezing* and it's defined as a defensive state characterised by reduced movement, heightened attentional focus and HR deceleration (called "*fear bradycardia"*) in response to a perceived threatening situation. It has been observed in both animal and human research and its considered an adaptive response: this

state reduces background noise and enhances perception, and this facilitates threat identification; additionally it reduces the subject's detectability (as the animal becomes less visible or audible, the human becomes less attention-grabbing) and helps preserving resources, thus preparing the organism for potential action (Roelofs, K., 2017).

As for the immediate response, unlike Arnaudova and Hagenaars, no significant differences emerged between positive and neutral clips, again suggesting that positive affect is more difficult to induce physiologically in laboratory settings, especially considering that negative stimuli tend to induce an automatic evolutionarily conserved defensive responses, whereas positive affect often relies on higher-order, context-dependent processes and is typically less intense and shorter-lived (Lang et al., 1993).

In contrast to Arnaudova and Hagenaars (2017), in the present study we complemented the cardiac index with a second physiological measure, EDA, to capture sympathetic arousal more directly. Including this additional measure was deemed relevant, as EDA reflects changes in sweat gland activity tightly linked to sympathetic nervous system output, thereby providing information on emotional activation that is not necessarily paralleled by HR dynamics. While HR is primarily influenced by the interplay between sympathetic and parasympathetic branches and can therefore index defensive parasympathetic dominance, EDA represents a purer marker of sympathetic excitation (Cacioppo et al., 2016; Dawson et al., 2007). More specifically, EDA predominantly reflects alpha-adrenergic sympathetic activity related to sudomotor responses, whereas cardiac activity is also modulated by beta-adrenergic sympathetic influences acting on the heart. As these autonomic components can be partially dissociated, concurrent HR deceleration and increased EDA may reflect complementary but mechanistically distinct processes within the defensive response (Dawson et al., 2007).

All negative videos produced higher EDA phasic responses (CDA.AmpSum, CDA.SCR, and CDA.ISCR indices) compared to neutral and positive ones. This suggest that the negative stimuli induced greater sympathetic activation.

However, while HR showed a decrease in response to negative content (a pattern potentially reflecting increased parasympathetic influence or reduced sympathetic activity), EDA showed an increase (a sympathetic marker). At first glance, these patterns might appear contradictory, yet they represent two complementary facets of the same

defence cascade. HR deceleration has been associated with freezing or attentive states, which are often, but not exclusively, linked to parasympathetic dominance, whereas increased EDA reflects simultaneous sympathetic arousal, signalling emotional intensity and readiness for action.

An alternative (and not mutually exclusive) explanation is that EDA captures a more tonic, underlying arousal state experienced by the participants throughout the entire video session, whereas HR reflects a phasic, immediate response to threat cues, that could represent the tentative of the subject to autoregulate himself during negative videos. This would account for their partial dissociation: the HR responds rapidly and transiently to sudden aversive cues, while EDA integrates slower, cumulative sympathetic changes over time (Cacioppo et al., 2016). However, this interpretation should be taken cautiously, as no significant effects emerged for the tonic EDA component (CDA.Tonic), suggesting that the differences primarily concern phasic reactivity rather than sustained baseline activation.

Moreover, methodological aspects may have contributed to this divergence: EDA data were analysed on a fixed 60-second window centred around the emotional peak, unlike HR, which was computed over the full duration of each clip as it's not influenced by stimulus duration. Given that EDA is a highly non-linear, context-sensitive signal (Benedek & Kaernbach, 2010a), its interpretation can be more challenging, particularly when comparing stimuli of different lengths and dynamic structures. Consequently, although the direction of results supports the validity of the paradigm, EDA effects should be interpreted with caution and viewed as reflecting general sympathetic activation rather than precise temporal dynamics. In fact, these EDA indices did not show any significant correlations with the other immediate, delayed, psychological, or physiological responses, as illustrated in the results section.

Also in this case, no differenced emerge when comparing positive and neutral videos' responses.

When comparing all negative videos across themes, both HR and EDA analyses consistently pointed to the relational videos as the most effective stimuli, followed by the physical and, finally, the traffic videos. So, the same hierarchical pattern observed in the psychological immediate response. The only slight difference is that in the case of

physiological indices, RT1 emerged as the most physiologically engaging clip, producing both the significant largest HR deceleration and the highest EDA phasic indices.

In summary, the objective physiological data corroborate the subjective findings, underscoring the robustness of the negative videos included in the paradigm in eliciting stress-related responses in the immediate.

5.3.2 Delayed Response: Impact on Sleep

Compared to Arnaudova and Hagenaars (2017) we were interested in investigating the effects of stressful videos on sleep. As underlined in the introduction, the subjective experience of sleep plays a central role when we consider ID. Indeed, to meet the diagnostic criteria for ID none objective examination is required, and in some cases, sleep disturbance is perceived by the patient even in the presence of an adequate amount of objectively measured sleep (Castelnovo et al., 2019). This underlines the central role of subjective perception in determining the presence of sleep disturbances.

To better characterize the temporal dynamics of the potential sleep disturbance, two distinct time windows were analysed: the entire week before versus the entire week after the experimental session, and a narrower period encompassing the two nights immediately preceding and following video exposure. From this distinction in examining the subjective sleep data, a clear temporal gradient in the impact of the experimental session emerged. While some significant differences were observed when comparing the entire pre- and post-week, the most pronounced and consistent effects appeared in the acute phase analysis.

Specifically, the comparison between the entire pre- and post-week revealed few changes that indicates a worsening in perceived sleep quality: a significant decrease in sSE% and an increase in WASO, accompanied by non-significant trends toward longer SL and lower cSE%) in the week after the experimental session. This suggests a mild alteration in perceived sleep continuity following the experimental session.

However, when narrowing the observation window, the results became markedly more robust. Participants reported a significant deterioration in nearly all key indices of subjective sleep quality: increased SL, decreased both subjective and calculated SE%,

and increased WASO. Interestingly, while no significant differences emerged in the number of awakenings, the robust increase in WASO indicates that participants did not experience a more fragmented sleep, but rather that their awakenings tended to last longer. One possible interpretation is that, once awake, participants may have been disturbed by intrusive or emotionally charged thoughts related to the negative video content, which in turn may have hindered their ability to fall back asleep. This interpretation would be consistent with evidence linking nocturnal awakenings and difficulty returning to sleep with heightened cognitive and emotional arousal that can be caused by negative mental content. For instance, in a recent study Kalmbach and colleagues (2019) investigated nocturnal cognitive arousal in a laboratory setting using both self-reported, PSAS questionnaire, and objective measures, including HR variability and ACT indices (Kalmbach et al., 2019). They found that participants who experienced higher levels of pre-sleep rumination and intrusive thoughts showed poorer sleep continuity, including longer periods of WASO, despite not having more awakenings overall. Similarly, another study compared sleep-related physiological arousal with general cognitive arousal, measured with PSAS scale, and demonstrated that cognitive arousal was more strongly associated with reduced subjective sleep quality than physiological indices, suggesting that mental activity during or after awakenings plays a central role in maintaining sleep disturbances (Spiegelhalder et al., 2012). Moreover, Kalmbach and colleagues (2018) in another study found a positive correlation between WASO (and SL) and cognitive intrusions, measured with Intrusion scale of IES-R (Kalmbach et al., 2018b).

Notably, the acute phase was also the only time window in which participants' qualitative evaluations of their sleep, in terms of perceived deepness and restorativeness, showed significant declines. This indicates that the negative impact of the emotional manipulation extended beyond quantitative estimates of sleep continuity, affecting participants' overall satisfaction with their sleep experience.

Furthermore, the acute phase analysis revealed a significant increase in the number of dreams reported after the video session. This finding aligns with the extensive literature suggesting that dreaming plays a key role in emotional regulation and memory processing. As discussed in the introduction, according to models such as the Walker's Emotional Regulation Hypothesis (Walker & van der Helm, 2009; van der Helm & Walker, 2011), REM sleep and dream activity facilitate the reprocessing of affective

experiences (the theory talks about "*overnight therapy"*), promoting the integration of emotional memories while reducing their psycho-physiological arousal. In this way, we store the information while releasing the associated emotional charge. Evidence supporting this theory shows how following exposure to emotionally charged material, dream frequency and vividness often increase, reflecting the brain's attempt to integrate and symbolically elaborate the stressful experience (Levin & Nielsen, 2007; van der Helm & Walker, 2012). For instance, an fMRI study investigating the neural correlates of emotional processing after sleep by exposing participants to emotionally negative and neutral images found that, after sleep, particularly REM-rich periods, compared to after wakefulness, amygdala reactivity to negative stimuli was significantly reduced, while functional connectivity between the amygdala and the medial prefrontal cortex increased, suggesting enhanced top-down regulation of emotional responses (van der Helm et al., 2011). Similarly, Gujar and colleagues (2010) examined the role of REM sleep in emotional reactivity by comparing participants who had a full night of sleep with those who were sleep-deprived. They observed that sleep deprivation led to a 60% amplification of amygdala responses to negative stimuli and a disruption of its functional coupling with the prefrontal cortex, indicating a breakdown in emotional control mechanisms normally restored by REM sleep (Gujar et al., 2010). Converging evidence from dream research also supports the idea that dreaming contributes to the symbolic and emotional reprocessing of waking experiences (Scarpelli et al., 2019). For instance, Cartwright in 2010, studied individuals recovering from stressful life events, and found that dreams often incorporate recent emotional themes in a way that predicts better next-day mood and emotional adaptation (Cartwright et al., 2010). Therefore, the observed increased in number of dreams could reflect this mechanism, a functional marker of ongoing emotional processing triggered by the traumatic videos. Unfortunately, we did not ask the subjects to record the content of their dreams, but it would have been interesting to investigate whether and how dream content related to the negative video scenarios.

To conclude, this temporal pattern is particularly meaningful when interpreted in light of the characteristics of the TFP. As previously discussed, the paradigm was designed to experimentally mimic the exposure to a real traumatic event, eliciting acute distress reactions similar those observed following real-life trauma, yet with milder intensity and shorter duration (Holmes & Bourne, 2008; James et al., 2016). Consistent with this, our

data show that the disturbance in sleep was most pronounced in the days immediately following the exposure but tended to attenuate over the following week. In other words, the short-lived alteration in sleep reflects the expected temporal course of the induced stress response, confirming the ecological validity of the paradigm while reinforcing its ethical appropriateness.

The findings suggest that exposure to these negative videos can temporarily disrupt perceived sleep quality and continuity, particularly during the nights immediately following the vision, while also triggering increased dream activity that may reflect adaptive emotional processing, which in turn could account for the rapid resolution of the videos' traumatic impact observed here, as well as in the context of the intrusive phenomena that will be explored in the next chapter.

5.3.3 Delayed Response: Intrusion and others Trauma-like Symptoms

Following the analysis of sleep-related outcomes, we examined the delayed psychological impact of the emotional videos through the measures of post-traumatic symptoms, using the Intrusion Diary to quantify and investigate intrusions and the IES-R questionnaire to evaluate PTSD-like symptoms in general. These measures were intended to capture the persistence and phenomenology of trauma-like reactions in the days following exposure, and to evaluate if the temporal course of these reactions paralleled the short-lived effects observed in sleep measures.

As in Arnaudova and Hagenaars (2017), participants showed relatively low compliance with the intrusion diary procedure. Nevertheless, a considerable number of intrusions were reported during the week following the experimental session, particularly in the first two days after exposure. Consistent with the sleep findings, the frequency of intrusions declined markedly over time, (the number of intrusions in the first two days following the session was significantly higher compared to the one of the last two days) indicating a rapid attenuation of trauma-related cognitive activity, similar to the sleep one. This pattern parallels prior TFP studies reporting that intrusion frequency and distress typically peak within 24–48 hours and decline sharply thereafter (James et al., 2016; Holmes & Bourne, 2008).

Interestingly, the videos that elicited the highest number of intrusions were those depicting relational and the first physical video (RT1, RT2, PT1), whereas traffic-related videos and the second physical video were associated with substantially fewer intrusive contents. This distribution is consistent with the pattern observed in the immediate emotional and physiological responses, where relational and physical videos elicited the stronger reactions. Such convergence across measures strengthens the interpretation that emotional proximity and social content play a crucial role in determining the persistence of intrusive re-experiencing (James et al., 2016; Varma et al., 2024).

The qualitative ratings of the intrusions provide further insight into their phenomenological features. On average, participants rated their intrusions as moderately to highly vivid and moderately distressing, while reporting a relatively high sense of control. The spontaneity ratings were medium-low, indicating that many intrusions were cue-dependent rather than fully spontaneous. This suggests that the reappearance of the video-related images was often triggered by environmental or internal reminders rather than arising involuntarily. The moderate vividness and distress ratings, combined with a relatively high perceived sense of control and the predominance of cue-dependent rather than fully spontaneous intrusions, indicate a reduced magnitude of trauma-like impact from the videos, once again in line with the literature (Holmes & Bourne, 2008; James et al., 2016).

Correlation analyses further clarified the relationship between the immediate response to the videos and the subsequent emergence of intrusions. Stronger emotional and physiological reactions during the physical and relational themes were associated with a greater number of intrusions, particularly in the first two days after exposure, whereas traffic videos elicited only weak or inconsistent associations. Within the physical theme, intrusion frequency correlated positively with distress, disgust, immobility, HR, and involvement, while more unpleasant valence ratings predicted higher intrusion occurrence. A comparable pattern emerged also for the relational videos, where high distress, arousal, disgust, and involvement were linked to increased intrusion frequency.

This result together underscores the role of negative emotional valence and physiological activation in intrusive memory formation. This interpretation is further supported by evidence showing that stronger physiological and emotional activation during or immediately after exposure to a stressful stimulus predicts a higher frequency

of subsequent intrusive memories. For instance, Chou and colleagues (2013) investigated the relationship between short term physiological responses and the development of intrusive memories using a TFP: higher HR acceleration and enhanced startle reactivity during the film were significantly associated with more frequent and distressing intrusions in the subsequent week (Chou et al., 2013). Similarly in prospective study to examine how peritraumatic emotions and cognitive appraisals predict the emergence of intrusive memories after an analogue trauma. Participants completed continuous self-assessments of emotional intensity, including measures of fear, sadness, and helplessness, while watching a highly negative video. Stronger peritraumatic negative emotions (particularly fear and helplessness) significantly predicted both the frequency and distress of intrusive memories, independently of baseline anxiety or general negative affect (Ripley et al., 2017). These results indicate that the combination of heightened emotional engagement and physiological arousal during exposure constitutes a critical predictor of subsequent intrusions, reflecting the strength of memory encoding for emotionally salient material.

These results suggest that socially salient and emotionally engaging material is more likely to be consolidated into intrusive memories, consistent with evidence that interpersonal and moral emotions enhance post-event cognitive engagement as we have already discussed (Andrews et al., 2000; Lee & Turner, 2001).

These results suggest that the intrusive re-experiencing observed here was mild, transient, and modulated by the emotional salience of the stimuli, with a clear decline across days. This rapid attenuation may also be linked to the increase in dream frequency observed in the sleep analysis, reflecting an adaptive emotional processing mechanism.

Considering other PTSD-like symptoms, IES-R total mean score and subscales scores were low, far below the clinical cut-off, confirming that the traumatic impact of the videos was mild and transient, consistent, once again, with the ethical and methodological intentions of the TFP (Holmes & Bourne, 2008; James et al., 2016).

The low Intrusiveness subscale score coherently reflects the rapid decline in the number of intrusions observed in the diary, reinforcing the notion that the emotional material was reprocessed quickly and effectively.

Correlation analyses between IES-R subscales and immediate responses revealed only a few significant associations, predominantly within RT1. Avoidance and hyperarousal showed the most consistent relationships with distress, arousal, and involvement during viewing, while the intrusiveness scale was not significantly correlated with any immediate measure. These findings suggest that, although participants experienced transient distress and physiological activation during the videos, these reactions did not translate into persistent intrusive re-experiencing or post-traumatic symptomatology.

5.3.4 Limits

The present study presents some limitations. First, regarding participants, it is important to note that those who chose to take part in this study may possess specific characteristics. In particular, they might be relatively resilient individuals, given that they agreed to participate in a study explicitly designed to induce stress. Furthermore, our sample consisted mainly of highly educated individuals, such as university students or professionals in high-level positions. This aspect may be problematic, as higher education has been associated with greater psychological resilience (Bonanno et al., 2007). Consequently, our findings might overestimate the general population's resilience to distressing stimuli. Moreover, the relatively small sample size may have limited the statistical power to detect subtle effects or interactions, particularly in the correlational analyses involving physiological and psychological variables, as well as in the comparisons between positive and neutral videos.

Another factor that could not be entirely avoided and may have affected the paradigm is the habituation phenomenon. Indeed, although the order of the videos was randomized, repeated exposure to emotionally intense stimuli may have led to partial habituation, potentially reducing the overall magnitude of the observed responses.

As expected, compliance with the Intrusion Diary was relatively low. This limitation may have partially influenced our results, potentially leading to an underestimation of delayed effects in terms of PTSD-like symptomatology.

Finally, the exclusive reliance on subjective indicators of sleep response represents another major limitation. Including physiological measures would not only strengthen our conclusions regarding the impact of the negative video set on sleep but would also allow to examine specific aspects of sleep architecture, such as sleep onset latency, REM

density, or micro-arousals, that cannot be captured through subjective reports alone but that can be affected by the video viewing. Nevertheless, the broader impact of the TFP on both subjective and objective sleep parameters will be a primary focus of the main experiment of this research project.

5.3.5 Conclusion

Overall, the present study confirmed the efficacy of the selected video set in eliciting both immediate and delayed stress-like responses within the framework of the TFP, both the old and the new ones. From the results emerges that all negative clips successfully induced immediate negative subjective emotional reactions and objective autonomic activation that indicated the involvement of ANS, demonstrating their reliability as experimental stressors. Moreover, the delayed effects, reflected in temporary alterations of subjective sleep quality and in the occurrence of intrusive memories, further support the TFP's ability to produce mild, transient trauma-like responses. These effects were short-lived and of moderate intensity, particularly regarding PTSD-like symptomatology, which remained well below clinical thresholds. Therefore, these stimuli can be successfully implied to study sleep response to a stressor in the short-term.

In general, the results obtained with the negative videos originally validated by Arnaudova and Hagenaars (2017) were successfully replicated, confirming the robustness of their stimuli. Furthermore, the efficacy of the additional stimuli introduced in the present study was comparable to their results. However, we did not replicate the gender-related differences and the effects observed for positive videos in the original study. As previously discussed, this discrepancy may stem from cultural differences between samples or from the reduced sample size and the limited number of participants in the gender subgroups, which may have prevented the detection of subtler effects.

Among all the videos, those depicting relational scenarios consistently emerged as the most effective stimuli across psychological, physiological, and delayed measures. Their pronounced impact likely stems from their strong interpersonal and moral-emotional content, which enhances emotional involvement and the consequent distress. In conclusion, RT1 and RT2 seems to be the best candidate for the role of stressor for studying the mediating role of attentional abilities in shaping the sleep response to stressful experiences.

6. Experiment 2: How Attention Shapes Sleep Responses to Stress in Subjects at Risk and Not at Risk of Developing Insomnia

The aim of this project arises from one fundamental question: since stress constitute one of the main precipitating factors for the development of ID, what can we target to enhance sleep system's resilience?

We began by reviewing the literature to better understand how the impact of stress on sleep has been studied so far. The negative influence that stressful events have on sleep has been demonstrated, though with a wide range of different consequences. We concluded that the available literature is insufficient to clarify these effects, and the existing studies are highly heterogeneous in terms of stressors and paradigms, limiting the comparability of results. We analysed the different approaches and concluded that the best compromise between an ecological and, as much realistic as possible, yet ethical and experimental approach is the TFP. Among the articles included in our systematic review, only one applied a true TFP. However, although the authors verified the effects of the stressor on sleep retrospectively, the negative video used by Talamini et al. (2013) had not been previously validated. This likely reflects the fact that, unlike emotional images, there is no universally validated database of videos used by the scientific community for TFP studies. Therefore, to maintain maximal control over our experimental paradigm and ensure that our traumatic video effectively induced stress, we replicated and extended the validation process on a new set of videos for the TFP. All the tested negative videos successfully induced both psychological and physiological distress and short-term trauma-like symptoms. Three out of six videos, mainly falling in the relational category, proved to be the most effective. Based on these results, we selected one of the most effective videos and developed a two-week protocol to evaluate the moderating role of attentional performance on subjective and objective sleep following stressor exposure in a sample of healthy participants with and without ID symptoms, as our main interest lies in the aetiopathogenesis of the disorder.

We decided to focus on a pre-clinical window (i.e. participants with ID symptoms), as our main interest lies not in what happens in the chronic phase of ID, but in how individuals transition from a state of vulnerability to the clinical condition. Therefore, we aimed to investigated, in a detailed manner, the effects of stress on sleep both from the psychological and the physiological perspective in subjects with and without pre-clinical

condition. Our goal was to clarify the inconsistent findings that emerged from the systematic review and to develop an experimental design that, within our practical limitations, could address the methodological shortcomings highlighted in previous studies. Moreover, within the Wells' CAS model, we aimed to examine, first, whether individuals at risk compared to those not at risk display impaired attentional functioning, and second, whether attentional performance itself can predict sleep disturbance outcomes following stressor exposure. Wells hypothesised that CAS is associated with a reduced ability to disengage attention, the orienting network (Wells, 1995; Wells & Matthews, 1996; Wells, 2009). Nevertheless, we sought to consider attention across all its major functional domains in order to identify which components best explain the sleep response to stress, and in which direction.

In this perspective, one specific component of attention, for example, orienting, consistent with Wells' model, may represent a protective factor: the greater the individual's orienting performance, the higher their sleep system's resilience to stress. However, the opposite may also occur: for instance, an excessively active alerting system could foster a state of heightened self-monitoring that interferes with the normal course of sleep.

Understanding these mechanisms could be particularly meaningful from a therapeutic perspective, as it may lead to the development of more targeted clinical protocols that take into account the role of attention. Strengthening specific components within the attentional domain could help restore the natural process of sleep, and might also serve as a preventive strategy against the development of ID, at least in individuals known to be at risk.

6.1. Methods

6.1.1. Participants

Participants were recruited on voluntary basis from the general adult population, with a primary focus on the university community. Recruitment was mainly conducted through online advertisements, posters, and word of mouth. A total number of 98 individuals completed the online screening questionnaires, and a final number of 40 participants

(20/20 M/F; age: 29.2 ± 7.0 years; range: 20.0 – 58.0) met the inclusion criteria and successfully complete the entire protocol.

Recruitment was stopped once the target sample size of 40 participants (20 per group) was reached. This number was determined using G*Power software (Faul et al., 2007) for power analysis. This analysis aimed to test the difference between repeated measurements (pre/post) with a two-tailed rmANOVA, assuming a large effect size (f = 0.40) and an alpha level of 0.05. Moreover, our sample size was aligned with previous experimental paradigms investigating the relationship between stress and ID (Kleim et al., 2016; Chen et al., 2017; Richards et al., 2023).

Participants were matched across groups for gender and age, with a tolerance of ±2 years for age pairing, to ensure demographic comparability between the two groups.

Inclusion criteria required participants to be adults between 18 and 65 years of age, fluent in Italian, and with normal or corrected-to-normal vision and hearing, with and without ID complaints.

The screening questionnaire was administrated using Google Form and included demographic information, inclusion/exclusion criteria (assessed using closed-ended questions), the Sleep Condition Indicator (SCI) (Espie et al., 2014; Palagini et al., 2015), the BDI-II (Beck et al., 1996; Solaro et al., 2016) and the Beck Anxiety Indicator (BAI) (Beck et al., 1988; Sica & Ghisi, 2007).

Participants were excluded if they met any of the following conditions:

- Having a current of diagnosis or undergoing treatment for a depressive or an anxiety disorder;
- Having a current of diagnosis for a sleep disorder made by a sleep medicine specialist;
- Having experienced a traumatic event related the theme of the video (sexual violence), in order to avoid potential bias from participants who might be particularly vulnerable due to their personal experiences;
- Exciding the clinical cut-off for anxiety and/or depression symptoms;
- Suffering from neurological, cognitive, or psychiatric disorders;
- Current substance abused;
- Suffering from severe or degenerative neurological, cognitive, or psychiatric disorders.

6.1.2. Experimental Protocol

The study received formal ethical assessment by the competent Ethics Committee ("Comitato Etico Territoriale Lombardia 1"; Protocol code: SREEG; Ethics Committee registry number: CET 403-2024). All procedures were conducted in accordance with the principles of the Declaration of Helsinki, and all participants provided written informed consent prior to participation.

The main hypothesis of this study was that the attentional system and SR interact in determining the risk of ID. To test this, SR was experimentally manipulated to assess the effects of stress induction on sleep. Specifically, we adopted a within-subjects pre/post design, in which all participants will be exposed to the same stress induction.

Based on the SCI score obtained during the screening phase the participants were divided into two groups (i.e. 20 subjects with ID symptoms [IS group] and 20 good sleepers [GS group]) to allow a between-group comparison of the differential impact of stress on sleep. We applied the clinical cut-off score of 16 and excluded participants scoring between 17 and 21 to avoid including individuals without ID symptoms but with suboptimal sleep quality:

1. GS group: 20 healthy individuals without ID symptoms (SCI > 21);
2. IS group: 20 healthy individuals with ID symptoms (SCI≤16).

Participants who met the inclusion criteria and agreed to participate in the protocol, were first instructed by telephone to complete a baseline questionnaire comprising a set of questionnaire investigating sleep from different perspectives, and how to complete an online Sleep Diary within 10 minutes after awakening, for the entire duration of the study (17 days in total). The first seven days of sleep monitoring allowed us to verify that participants maintained a regular sleep schedule and that the GS group exhibited good sleep quality. After this initial week of Sleep Diary, participants attended the Sleep Laboratory to undergo a neuropsychological evaluation of their attentional functioning, including the Attention Network Test (ANT) (Fan et al., 2002), the Stroop task (Stroop, 1935), and the Go/No-go task (Newman & Kosson, 1986). In the same session, they were trained in the use of the Sleep Profiler (SP), a portable sleep-monitoring device, which they were instructed to wear for two consecutive nights serving as the objective sleep baseline (Levendowski et al., 2017). Following the initial two nights of baseline recordings, participants returned to the laboratory for the TFP session. A subsequent two-

day recording period with the SP then followed, during which participants completed an Intrusion Diary, supported by the Experience Sampling Method (ESM) to facilitate consistent and systematic reporting of intrusive memories (Larson & Csikszentmihalyi, 2014). On the third day following stress exposure, participants returned to the laboratory to end the PSG experimental session. During this session, they had the opportunity to briefly discuss the data collected during the four days of SP recordings. They were then instructed to continue completing both diaries for an additional six days. On day 17 they completed one last questionnaires battery.

The timeline of the experimental protocol is illustrated in Figure 24.

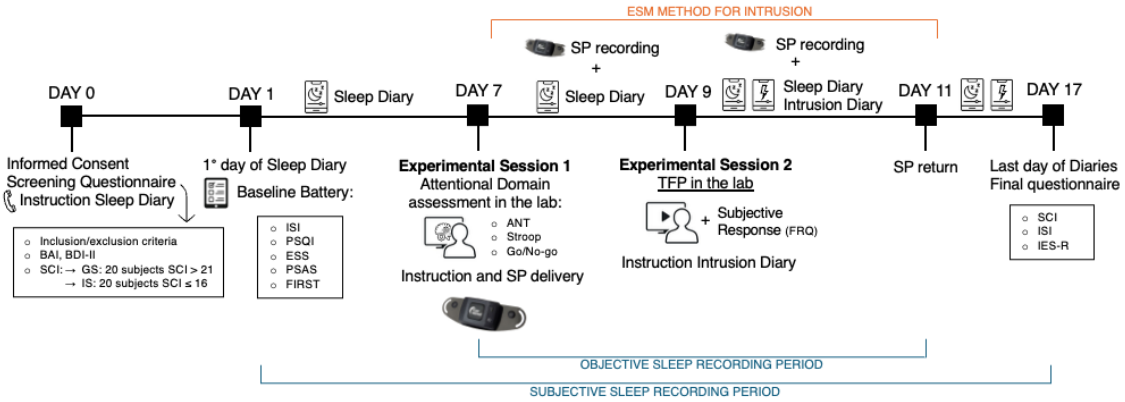

Figure 24. *Schematic overview of the 17-day experimental protocol. The figure illustrates the chronological sequence of the study phases, specifying the different instruments used for data collection at each stage of the protocol.*
BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory - II; SCI = Sleep Condition Indicator; GS = Good Sleepers (group); IS = Insomnia Symptoms (group); ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; PSAS = Pre-Sleep Arousal Scale; FIRST = Ford Insomnia Response to Stress Test; ANT = Attention Network Test; SP = Sleep Profiler; ESM = Experience Sampling Method; FRQ = Film Response Questionnaire; IES-R = Impact of Event Scale Revised.

6.1.3. Materials

6.1.3.1 Questionnaires and Diaries

All the questionnaires and diaries included in the protocol were administrated online using Google Form.

The screening questionnaire, aside assessing eligibility criteria and collecting demographical information, included BAI and BDI-II to evaluate the presence of anxiety

and depressive symptoms, and SCI. Participants were included in the protocol only if their BAI and BDI-II scores were below the clinical threshold (BDI-II score < 14; BAI score < 16) (Beck et al., 1996; Solaro et al., 2016; Beck et al., 1988; Sica & Ghisi, 2007). The SCI was used to determine the presence of ID symptoms and to divide the sample in GS and IS groups. This tool is an 8-item self-report questionnaire developed to screen for ID in accordance with DSM-5 diagnostic criteria. It assesses perceived sleep quality and difficulties over time, and it is specifically designed for use in non-clinical samples, making it particularly suitable for identifying subclinical ID symptoms and vulnerability to sleep disturbance (Espie et al., 2014; Palagini et al., 2015).

The baseline battery comprised five self-report questionnaires assessing various dimensions of sleep functioning:

1. ISI: a 7-item questionnaire assessing the severity, and impact of ID. Items investigate sleep onset, maintenance, satisfaction, daytime impairments, and distress related to sleep problems (Bastien et al, 2001; Castronovo et al., 2016). Although the SCI was used to classify participants into groups, the ISI was administered to provide convergent validation for the SCI-based classification.
2. Pittsburgh Sleep Quality Index (PSQI): a 19-item questionnaire evaluating subjective sleep quality and disturbances over the previous month to obtain a comprehensive index of subjective sleep quality across multiple dimensions (Buysse et al., 1989; Curcio et al., 2013).
3. Epworth Sleepiness Scale (EES): a 8-item questionnaire measures daytime sleepiness and propensity to fall asleep in different everyday situations (Johns, 1991; Vignatelli et al., 2003). It was included as daytime sleepiness may reflect insufficient or disturbed nocturnal sleep.
4. PSAS: a 16-item questionnaire assesses cognitive and somatic arousal experienced at bedtime, included to assess the presence of pre-sleep hyperarousal (Nicassio e al., 1985; Palagini et al., 2016).
5. FIRST: a 9-item questionnaire assessing individual differences in trait SR (Drake et al., 2004; Palagini et al., 2014).

Immediately after the TFP session, subjects completed the FRQ, the same questionnaire developed by Arnaudova & Hagenaars (2017), and included in the previous experiment, to evaluate subjective vision experience (see Appendix B).

In the final questionnaire, the SCI was re-tested to verify whether participants' classification into the two groups changed after video exposure. The ISI was also included to monitor potential variations in the perceived severity and impact of ID symptoms following the stress-induction procedure. Finally, the IES-R, adapted to the content of the traumatic film, was administered to assess the presence and intensity of PTSD-like symptoms experienced during the week after video exposure (Weiss & Marmar, 1997; Pietrantonio et al., 2003; Arnaudova & Hagenaars, 2017).

Finally, the same online Sleep Diary and Intrusion Diary described in the first experiment (and reported in Appendix A) were used in this second study to monitor subjective sleep parameters and intrusive memories across the experimental phases. However, to address the issue of low compliance observed in the daily completion of the Intrusion Diary, we implemented ESM. This is a research approach that prompts individuals to provide systematic self-reports in response to scheduled alerts. In this study, ESM was employed to monitor intrusive, involuntary mental thoughts or images related to the TFP. Participants received messages at fixed times (9:00 a.m., 1:00 p.m., 5:00 p.m., and 9:00 p.m.) throughout the seven-day after video viewing, reminding them to complete the Intrusion Diary. Each entry recorded whether intrusions had occurred since the previous alert. Moreover, each time participants received the reminder, they were required to report via message the number of intrusions experienced since the last ESM alert (Larson & Csikszentmihalyi, 2014). From the Sleep Diary we extracted the same indices used in the first experiment listed in Table 13.

6.1.3.2 Attentional Domain Assessment

Attentional performance was assessed through three computerized tasks: the ANT, the Stroop Task, and the Go/No-Go Task. These measures were included to evaluate distinct but complementary components of attentional functioning: alerting, orienting, and executive control; and to examine how individual differences in attentional efficiency might modulate sleep response to stress.

All tasks were administered on a personal computer (15.6-inch monitor, 60 Hz refresh rate) in a quiet, dark room. The screen brightness was maintained at 80% for all participants, and all individuals had normal or corrected-to-normal vision. Participants were seated approximately 60 cm from the screen. Each testing session took place in the

afternoon, between 3:00 p.m. and 6:00 p.m., ensuring that at least two hours had elapsed since lunch to minimize post-prandial fatigue. Participants were instructed to abstain from caffeine consumption on the day of testing unless they were habitual coffee drinkers, in which case they were permitted a maximum of one cup before noon. Before starting the session, participants were asked to complete the KSS to evaluate their sleepiness. The testing proceeded only if the self-reported score was above 7, ensuring an adequate level of alertness.

Each task was preceded by a short practice session to ensure participants had correctly understood the instructions. The experimenter verified comprehension and clarified any doubts prior to starting the main trials.

Reaction times (RTs) and accuracy rates were recorded for all tasks.

Participants completed the attentional tasks described below in fixed order:

1) The ANT was administered using the Presentation software (version 23.1 Build 09.29.22) (Neurobehavioral Systems, 2022). This task provides an integrated measure of the three major attentional networks: alerting, orienting, and executive control within a single session lasting approximately 30 minutes divided in three identical (in terms of duration and instruction) blocks. Specifically, it consisted of three blocks of 96 trials each, with the number of trials balanced across cue and flanker conditions to ensure an even distribution of experimental manipulations. During the task, participants were required to respond as quickly and accurately as possible to the direction (left or right) of a central arrow that appeared on the screen, by pressing the corresponding button on the mouse. At the beginning of each trial, a fixation cross was presented at the centre of the screen and remained visible throughout the task. The target arrow appeared either above or below this fixation point. Each target was preceded by different cue conditions that manipulated warning (presence or absence of a temporal cue) and spatial (valid or invalid location cues) information, and was accompanied by flanker stimuli, two arrows positioned on each side of the target, that could be congruent (pointing in the same direction as the target) or incongruent (pointing in the opposite direction) or neutral (the central arrow is surrendered by dashes "-") . These manipulations allowed for the estimation of the efficiency of the three attentional networks. A schematic representation of the task is shown in Figure 25. For each participant, mean reaction times (RTs) were computed for correct

responses only, and extreme values (RTs shorter than 200 ms or exceeding ± 2 standard deviations (SD) from the individual mean) were excluded. The efficiency of the three attentional networks was calculated according to the procedure described by Fan and colleagues (2002):

- The *alerting effect* was obtained by subtracting the mean RT of the double cue condition from that of the no cue condition, reflecting the benefit of temporal warning, without spatial indication, on response speed.
- The *orienting effect* was computed as the difference between the mean RTs in the centre cue condition and those in the spatial cue condition, representing the efficiency of spatial attention in guiding perceptual processing, while controlling for the alerting component, which is present in both cue conditions.
- Finally, *the executive control effect* was calculated by subtracting the mean RT of all congruent trials from that of all incongruent trials, indexing the efficiency of conflict resolution and response inhibition.

Higher scores in the alerting and orienting effects indicate greater efficiency of those networks, whereas larger conflict effects reflect reduced executive control efficiency (Fan et al., 2002).

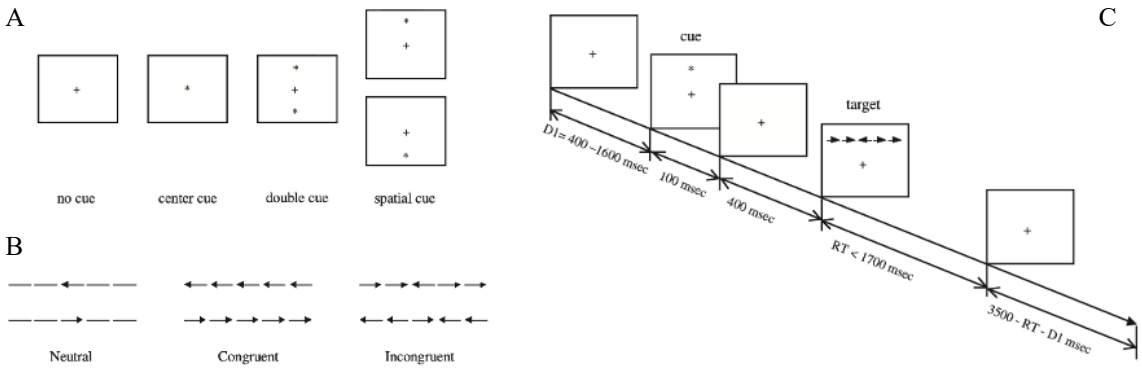

Figure 25. *Schematic representation of the Attention Network Test.*
(A) Illustration of the four cue conditions used to manipulate temporal and spatial attention: no cue (baseline), central cue (warning without spatial information), double cue (temporal warning with two simultaneous cues), and spatial cue (valid or invalid location cue indicating the probable position of the upcoming target). (B) Example of the three flanker conditions: neutral (non-directional flankers), congruent (flanker arrows pointing in the same direction as the target) and incongruent (flankers pointing in the opposite direction). (C) Example of a single trial sequence: each trial begins with a central fixation cross, followed by a cue (depending on condition), a brief

interstimulus interval with the central fixation cross, and then the target display containing the central arrow and flankers. Participants respond as quickly and accurately as possible to the direction of the target arrow and we measure reaction times. RT = Reaction Times; msec = milliseconds (Adapted from Fan et al., 2002).

2) The Stroop Task, implemented using PsychoPy (version 2024.2.2) (Peirce et al., 2019), was included as an additional measure of executive control, focusing specifically on inhibitory processes and cognitive interference control. Participants were instructed to respond as quickly and accurately as possible to the colour of visually presented words displayed at the centre of the screen. The task lasted for approximately 5 minutes and consisted of 150 trials. Each word stimulus remained on the screen for 1000 ms, followed by an interstimulus interval (ISI) of 500 ms. Stimuli consisted of the Italian colour words "*rosso*" (red), "*verde*" (green), and "*blu*" (blue). Responses were given using the arrow keys on the keyboard: the left arrow corresponded to red, the right arrow to blue, and the down arrow to green. Two types of trials were presented: congruent (e.g., "rosso" written in red) and incongruent (e.g., "rosso" written in blue). The Stroop interference effect, defined as the increase in reaction times and/or errors in incongruent trials compared with congruent ones, reflects the capacity to inhibit automatic responses (word reading) in favour of goal-directed control (colour naming). This task complements the ANT by isolating top-down inhibitory control from spatial or alerting influences, thus providing a purer measure of executive attention (Stroop, 1935).
3) Lastly, the Go/No-Go Task was administered using the Test of Attentional Performance (TAP) (version 2.3) (Zimmermann & Fimm, 2016) and lasted approximately 10 minutes. The task consisted of 25 visual stimuli, each presented for 1000 ms followed by an ISI of 500 ms. It consisted of five geometric square stimuli (see figure 26), each containing a different Greek-like pattern. Among these, two were designated as "go" stimuli and three as "no-go" stimuli. Participants were instructed to press the button key as quickly and precisely as possible to "go" stimuli and to withhold responses to "no-go" stimuli. The primary dependent variable was the accuracy, reflecting failures of response inhibition. The Go/No-Go Task targets the motor inhibition component of executive control, which is critical for impulse suppression and behavioural regulation. Including all the paradigms allowed for a more comprehensive assessment of inhibitory mechanisms, disentangling cognitive

interference control (Stroop) from motor response inhibition (Go/No-Go) (Newman & Kosson, 1986).

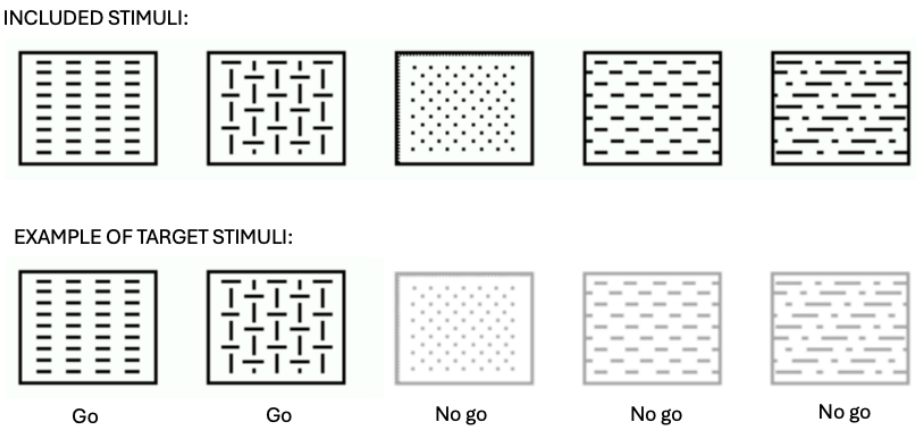

Figure 26. *Visual representation of the Go/No-Go task stimuli. The figure displays the five square-shaped visual stimuli used in the Go/No-Go task, each containing a different Greek-like pattern. Two of these stimuli served as Go signals, requiring a speeded button press, while the remaining three served as No-Go signals, to which participants were instructed to withhold their response.*

6.1.3.3 Objective Sleep Assessment: the Sleep Profiler

To monitor objective sleep in the home environment, we used a portable sleep monitoring device: the Sleep Profiler (SP). This is a lightweight portable EEG system consisting of three frontopolar sensors providing EEG, EOG, and EMG signals. In addition, submental EMG electrodes were included. The device consists of an easy-to-apply elastic headband, which is adjustable to the participant's head size, and is capable of recording multiple consecutive nights (Levendowski et al., 2017). Participants were instructed to recharge the device for at least two hours during the day between recording sessions.

During the first laboratory session, following the attentional domain assessment, participants were instructed on how to apply and use the device. Each participant was provided with a case containing:

- The SP device and its charging cable;
- Disposable electrodes (including the submental ones) sufficient for two nights of recording;

- Two alcohol wipes, few cotton swabs, and a small container with a dedicated skin scrub;
- The connecting cables for the submental electrodes;
- A printed summary of the instructions.

Participants were instructed to wear the device immediately before going to bed to fall asleep. To do so, they were asked to stand in front of a mirror and first wash their face carefully, with special attention to the forehead and chin. Once the skin was dry, they were asked to wipe the electrode sites with the alcohol pad and gently apply a small amount of the scrub using the cotton swabs to clean the skin. Next, they attached the disposable electrodes to the device and placed the headband on their head without removing the protective plastic covers, in order to adjust the fit and ensure stability. Once properly positioned, they removed the protective films and reapplied the headband, making sure that the electrodes adhered well to the skin. Participants were then instructed to turn on the device, remain still during the brief calibration, and turn it off upon their final morning awakening

Participants were instructed to wear the device for four consecutive nights, during which they were asked to maintain their regular sleep-wake schedules and to behave as naturally as possible during the night, as if they were not wearing the device. They were required to bring the device back to the laboratory during the second visit in order to download the data from the first two nights of recording, clean the device and recharge the SP case.

The device software performs automatic sleep staging in 30-second epochs with an accuracy comparable to manual scoring (Levendowski et al., 2017). The output includes a comprehensive report containing, in addition to signal quality indices, several macro- and microstructural sleep parameters. Nights with EEG signal quality below 80% or EOG quality below 60% were excluded from the analyses. The report includes the hypnogram with percentages and latencies of each sleep stage, SL, WASO, TST, %SE, NAWK, as well as the number and density of cortical arousals and microarousals. Additionally, the device provides exportable signal files from which specific features were extracted, including the number and duration of sleep spindles (detected using the device's proprietary automated detection algorithm, as described by Lewandowski et al. 2017) and

frequency band information. The entire recorded signal can be visually inspected, and manual corrections can be applied to both sleep staging and arousal/spindle detection. All automatically scored recordings were manually reviewed and corrected to increase scoring accuracy. A new detection of arousals was performed to obtain the number of REM-related arousals, and automatically detected spindles were filtered according to standard duration criteria, which represent the gold standard for spindle identification (Iber et al., 2007). Table 18 lists the sleep indices and their descriptions that were considered for the present study.

Sleep Index	Definition and Extraction Method
oSL	Sleep Latency (expressed in minutes): automatically detected as the time elapsed from the start of the recording to the first N1 epoch
oTST	Total Sleep Time (expressed in minutes): automatically detected as the time elapsed from the first N1 epoch to the last awakening before the end of recording
oSE%	Sleep Efficiency (expressed in percentage): automatically detected as the ratio between oTST and the entire duration of recording
N1%	Stage 1 length (express in percentage): automatically detected as total amount of N1 sleep occurring after sleep onset, measured from the first N1 epoch to the end of recording
N2%	Stage 1 length (express in percentage): automatically detected as total amount of N2 Sleep occurring after sleep onset, measured from the first N1 epoch to the end of recording
N3%	Stage 1 length (express in percentage): automatically detected as total amount of N3 Sleep occurring after sleep onset, measured from the first N1 epoch to the end of recording
REM%	Stage 1 length (express in percentage): automatically detected as total amount of REM Sleep occurring after sleep onset, measured from the first N1 epoch to the end of recording
N3λ	Latency to N3 (expressed in minutes): automatically detected as the time interval from the sleep onset (first N1 epoch) to the first occurrence of an N3 epoch
REMλ	Latency to REM (expressed in minutes): automatically detected as the time interval from the sleep onset (first N1 epoch) to the first occurrence of an REM epoch
oWASO	Wake after Sleep Onset (expressed in minutes): automatically detected as the total amount of wakefulness occurring after the first sleep onset (first N1 epoch) and before the end of recording
oNAWK	Number of Awakenings: automatically detected as the total number of awakening (transition to W) occurring after the first sleep onset (first N1 epoch) and before the last awakening
CA	Density of cortical arousals (expressed in number per hour): manually detected as total number of cortical arousals divided by the oTST multiplied by 60. Cortical arousals were defined according to AASM criteria as abrupt shifts in EEG frequency lasting at least 3 seconds and following at least 10 seconds of stable sleep
MA	Density of movement arousals (expressed in number per hour): manually detected as total number of movement arousal divided by the oTST multiplied by 60. Movement arousals were defined as brief awakenings or EEG activations associated with increased EMG or body movement activity
REM_A	Density of REM arousals (expressed as number per hour) was calculated as the total number of REM arousals divided by the number of minutes spent in REM sleep and multiplied by 60. REM arousals were defined as abrupt shifts in EEG frequency lasting at least 3 seconds, accompanied by an increase in submental muscle tone lasting at least 1 second.
Spindle_d	Density of Spindle (expressed in number per hour): manually detected as total number of sleep spindles divided by N2 (min), multiplied by 60. Sleep spindles were defined as distinct EEG oscillations in the sigma frequency range (11–16 Hz) lasting between 0.5 and 2 seconds, according to standard AASM criteria.
fDelta	Relative power of the delta frequency band (0.5–4 Hz) during NREM sleep: automatically detected as the ratio between delta band power and total EEG spectral power during NREM stages.

fTheta	Relative power of the theta frequency band (4-8 Hz) during NREM sleep: automatically detected as the ratio between delta band power and total EEG spectral power during NREM stages.
fAlpha	Relative power of the delta frequency band (8-12 Hz) during NREM sleep: automatically detected as the ratio between delta band power and total EEG spectral power during NREM stages.
fSigma	Relative power of the delta frequency band (12-16 Hz) during NREM sleep: automatically detected as the ratio between delta band power and total EEG spectral power during NREM stages.
fBeta	Relative power of the delta frequency band (16-30 Hz) during NREM sleep: automatically detected as the ratio between delta band power and total EEG spectral power during NREM stages.

***Table 18**. Sleep Indeces. This table summarizes the objective sleep indices derived from the Sleep Profiler recordings. Each index is reported with its abbreviation, definition, and a concise description of the extraction procedure. All indices were computed after manual revision and correction of the automatic sleep staging to ensure accuracy and consistency across participants. Macrostructural indices (e.g., sleep stages, latencies, and efficiency measures) were extracted from the corrected hypnogram generated by the Sleep Profiler software, while microstructural indices (e.g., arousals and spindles) were manually detected and verified by the experimenter according to AASM criteria (Iber et al., 2007).*

6.1.3.4 Stress Induction Procedure

The second laboratory session always took place between 6:00 p.m. and 8:00 p.m., ensuring that the stress induction occurred not long before participants' bedtime. Participants were informed that they would be exposed to a video containing traumatic content and were explicitly told that they could interrupt the session at any time if they wished. Otherwise, they were instructed to keep their attention on the screen for the entire duration of the video. The validated RT1 video from our previous experiment was used: the rape scene from Irreversible (Gaspar Noé, 2002). As it had proven to be one of the most effective stimuli in eliciting both immediate psychological and physiological responses, and delayed emotional effects. The video was presented in a dark and silent room on a personal computer (15.6-inch monitor) positioned approximately 60 cm from the participant. The video was played using VLC Media Player (version 3.0.20) (VideoLAN, 2023), with the audio volume set to 80% and the screen brightness fixed at 80%. After viewing the video, participants completed the FRQ (Arnaudova & Hagenaars, 2017) on a separate computer. They were then given time to remain alone if they wished and were offered the opportunity to discuss the content with the experimenter, who was a licensed psychologist.

Before leaving the laboratory with the recharged Sleep Profiler kit, participants were reminded of the intrusion diary procedure and trained in the ESM that would follow.

6.1.4. Statistical Analysis

All statistical analyses were performed using Jeffreys's Amazing Statistics Program (JASP, Version 0.18.2.0; 2024) and MATLAB (R2021b; MathWorks, Inc., Natick, MA, USA). The level of statistical significance was set at p-value < 0.05 for all tests, and given the large number of statistical comparisons, p-values were adjusted using Bonferroni correction.

For the analyses focused on both objective sleep indices, obtained from the Sleep Profiler, and subjective indices, derived from the Sleep Diary, we performed pre/post comparisons across the whole sample and further examined group × time interactions using rmANOVAs. For the Sleep Diary data, we maintained the same analytical approach used in the previous experiment, conducting comparisons across two temporal windows: the entire week before versus the entire week after the experimental session, and the two days before versus the two days after the session. This approach allowed us to evaluate both short-term and longer-term effects of the manipulation on subjective sleep quality.

Paired-samples t-tests were conducted to assess differences between pre-and post-scores on the SCI and the ISI, as well as potential differences between the two experimental groups.

Finally, to investigate how attentional performance modulated the sleep response, we conducted a Generalized Linear Mixed Model (GLMM) analysis that included attentional indices as predictors, sleep-related measures as dependent variables, and group as a fixed factor to assess whether this relationship differed between the two experimental conditions. All GLMMs were specified with a Gaussian family and identity link function. Participant ID was included as a random factor to account for within-subject variability across repeated measures. Models were estimated using restricted maximum likelihood, and model diagnostics confirmed adequate convergence and homoscedastic residuals.

When assumptions of normality or sphericity were not met, appropriate non-parametric alternatives were applied.

6.2. Results

6.2.1. Sample Characteristics

Descriptive statistics are reported in Table 19. The final sample included 40 participants, divided between the two groups (20/20 M/F), matched by age and gender. The mean age of the entire sample was 29.2 ± 7.0 years, with a range: 20-58.

As shown in the table, the baseline sleep profile significantly differed between the two groups for all the questionnaires included, except for ESS. These differences were assessed using one-tailed independent-samples t-tests6 for normally distributed variables and Mann–Whitney U tests for non-normally distributed ones, revealed a generally more impaired sleep pattern in the IS group, characterized by poorer subjective sleep quality, higher pre-sleep arousal, and greater SR compared to good sleepers.

The immediate subjective response to the video, as measured by the FRQ, indicated high scores for negative valence (-1.5 ± 0.8), arousal (6.4 ± 2.3), distress (6.7 ± 2.0), disgust (8.5 ± 1.1), involvement (6.4 ± 2.1) and attention (8.3 ± 0.8) and medium scores in terms of immobility (4.1 ± 2.6) and embarrassment (4.0 ± 2.6). Moreover, the video was equally effective in immediately eliciting a negative psychological response in both groups.

No significant differences emerged between groups also when considering the subjective delayed impact of the video, as measured by the number and characteristics of reported intrusions and by the IES-R score. Compliance improved markedly compared to the previous validation study, thanks to the implementation of the ESM method. Participants reported, on average, a higher number of intrusions (4.4 ± 5.5) which, however, were rated as less vivid (4.0 ± 1.3) and less uncomfortable (3.5 ± 1.6), compared to the previous experiment, yet characterized by a high perceived control (5.2 ± 1.4) and similar level of spontaneity (3.3 ± 1.3). Mean IES-R scores were very low in both groups (12.2 ± 11.0).

Regarding ID symptoms, a significant increase was observed across the entire sample from baseline to the end of the protocol, as measured by the SCI ($Z = 2.77$, $p = 0.006$) and ISI ($Z = 1.97$, $p = 0.035$) questionnaires. This indicates that the TFP successfully induced sleep disturbances at a global level. Consistently, the rmANOVA on ISI scores, with time (pre/post) as a within-subject factor and group as a between-subject factor,

revealed a significant time-by-group interaction ($F(1, 38) = 4.60$, $p = 0.038$), indicating that the increase in ID complaints was more pronounced in the IS group than in the GS group.

	n (%)	Mean ± SD		Range (min-max)	*p-value*
Age		29.2 ± 7.0 years		20-58	
Sex (M/F)	20/20 (50%/50%)				
		GS	**IS**		
Questionnaires					
SCI_pre		28.5 ± 12.4	13.5 ± 1.9	8-32	< 0.001†
ISI_pre		3.0 ± 3.9	13.1 ± 3.1	0-18	< 0.001†
PSQI		4.1 ± 2.8	8.9 ± 3.3	0-15	< 0.001
FIRST		17.6 ± 4.2	23.6 ± 5.3	10-31	< 0.001
ESS		5.2 ± 3.6	5.0 ± 3.6	0-14	0.552†
PSAS		30.7 ± 9.4	50.1 ± 13.4	22-75	< 0.001
SCI_post		28.1 ± 3.2	12.6 ± 2.8	5-32	< 0.001†
ISI_post		3.4 ± 0.7	20.7 ± 3.7	0-24	< 0.001†
IES-R		12.4 ± 10.6	12.1 ± 11.7	1-43	0.612†
FRQ					
Valence		-1.4 ± 0.9	-1.5 ± 0.8	-4 - -1	0.769†
Arousal		6.6 ± 2.2	6.2 ± 2.4	2-9	0.691†
Immobility		4.1 ± 2.4	4.0 ± 2.8	1-9	0.681†
Distress		6.9 ± 1.6	6.5 ± 2.4	1-9	0.868†
Disgust		8.5 ± 0.7	8.4 ± 0.5	3-9	0.663†
Embarrassment		3.9 ± 2.4	4.0 ± 2.9	1-9	0.956†
Involvement		6.5 ± 1.8	6.4 ± 2.4	1-9	0.978†
Attention		8.3 ± 0.8	8.3 ± 0.9	6-9	0.824†
Intrusions					
N°		4.0 ± 5.0	4.8 ± 6.0	0-26	0.459†
Vividness		4.0 ± 1.2	3.9 ± 1.4	1-6	0.925
Discomfort		3.8 ± 1.6	3.2 ± 1.6	1-6	0.516†
Control		5.1 ± 1.2	5.3 ± 1.6	2-7	0.171†
Spontaneity		3.0 ± 1.1	3.6 ± 1.4	1-5	0.797†

Table 19. *Descriptive statistics and group comparison on demographic variables, questionnaire scores, and immediate and delayed subjective impact of the video.*
SCI = Sleep Condition Indicator; ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; FIRST = Ford Insomnia Response to Stress Test; ESS = Epworth Sleepiness Scale; PSAS = Pre-Sleep Arousal Scale; IES-R = Impact of Event Scale – Revised; FRQ = Film Response Questionnaire; N° = total number of intrusions; M/F = male/female; n = number of subjects; SD = standard deviation; min = minimum; max = maximum.
† Mann–Whitney U test was conducted

6.2.2. Subjective Sleep Response

As in the previous experiment, two temporal windows were considered to examine subjective sleep changes following the TFP: a short-term window, comparing the two nights immediately before and after the exposure, and a long-term window, encompassing the entire week preceding and the week following the video session.

To evaluate pre–post differences and determining whether these changes differed between groups, rmANOVAs were performed with time (pre/post) as a within-subject factor and group as a between-subject factor. Significant results relative to the group-by-time interaction are illustrated in Figure 27. In the short-term window, a significant group-by-time interaction emerged for both subjective and calculated TST (sTST: $F(1, 38) = 6.7$, $p = 0.013$; cTST: $F(1, 38) = 9.9$, $p = 0.003$) and both subjective and calculated SE% (sSE%: $F(1, 38) = 10.9$, $p = 0.002$; cSE%: $F(1, 38) = 5.6$, $p = 0.023$), indicating a larger post-exposure decline in the IS group compared with the GS group. Considering the weekly window, a significant group-by-time interaction was also found for sTST ($F(1, 38) = 5.5$, $p = 0.025$), again reflecting a more pronounced deterioration in the IS subgroup compared with GS. Two additional near-significant trends were observed for sSE% ($F(1, 38) = 3.3$, $p = 0.075$) and cTST ($F(1, 38) = 2.9$, $p = 0.093$).

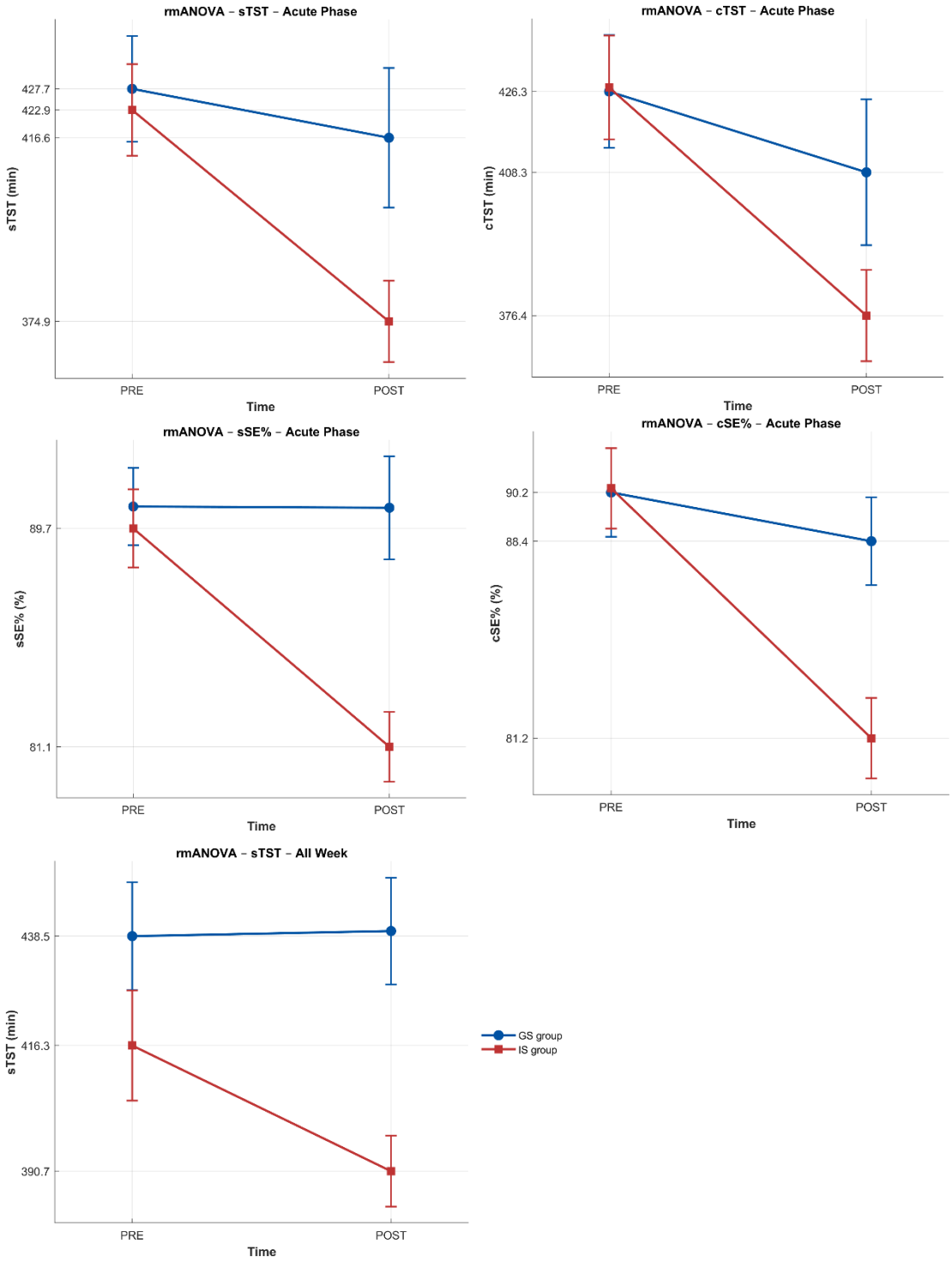

Figure 27*. Line plots depicting pre–post significative changes in subjective sleep parameters for the GS and IS groups obtained from the rmANOVA.*
Each panel shows mean ± Standard Error of the Mean (SEM) values for GS (blue) and IS (red). In the short-term window, significant group × time interactions were found for subjective and calculated total sleep time (sTST, cTST) and for subjective and calculated sleep efficiency (sSE%, cSE%), indicating a greater post-exposure decline in the IS group compared with the GS group. In the weekly window, a significant interaction emerged for sTST. sTST = Subjective Total Sleep Time; sSE% = Subjective Sleep Efficiency; cTST = Calculated Total Sleep Time; cSE% =

Calculated Sleep Efficiency; min = minutes; IS = Insomnia Symptoms Group; GS = Good Sleepers Group.

To further characterise these interaction effects, post hoc analyses were conducted.

We investigated the main effect of the time using a paired-sample t-tests, and when assumptions of normality were violated, Wilcoxon signed-rank tests were applied. Results are presented in Table 20. Within-group comparisons across the whole sample confirmed a general worsening of subjective sleep following the exposure. Considering the *short-term window*, a significant worsening was observed across the whole sample in SL ($Z = 90.0$, $p < 0.001$), in both subjective and calculated TST (sTST: $t = 3.9$, $p < 0.001$; cTST: $Z = 648.0$, $p < 0.001$), in both subjective and calculated SE% (sSE%: $t = 3.0$, $p = 0.002$; cSE%: $t = 3.8$, $p < 0.001$), in WASO ($Z = 221.0$, $p = 0.040$), and in perceived depth of sleep ($t = 3.9$, $p < 0.001$). Considering the *long-term window*, significant deteriorations were also found for both subjective and calculated TST (sTST: $t = 2.0$, $p = 0.024$; cTST: $t = 2.5$, $p = 0.007$) and for both subjective and calculated SE% (sSE%: $t = 1.5$, $p = 0.027$; cSE%: $Z = 608.0$, $p = 0.003$).

Sleep Index	Derivation Method	2 Days Pre (M ± SD)	2 Days Post (M ± SD)	Stat	Week Pre (M ± SD)	Week Post (M ± SD)	Stat
SL_min	Estimated by the subject	9.6 ± 4.8	15.0 ± 10.5	90.0**†	12.8 ± 8.8	14.9 ± 9.7	346.0†
TIB_min	Lights out time – (Woke up time + Time in bed after awakening)	475.4 ± 70.1	462.7 ± 62.7	1.4	490.3 ± 53.6	483.7 ± 46.4	2.5
sTST_min	Estimated by the subject	425.3 ± 49.7	395.7 ± 61.0	3.9**	437.4 ± 50.3	411.1 ± 47.6	2.0*
sSE%	sTST/TIB	90.2 ± 6.8	85.8 ± 9.0	3.0*	90.6 ± 7.5	86.3 ± 7.9	1.5*
cTST_min	Woke up time – (Lights out time + SL + WASO)	426.7 ± 53.2	392.4 ± 61.9	648.0†**	431.8 ± 48.5	418.5 ± 43.4	2.6*
cSE%	cTST/TIB	90.3 ± 6.8	84.8 ± 7.7	3.8**	91.3 ± 6.3	86.6 ± 6.0	608.0*†
NAWK	Estimated by the subject	1.4 ± 1.2	1.7 ± 1.5	1.5	1.3 ± 1.0	1.4 ± 1.1	0.8
WASO_min	Estimated by the subject	6.9 ± 5.8	10.8 ± 12.7	221.0*†	11.4 ± 12.4	11.5 ± 9.7	324.0†
Dreams	Reported by the subject	0.5 ± 0.3	0.6 ± 0.4	357.5†	0.6 ± 0.3	0.6 ± 0.3	-0.5
Deepness	Reported by the subject	3.2 ± 0.7	2.5 ± 0.8	3.9*	3.4 ± 0.6	3.3 ± 0.6	0.6
Quietness	Reported by the subject	3.1 ± 0.8	2.2 ± 0.9	4.3	2.2 ± 0.6	2.2 ± 0.8	0.6
Restorativeness	Reported by the subject	3.1 ± 0.8	3.2 ± 0.7	129.0†	3.2 ± 0.6	3.1 ± 0.7	0.9

***Table 20**. Comparison in sleep quality indices from the Sleep Diary collected during the two days before and the two days after the video session (left panel), and during the week before and the week after the experimental session (right panel). Derivation mode indicates how each index was obtained. "Lights out time" refers to the moment participants decided to stop any activities to fall asleep.*
Stat = Statistic; SL_min = Sleep Latency (minutes); TIB_min = Time in Bed (minutes); sTST_min = Subjective Total Sleep Time (minutes); sSE% = Subjective Sleep Efficiency; cTST_min = Calculated Total Sleep Time (minutes); cSE% = Calculated Sleep Efficiency; NAWK = Number

of Awakenings; WASO_min = Wake After Sleep Onset (minutes); Dreams = Number of Dreams; Deepness = Perceived Sleep Depth; Quietness = Perceived Sleep Quietness; Restorativeness = Perceived Sleep Restorativeness; TFP = Trauma Film Paradigm; M = mean; SD = standard deviation.
† Wilcoxon signed-rank test was conducted
** $p < 0.05$; ** $p < 0.001$.*

Between-group post hoc comparisons further clarified the source of the interactions using an independent-samples tests (parametric when assumptions were met; otherwise, Mann–Whitney) as showed in figure 20. Starting with the *short-term window*, during the two days preceding the TFP exposure the two groups did not differ on any of the sleep diary indices considered, with the sole exception of perceived restorativeness (t = 2.9, p = 0.003). In the two days following the exposure, however, the IS group showed a markedly more impaired sleep profile than the GS group, particularly in terms of both subjective and calculated TST (sTST: t = 2.3, p = 0.014; cTST: U = 284.5, p = 0.012), both subjective and calculated SE% (sSE%: U = 346.0, p < 0.001; cSE%: U = 347.0, p < 0.001), and perceived restorativeness (U = 280.0, p = 0.011).

At baseline, in the *long-term window* diary-derived estimates were significantly poorer in the IS group for WASO (U = 37.5, p < 0.001), in both subjective and calculated SE% (sSE%: U = 270.0, p = 0.030; cSE%: U = 292.5, p = 0.006), perceived depth (t = 3.9, p < 0.001), and perceived restorativeness (t = 5.5, p < 0.001). A near-significant trend was observed for SL (U = 148.0, p = 0.082), sTST (t = 1.4, p = 0.082), and cTST (t = 1.5, p = 0.065). At post-exposure, the IS group showed a markedly poorer sleep profile than the GS group, with significant group differences for sTST (t = 7.6, p < 0.001), both subjective and calculated SE (sSE%: t = 24.6, p < 0.001; cSE%: t = 27.9, p < 0.001), WASO (U = 84.0, p < 0.001), NAWK (U = 132.5, p = 0.035), perceived depth (t = 2.6, p = 0.006), and perceived restorativeness (t = 3.7, p < 0.001).

Sleep Index	Baseline GS (M ± SD)	Baseline IS (M ± SD)	Stat	Post TFP GS (M ± SD)	Post TFP IS (M ± SD)	Stat
			SHORT-TERM WINDOW			
SL_min	9.4 ± 4.3	9.8 ± 5.4	203.5†	12.7 ± 8.6	17.4 ± 11.9	148.0†
TIB_min	475.4 ± 72.6	475.4 ± 69.5	0.3	461.5 ± 70.9	374.9 ± 41.3	1.6
sTST_min	427.7 ± 53.8	422.9 ± 46.5	0.3	416.6 ± 70.9	374.9 ± 41.3	2.3*
sSE%	90.6 ± 6.8	89.7 ± 6.9	204.0†	90.6 ± 9.1	81.1 ± 6.2	346.0†**
cTST_min	426.3 ± 56.2	427.2 ± 51.6	0.5	408.3 ± 72.5	376.4 ± 45.5	284.5†*
cSE%	90.2 ± 7.2	89.7 ± 6.5	0.1	88.4 ± 7.2	81.2 ± 6.6	347.0†**
NAWK	1.8 ± 1.5	1.6 ± 1.5	225.0†	1.3 ± 1.0	1.6 ± 1.4	192.0†
WASO_min	6.6 ± 5.8	7.2 ± 5.9	193.5†	7.6 ± 9.0	13.9 ± 15.2	142.5†
Dreams	0.6 ± 0.3	0.5 ± 0.3	299.5†	0.6 ± 0.5	0.7 ± 0.3	175.0†
Deepness	3.3 ± 0.6	3.2 ± 0.8	226.0†	2.3 ± 0.8	2.6 ± 0.9	1.0
Quietness	2.3 ± 0.8	2.1 ± 1.0	236.0†	3.4 ± 0.6	2.7 ± 0.8	2.9
Restorativeness	3.4 ± 0.6	2.7 ± 0.8	2.9*	3.4 ± 0.5	2.9 ± 0.7	2.5*
			LONG-TERM WINDOW			
SL_min	11.1 ± 7.6	14.6 ± 9.8	148.0†	11.9 ± 8.0	15.9 ± 11.0	154.5†
TIB_min	489.2 ± 56.0	491.5 ± 52.6	216.5	487.7 ± 49.7	479.7 ± 43.6	2.5
sTST_min	438.5 ± 49.1	416.2 ± 50.1	1.4	439.6 ± 48.5	390.7 ± 32.4	7.6**
sSE%	90.1 ± 5.9	85.2 ± 8.3	270.0*†	90.4 ± 5.2	82.2 ± 8.0	24.6**
cTST_min	443.4 ± 50.5	420.1 ± 44.6	1.5	438.9 ± 47.2	398.2 ± 27.7	4.2
cSE%	90.8 ± 4.6	85.8 ± 6.9	292.5*†	89.9 ± 3.6	83.2 ± 6.0	27.9**
NAWK	1.0 ± 0.7	1.7 ± 1.2	198.5†	1.0 ± 0.9	1.6 ± 1.0	132.5*†
WASO_min	4.5 ± 3.1	18.3 ± 14.4	6.5**†	6.4 ± 5.5	16.6 ± 10.4	84.0**†
Dreams	0.6 ± 0.2	0.6 ± 0.3	198.5†	0.6 ± 0.3	0.7 ± 0.3	181.0†
Deepness	3.7 ± 0.6	2.9 ± 0.4	3.9**	3.6 ± 0.5	3.2 ± 0.5	2.6*
Quietness	2.4 ± 0.7	2.1 ± 0.6	1.6	2.4 ± 0.8	1.9 ± 0.7	1.8
Restorativeness	3.6 ± 0.5	2.7 ± 0.4	5.5**	3.4 ± 0.7	2.7 ± 0.5	3.7**

Table 21. *In the upper section of the table, we report the results of the group comparisons on sleep quality indices from the Sleep Diary collected during the two days before (left panel) and the two days after (right panel) the video session. The lower section presents the results of the group comparisons based on the Sleep Diary data collected during the week before (left panel) and the week after (right panel) the video session.*
Stat = Statistic; SL_min = Sleep Latency (minutes); TIB_min = Time in Bed (minutes); sTST_min = Subjective Total Sleep Time (minutes); sSE% = Subjective Sleep Efficiency; cTST_min = Calculated Total Sleep Time (minutes); cSE% = Calculated Sleep Efficiency; NAWK = Number of Awakenings; WASO_min = Wake After Sleep Onset (minutes); Dreams = Number of Dreams; Deepness = Perceived Sleep Depth; Quietness = Perceived Sleep Quietness; Restorativeness = Perceived Sleep Restorativeness; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; TFP = Trauma Film Paradigm; M = mean; SD = standard deviation.
† Mann–Whitney U test was conducted
** $p < 0.05$; ** $p < 0.001$.*

6.2.3. Objective Sleep Response

To analyse the impact of the traumatic video on participants' objective sleep, data from the four recorded nights with SP were considered. Nights with EEG signal quality below 80% or EOG quality below 60% were excluded from the analyses.

To evaluate pre–post differences and determining whether these changes differed between groups, rmANOVAs were conducted with time (pre/post) as a within-subject factor and group (GS/IS) as a between-subject factor. Results are illustrated in Figure 28.

A significant group-by-time interaction emerged for SE% ($F(1, 38) = 7.3$, $p = 0.010$), indicating a larger decrease from pre- to post-video in the IS group, and for REM arousal density ($F(1, 38) = 7.7$, $p = 0.008$), which remained stable from pre to post in GS but increased in IS. Two additional effects approached significance: TST ($F(1, 38) = 3.3$, $p = 0.078$), showing a greater reduction from pre to post in IS, and REM% ($F(1, 38) = 3.1$, $p = 0.086$), remaining stable in GS but declining in IS.

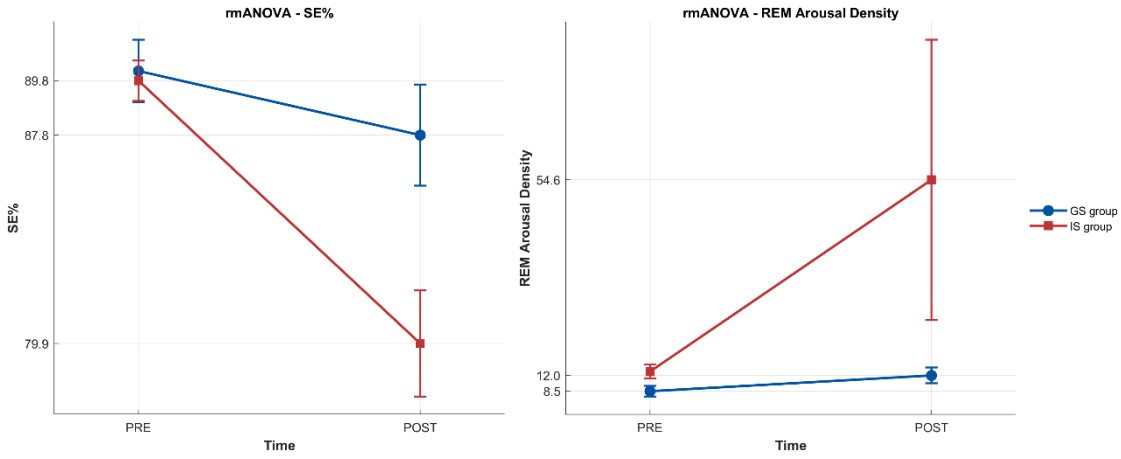

Figure 28. *Line plots depicting pre–post significant changes in objective sleep parameters for the GS and IS groups obtained from the rmANOVA.*
Each panel shows mean ± Standard Error of the Mean (SEM) values for GS (blue) and IS (red). A significant group × time interaction emerged for sleep efficiency (SE%), indicating a larger pre-to-post decrease in the IS group compared with the GS group. A significant interaction was also observed for REM arousal density, which remained stable from pre to post in the IS group but decreased in the GS group. SE% = Sleep Efficiency; IS = Insomnia Symptoms Group; GS = Good Sleepers Group.

To further characterise these interaction effects, post hoc analyses were conducted.

To evaluate the main effect of time, so pre–post differences across the entire sample, paired-sample t-tests were conducted; when normality assumptions were violated, Wilcoxon signed-rank tests were used. Results are presented in Table 22.

Sleep Index	Baseline (M ± SD)	Post TFP (M ± SD)	Stat
oSL_min	12.4 ± 10.4	11.1 ± 8.3	378.5.0†
oTST_min	381.7 ± 54.6	348.1 ± 66.9	3.1*
oSE%	90.0 ± 4.4	83.8 ± 9.5	692.0**†
N1%	3.8 ± 2.3	4.5 ± 2.0	208.0†*
N2%	42.7 ± 10.9	40.4 ± 11.1	2.3*
N3%	31.9 ± 12.8	35.5 ± 14.6	235.0†
REM%	21.5 ± 5.4	19.6 ± 7.9	2.3*
N3λ_min	27.4 ± 18.0	15.4 ± 16.3	181.0†
REMλ_min	88.1 ± 32.4	100.5 ± 56.0	2.6*†
oWASO_min	30.8 ± 16.0	57.4 ± 51.3	144.5**†
oNAWK	3.9 ± 1.1	4.2 ± 1.2	2.0*
CA	14.4 ± 6.5	14.0 ± 7.2	0.9
MA	9.0 ± 6.7	8.1 ± 6.1	1.1
REM_A	13.4 ± 6.4	15.5 ± 5.2	231.0*†
Spindle_d	2.8 ± 2.2	0.6 ± 0.5	741.0**†
fDelta	0.5 ± 0.1	0.5 ± 0.1	-0.6
fTheta	0.2 ± 0.0	0.2 ± 0.0	-1.9
fAlpha	0.1 ± 0.1	0.1 ± 0.0	1.1
fSigma	0.1 ± 0.0	0.1 ± 0.0	1.6
fBeta	0.1 ± 0.1	0.1 ± 0.0	389.0†

Table 22. *Comparison in sleep indices obtained from the Sleep Profiler recordings collected during the week before (left panel) and the week after (right panel) the video session. oSL_min = Objective Sleep Latency (minutes); oTST_min = Objective Total Sleep Time (minutes); oSE% = Objective Sleep Efficiency; REM = Rapid Eye Movements; N3λ_min = Latency to N3 (minutes); REMλ_min = Latency to REM (minutes); oNAWK = Objective Number of Awakenings; oWASO_min = Wake After Sleep Onset (minutes); CA = Cortical Arousal Density; MA = Movement Arousal Density; REM_A =REM Arousal Density; Spindle_d = Spindles Density; **f**Delta = Relative power of the delta frequency; **f**Theta = Relative power of the theta frequency; **f**Alpha = Relative power of the alpha frequency; **f**Sigma = Relative power of the sigma frequency; **f**Beta = Relative power of the beta frequency; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; TFP = Trauma Film Paradigm; M = mean; SD = standard deviation.*
† Wilcoxon signed-rank test was conducted
** $p < 0.05$; ** $p < 0.001$.*

Following exposure to the video, a significant deterioration in objective sleep quality was observed across the full sample, reflected by increased WASO (Z = 144.5, $p < 0.001$), NAWK (t = 2.0, $p = 0.023$) and REM_A (Z = 231.0, $p = 0.022$), reduced TST (t = 3.1, $p = 0.002$), SE% (Z = 692.0, $p < 0.001$) and spindles density (Z = 741.0, $p < 0.001$). In addition, a significant reduction in N2% (t = 2.3, $p = 0.013$) and REM% (t = 2.3, $p = 0.014$) was found, accompanied by an increase in N1% (Z = 208.0, $p = 0.003$).

Finally post hoc analyses were conducted to assess between-group differences (IS vs. GS) in sleep parameters at baseline and after the video exposure, using independent-samples tests. Results are reported in Table 23.

Sleep Index	Baseline GS (M ± SD)	Baseline IS (M ± SD)	Stat	Post TFP GS (M ± SD)	Post TFP IS (M ± SD)	Stat
oSL_min	11.0 ± 7.3	13.7 ± 12.8	180.5†	8.8 ± 8.6	13.5 ± 7.5	114.5.0*†
oTST_min	392.5 ± 59.9	370.8 ± 47.7	1.3	377.9 ± 71.2	318.3 ± 47.4	3.1*
oSE%	90.2 ± 5.3	89.8 ± 3.4	0.3	87.8 ± 8.5	79.9 ± 8.9	321.0**†
N1%	4.0 ± 2.6	3.5 ± 2.1	0.7	4.5 ± 2.2	4.5 ± 1.8	0.2
N2%	42.4 ± 10.2	43.0 ± 11.9	0.1	40.2 ± 10.5	40.7 ± 11.9	0.1
N3%	30.6 ± 11.4	33.4 ± 14.2	171.0†	32.8 ± 13.5	38.1 ± 15.6	1.1
REM%	22.9 ± 5.3	20.1 ± 5.3	1.7	22.5 ± 6.7	16.7 ± 8.0	1.4*
N3λ_min	26.7 ± 21.4	29.6 ± 18.9	213.0†	19.4 ± 18.5	11.9 ± 11.2	233.0†
REMλ_min	83.4 ± 31.9	92.8 ± 32.9	164.0†	94.2 ± 50.8	106.9 ± 61.3	164.0†
oWASO_min	32.4 ± 11.5	29.3 ± 19.7	199.0†	46.7 ± 43.3	68.2 ± 57.4	111.5*†
oNAWK	3.8 ± 1.1	4.1 ± 1.1	5.5	4.2 ± 1.2	4.2 ± 1.1	0.1
CA	14.8 ± 7.2	14.0 ± 6.0	0.4	12.9 ± 6.6	14.9 ± 7.7	206.0†
MA	4.0 ± 2.3	4.2 ± 2.9	0.2	4.3 ± 1.9	4.3 ± 2.0	0.1
REM_A	8.5 ± 0.5	12.0 ± 0.6	112.0*†	11.9 ± 7.6	54.6 ± 7.0	114.5*
Spindle_d	3.0 ± 2.5	2.6 ± 2.0	208.5†	0.6 ± 1.5	0.4 ± 0.5	227.5†
fDelta	0.5 ± 0.1	0.5 ± 0.1	0.7	0.6 ± 0.0	0.5 ± 0.0	0.2
fTheta	0.1 ± 0.0	0.2 ± 0.0	-0.8	0.1 ± 0.0	0.2 ± 0.0	-0.6
fAlpha	0.1 ± 0.1	0.1 ± 0.0	-1.2	0.1 ± 0.0	0.2 ± 0.1	-0.6
fSigma	0.1 ± 0.1	0.1 ± 0.0	0.2	0.1 ± 0.0	0.1 ± 0.0	0.7
fBeta	0.1 ± 0.0	0.1 ± 0.1	167.0†	0.1 ± 0.0	0.1 ± 0.0	162.0†

Table 23. *Group comparison on sleep indices obtained from the Sleep Profiler recordings collected during the week before (left panel) and the week after (right panel) the video session. oSL_min = Objective Sleep Latency (minutes); oTST_min = Objective Total Sleep Time (minutes); oSE% = Objective Sleep Efficiency; REM = Rapid Eye Movements; N3λ_min = Latency to N3 (minutes); REMλ_min = Latency to REM (minutes); oNAWK = Objective Number of Awakenings; oWASO_min = Wake After Sleep Onset (minutes); CA = Cortical Arousal Density; MA = Movement Arousal Density; REM_A = REM Arousal Density; Spindle_d = Spindles Density;* ***f****Delta = Relative power of the delta frequency;* ***f****Theta = Relative power of the theta frequency;* ***f****Alpha = Relative power of the alpha frequency;* ***f****Sigma = Relative power of the sigma frequency;* ***f****Beta = Relative power of the beta frequency; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; TFP = Trauma Film Paradigm; M = mean; SD = standard deviation.*
† Mann–Whitney U test was conducted
** $p < 0.05$; ** $p < 0.001$.*

During the two baseline nights, IS and GS groups did not differ significantly in their objective sleep profiles, except for REM_A, which was significantly higher in the IS group (U = 112.0, p = 0.008). Additionally, a near-significant trend was observed for REM%, with GS showing a tendency toward higher values (t = 1.7, p = 0.050).

In the two post-video nights, several significant differences emerged: IS participants displayed poorer sleep quality, with significantly higher oSL (U = 114.5, p = 0.011), oWASO (U = 111.5, p = 0.009), and REM_A (U = 114.5, p = 0.003), along with lower oTST (t = 3.1, p = 0.002), oSE% (U = 321.0, $p < 0.001$), and REM% (t = 1.4, p = 0.009) compared with the GS group.

6.2.4. Attentive Performance and the Influence on Sleep

Descriptive statistics for participants' attentional performance across the three tasks included in the paradigm are reported in Table 24. Before computing the reaction time (RT) indices, all expressed in milliseconds (ms), data were preprocessed to remove outliers and invalid trials. Anticipatory responses (RT < 150 ms), excessively slow responses (RT > 2500 ms), and incorrect trials were excluded. For each participant and condition, remaining RTs deviating more than three median absolute deviations (MADs) from the individual median were also removed.

Attention Index	All_ms (M ± SD)	GS Group_ms (M ± SD)	IS Group_ms (M ± SD)	Stat, p-value
RT_gonogo	533.5 ± 74.9	518.8 ± 64.6	548.2 ± 83.0	-1.2, 0.231
PES	115.0 ± 159.6	58.9 ± 80.3	230.7 ± 209.53	-2.1, 0.064
RT_stroop	848.4 ± 221.7	872.9 ± 248.9	823.8 ± 194.3	201.0†, 0.563
Interference	80.15 ± 89.6	64.4 ± 95.9	95.9 ± 92.4	139.0†, 0.234
RT_ANT	547.7 ± 83.9	561.8 ± 87.6	587.6 ± 69.2	-0.9, 0.337
Alerting	42.0 ± 30.1	44.6 ± 28.5	39.4 ± 29.1	0.5, 0.585
Orienting	28.6 ± 28.8	23.1 ± 28.2	34.2 ± 29.1	-1.2, 0.227
Ex.Control	95.5 ± 35.9	87.9 ± 36.5	103.1 ± 34.7	-1.4, 0.478

Table 24. *Descriptive statistics and group comparisons for attentional performance indices. RT_gonogo = Mean reaction time (Go/No-Go task); PES = Post-Error Slowing (Go/No-Go task); RT_stroop = Mean reaction time (Stroop task); Interference = Interference score (Stroop task); RT_ANT= Mean reaction time (Attention Network Test); Ex.Control = Executive Control score (ANT); Orienting = Orienting network score (ANT); ms = milliseconds; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; TFP = Trauma Film Paradigm; M = mean; SD = standard deviation. Stat = Statistic.*
† Mann–Whitney U test was conducted
** p < 0.05; ** p < 0.001.*

First, we evaluated whether there were performance differences between groups across all indices using the independent sample t-test. Overall, performance was comparable across tasks, with no significant group differences. A trend, however, emerged for the Post-Error Slowing (PES) in the Go/No-Go task, computed as the difference between

mean RTs in correct Go trials immediately following an error and mean RTs in correct Go trials following another correct response. Positive values indicate adaptive slowing after errors. It is important to note that only 11 out of 40 participants (7 GS, 4 IS) committed errors (and not exclusively at the beginning of the task), which limits the interpretability of this measure. Participants in the IS group tended to show greater post-error slowing than controls (trend-level, non-significant). Similarly, IS participants showed a trend toward longer mean RTs in the ANT task compared to controls, although this difference did not reach statistical significance.

Finally, we also tested whether participants' performance in the ANT varied across the three blocks by means of an rmANOVA, but results showed no significant differences, either when considering both groups together or separately. Therefore, subsequent analyses were conducted on the global indices only.

To evaluate the potential moderating role of attention on the sleep response to a stressor in healthy individuals with and without ID symptoms, we decided to use GLMMs. This approach allowed us to simultaneously consider both measurement points of the sleep variables and to more accurately estimate the effects of stress induction, as well as how these effects were modulated by attentional performance across the two groups.

A series of GLMMs was then run for each objective and subjective sleep parameter (both within the short-term window and the long-term window), evaluated before and after stress induction. In each GLMM, one attentional index derived from the cognitive tasks was included as a predictor to examine its potential modulatory role on sleep responses to stress in the two groups. All GLMMs were specified with a Gaussian family[7] and identity link, as visual inspection of residuals confirmed an approximately normal distribution for all dependent variables. Model diagnostics confirmed adequate convergence and homoscedastic residuals and all models were estimated using restricted maximum likelihood. Participant ID was included as a random factor to account for within-subject variability across repeated measures.

Only the models including ANT indices yielded significant results.

For the subjective sleep indices, the GLMMs revealed significant main effects of Time for perceived sleep depth ($\chi^2 = 4.4$, $p = 0.036$) and perceived restorativeness ($\chi^2 = 5.1$, p

[7] For percentage variables we maintain the Gaussian specification, as their distributions were approximately normal.

= 0.023), as well as significant Time × Executive Control interactions (Deep: $\chi^2 = 4.0$, $p = 0.047$; Restorativeness: $\chi^2 = 5.1$, $p = 0.024$). These results indicate that the association between perceived sleep depth and restorativeness and executive control performance changed following stress induction. Specifically, higher Executive Control scores (reflecting poorer executive performance) were associated with a greater reduction in perceived sleep depth and restorativeness from pre- to post-stress in both groups.

Significant effects also emerged for the model assessing subjective sSE% in the short-term window as a function of Orienting performance. The GLMM revealed a significant main effect of Time ($\chi^2 = 5.1$, $p = 0.025$), a significant Time × Group interaction ($\chi^2 = 12.5$, $p < 0.001$), and a significant Time × Group × Orienting interaction ($\chi^2 = 5.1$, $p = 0.048$). This indicates that Orienting performance modulated the effect of stress on sSE% differently across groups. In GS, better orienting performance were associated with a more stable estimated sSE% after stress induction, whereas in IS, sSE% estimates improved as orienting performance worsened.

A representation of the models based on the estimated indices is shown in Figure 29 below.

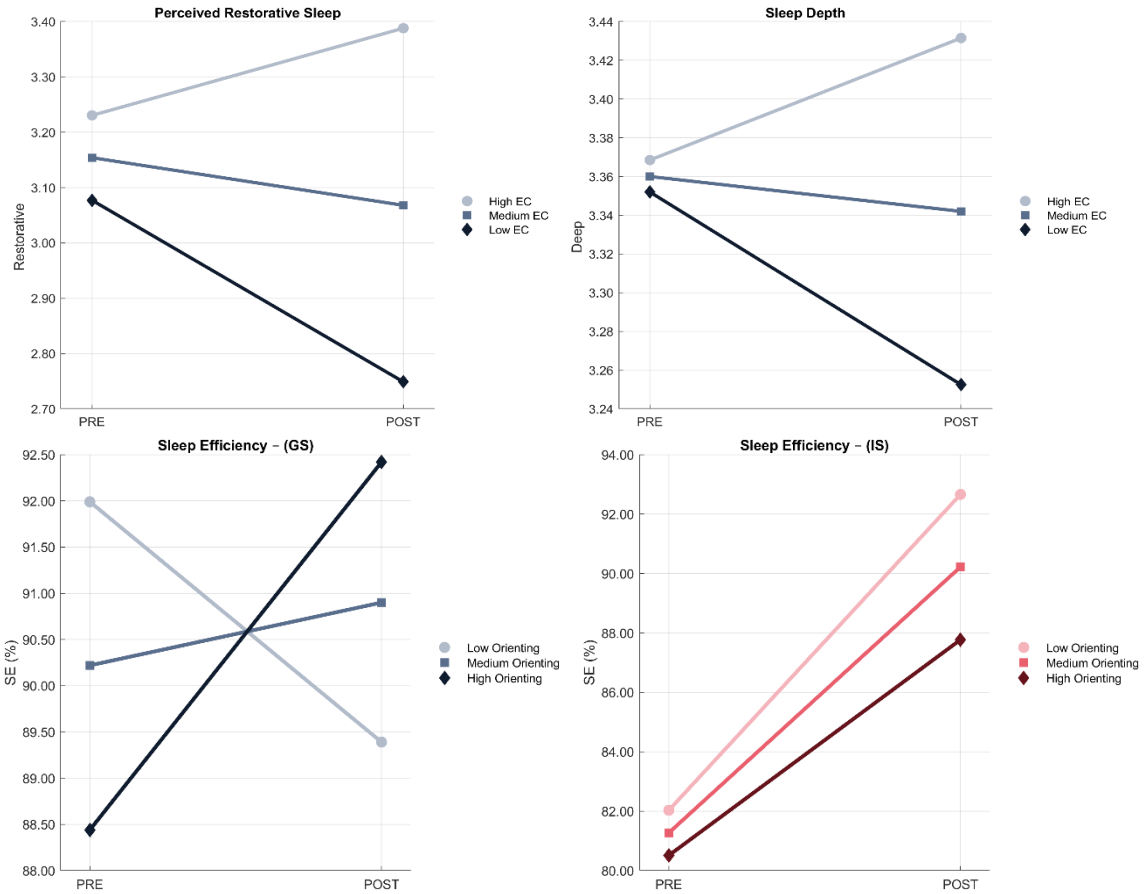

Figure 29. *A representation of the Estimated Marginal Means (EMMs) derived from the mixed-effects models (GLMMs) for the three subjective sleep indices (restorativeness, deep sleep, and SE%), with SE% displayed separately for the two experimental groups.*
For each variable, three levels of attentional performance are shown (Low, Medium, High), defined respectively as Low (−1 SD), Medium (mean), and High (+1 SD). The values represent the model's estimates at these specific levels and do not reflect the actual observed data trends. The PRE → POST lines illustrate how the model predicts changes in the outcome as a function of time across the three attentional performance levels, based on the relationships and effect sizes estimated in the GLMM. EC = Executive Control; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; SE% = Subjective Sleep Efficiency.

When considering objective sleep indices, a comparable pattern was found for SE%, mirroring the subjective results. The GLMM revealed significant main effects of Group ($\chi^2 = 9.8$, $p = 0.002$) and Time ($\chi^2 = 19.9$, $p < 0.001$), as well as a significant Group × Time interaction ($\chi^2 = 19.2$, $p < 0.001$), indicating that SE% changed differently across groups following stress induction. Moreover, a significant Group × Time × ANT Orienting interaction emerged ($\chi^2 = 11.1$, $p < 0.001$), showing that Orienting performance modulated the impact of stress on SE% in a group-dependent manner. In GS, better Orienting performance was associated with more stable SE% estimates after stress induction, whereas in IS, SE% improved as Orienting performance decreased.

Finally, the GLMMs involving Executive Control revealed group-specific modulation patterns for REM_A, with a significant Group × Time × Executive Control interaction ($\chi^2 = 5.9$, $p = 0.015$). In GS, better executive performance was associated with a reduced estimated number of arousals, whereas in IS, this relationship was reversed.

Figure 30 presents a depiction of the models derived from the estimated indices.

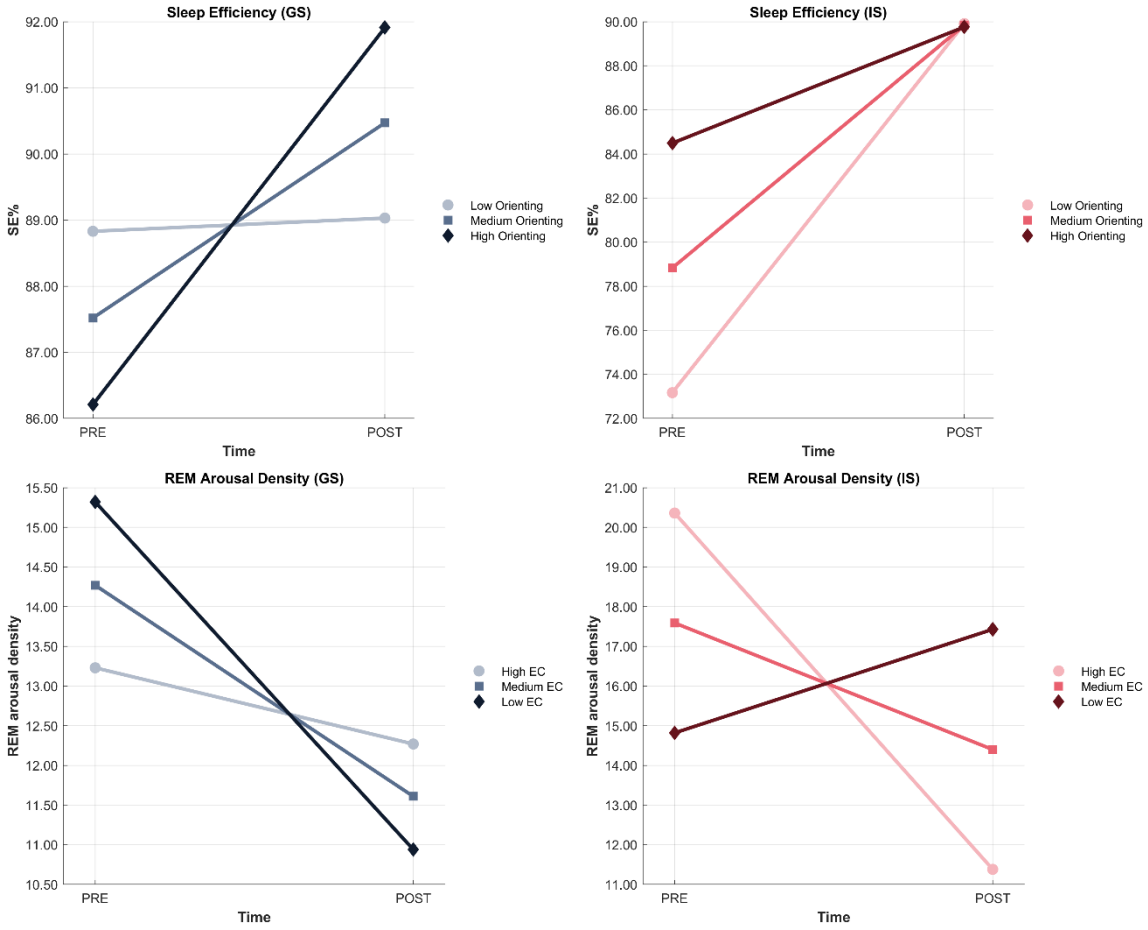

Figure 30. *A representation of the Estimated Marginal Means (EMMs) derived from the mixed-effects models (GLMMs) for objective SE% and REM arousal density, displayed separately for the two experimental groups.*
For each variable, three levels of attentional performance are shown (Low, Medium, High), defined respectively as Low (−1 SD), Medium (mean), and High (+1 SD). The values represent the model's estimates at these specific levels and do not reflect the actual observed data trends. The PRE → POST lines illustrate how the model predicts changes in the outcome as a function of time across the three attentional performance levels, based on the relationships and effect sizes estimated in the GLMM. EC = Executive Control; IS = Insomnia Symptoms Group; GS = Good Sleepers Group; SE% = Subjective Sleep Efficiency.

Lastly, we conducted correlational analyses exploring relationships among attentional performance, questionnaire scores, and intrusion variables and they did not reveal any significant associations except for an interesting positive correlation between FIRST score and Orienting score (r = 0.374, p = 0.017).

6.3. Discussion

With this experimental paradigm, we aimed to investigate the impact of a stressor (i.e. TFP) on both subjective and objective sleep indices and whether attentional abilities can modulate this effect in individuals with and without ID symptoms. We evaluated which of the three main attentional domains (i.e. Alerting, Orienting and Executive Control) might play a decisive role, and whether they would have a different impact in healthy subjects with and without ID symptoms.

As a first step, we validated a set of videos for the TFP in order to use one of these stimuli as a stressor with a certain degree of ecological plausibility. We measured sleep in a sample of 40 participants divided into two matched subgroups, before and after stress induction. Sleep quality was assessed over two weeks through a sleep diary to obtain subjective indices, and on the two days before and after the TFP using a portable EEG device, which provided objective sleep measures.

The attentional performance of these participants was assessed before the TFP through a test battery specifically designed to evaluate the performance of the three main components of attention.

6.3.1. How Stress Impact Differently on Subjective and Objective Sleep of Healthy Subjects with and without Insomnia Symptoms

The division obtained through the clinical cut-off of the SCI proved effective in identifying two subgroups with different sleep profiles. At baseline, in fact, the two groups differed on most of the questionnaires included to assess their sleep profile. Specifically, the IS subjects showed a significantly different pattern in terms of perceived sleep quality, measured through the ISI and the PSQI. Subjects with ID symptoms reported both physical and cognitive arousal, measured with the PSAS, that was significantly higher at baseline compared to controls, perfectly in line with the reference framework, the Hyperarousal Hypothesis (Dressle & Riemann, 2023). Finally, as expected, they also showed significantly higher scores on the FIRST, confirming the finding observed in the literature that individuals with ID exhibit high levels of SR compared to controls (Palagini et al., 2016).

The only questionnaire that did not significantly differ between the two groups was the ESS, indicating that GS and IS did not differ in terms of daytime sleepiness. For us, this is an encouraging finding because, due to the underlying hyperarousal state, several studies using the ESS or other objective indices, such as the MSLT, have shown the absence of sleepiness in ID (Arand et al., 2014; Fasiello et al., 2023). This allows us to hypothesize with greater confidence that the subjects classified as IS truly represent a preclinical sample of ID, rather than individuals with poor sleep hygiene or a circadian disorder, both of whom instead report daytime sleepiness (Hershner & Chervin, 2014; Steele et al., 2021).

Based on the assessment of the negative impact of stress, evaluated immediately through the FRQ and, over the following week, through the Intrusion Diary and the IES-R, the TFP appears effective in mimicking the effects of a stressful experience. Participants in fact provided ratings comparable to those reported by the subjects included in our previous video-validation study, describing the viewing experience as negative in terms of valence and high in terms of arousal, distress, and in particular disgust and embarrassment, two dimensions that proved especially important in determining the ability to induce a stressful experience in the previous experiment.

The number of reported intrusions was also comparable, whereas the IES-R score was considerably higher, although still below the clinical cut-off. The two groups did not differ statistically on any of these measures, indicating that the video produced a comparable stress impact in both groups. This allows us to assume that the differences we will soon present regarding the impact of the stressor on sleep represent a sleep-specific component of stress reactivity, rather than a general one, an aspect that is highly relevant and debated in the context of SR research. This is consistent with what was suggested by Jarrin and colleagues, who observed that baseline SR levels (measured with the FIRST) were able to predict the subsequent development of ID even when controlling for variables assessing mood symptoms and hyperarousal (Jarrin, 2014).

Focusing on our main outcomes, the exposure to TFP led to a deterioration in subjective sleep quality, and this effect was significantly more pronounced in the group with ID symptoms with both immediate impairments (short-term window) and more widespread disruptions over the following week (weekly window).

The significant time-by-group interactions emerging in both temporal windows involved two closely related indices, TST and SE%, with the pattern suggesting that individuals with ID symptoms exhibit greater vulnerability of their sleep system following exposure to a stressor. SE%, in particular, represents a key indicator of sleep quality, as it reflects the combined contribution of several phenomena (from sleep onset latency to the number and duration of nocturnal awakenings). This supports the hypothesis that high SR may constitute a crucial predisposing factor in the development of ID, confirming the literature identifying elevated SR as a risk factor for both the onset and maintenance of ID (Drake et al., 2004; Kalmbach et al., 2018). The fact that significant interactions emerged in both the short- and long-term windows indicates that the impact of pre-sleep stress may not be limited to immediate effects, but could extend into the subsequent nights. This raises the possibility that individuals with high SR may experience lingering or cumulative destabilization of the sleep–wake system.

In light of these findings, one might cautiously question whether the sleep system of individuals in the IS group is truly more sensitive to the effects of a stressor, or whether IS participants were simply responsive to the stimulus while GS participants were not. The post-hoc comparisons on the main effect of time allowed us to rule out this possibility. In both the short- and long-term windows, a significant deterioration was observed in SL, TST, and in SE%, and, in the short-term window, in WASO and perceived sleep depth. These results confirm that the TFP successfully induced subjective sleep disturbances across the entire sample. Thus, the differential impact revealed by the interaction effects cannot be attributed to the stressor being effective in one group but not in the other; rather, despite the stressor affecting both groups, their sleep systems differed in their resilience to its effects.

Finally, the post-hoc analyses on the main effect of group for the indices derived from the Sleep Diaries across the entire pre-manipulation and post-manipulation weeks revealed a pattern fully consistent with the fact that the two participant groups were correctly identified. Participants in the IS group, compared to those in the GS group, exhibited during the baseline week a significantly more impaired sleep profile for most of the indices considered: SE%, WASO, perceived depth, and restorativeness. In addition, SL, TST, as well as perceived quietness and NAWK showed worse average values in the IS group compared to GS, although these differences did not reach statistical significance,

while the number of nights in which dreams were reported remained stable. A similar, but even more pronounced pattern was observed in the post-manipulation week, with IS participants showing significantly worse scores than GS in terms of sTST, SE%, WASO, NAWK, perceived depth, and restorativeness. Also in this case, SL, sTST, and perceived quietness were poorer among IS participants, but without reaching statistical significance, and no differences emerged in the reported dream activity. These results provide a well-established picture in the literature of the subjective sleep characteristics of individuals with ID. Compared to GS, patients with ID show a generalized impairment of their sleep, including reduced SE%, shorter TST, and increased WASO (Baglioni et al., 2014).

The absence of an effect that involved SL was quite surprising as difficulties in falling asleep are one of the main complaints reported by individuals with ID (Ohayon, 2002). However, in a study by Buysse and colleagues (2010) examining night-to-night differences and variability in subjective sleep among ID patients compared to healthy controls, it emerged that, among all subjective indices, sleep latency (SL) shows the greatest intra-individual variability. Furthermore, SL does not display predictable patterns from one night to the next (Buysse et al., 2010). This large variability, combined with the pre-clinical nature of our sample, may explain why the trend observed, although consistent with the literature, remains below the threshold of statistical significance.

On the other hand, the absence of differences in dream activity is rather unexpected, as it is well established in the literature that individuals with ID tend to show higher dream recall frequency compared to good sleepers (Schredl et al., 1998). However, it should be noted that in the present study dream activity was assessed only with a single question addressing the presence of dream experiences during the night, without investigating the actual number of dreams recalled by participants. This methodological limitation may account for the lack of differences between the two groups.

When we restricted the analysis to the short-term window, no significant differences emerged between the groups at baseline, with the only exception of restorativeness. This absence of group differences in the two pre-manipulation nights is likely attributable to the limited temporal window considered and the high variability present, especially in the IS group, which reduces the reliability of indices that typically require several nights to stabilise and detect between-group distinctions. By contrast, in the two post-manipulation nights the groups differed significantly, with IS participants showing markedly lower

values in both calculated and subjective SE% as well as in both calculated and subjective TST, confirming the heightened vulnerability of their sleep system in response to the stressor.

The analysis of the objective PSG data revealed a complex picture, that is coherent with the hypothesis of greater vulnerability of the sleep–wake system in individuals with ID symptoms. First, the results showed particularly meaningful time-by-group interactions, indicating that the IS group displayed greater sleep impairment compared to the GS group. Specifically, the two significant effects indicate a worsening of sleep in terms of SE% and REM arousal density after the stressor, while two additional effects, TST and REM%, fell just above the significance threshold.

SE% showed an overall deterioration following exposure to the TFP in both groups, but this decrease was markedly more pronounced in the IS group. As previously mentioned, SE% represents one of the main indicators of sleep continuity. Thus, this suggests that the sleep system in individuals with ID is particularly sensitive to the effects of a stressor, mirroring what was observed in the subjective data and confirming a coherent pattern of heightened vulnerability, consistent with the construct of SR.

Even more interesting is the effect observed in REM arousal density. In the GS group, this index remained essentially stable, whereas in the IS group it increased significantly after exposure to the TFP. This result is particularly relevant given the strong body of evidence linking ID to a specific alteration of REM physiology. From an electrophysiological perspective, ID is associated with an increase in fast frequencies (beta and gamma) and a reduction in slow frequencies (delta) during REM (Dressle et al., 2023; Kang et al., 2022; Zhao et al., 2021), indicating a pattern of persistent cortical (hyper)activation that makes REM sleep less efficient in these individuals. In line with this, PSG studies show a marked increase in micro-arousals specifically during REM in patients with ID, a phenomenon directly linked to their perception of sleep quality, in terms of greater perceived wakefulness and poorer sleep continuity (Feige et al., 2008; Ren et al., 2023).

The most important consequence of this deterioration in REM sleep is a failure in the overnight downscaling of the emotional load of negative experiences. Wassing et al.

reported that while good sleepers show a clear attenuation of emotional reactivity after sleep, patients with ID show equal or even greater distress upon awakening (Wassing et al., 2016; Wassing et al., 2019a). This theory is further supported by the study of Galbiati and colleagues (2020), which demonstrated that REM physiology is closely associated with difficulties in emotion regulation: higher scores on the Difficulties in Emotion Regulation Scale (DERS) were correlated with a less efficient REM macrostructure (lower REM percentage, longer REM latency) and, most importantly, with a more unstable REM microstructure, characterized by increased REM arousal index and reduced REM density. In other words, greater difficulties in emotion regulation are systematically accompanied by more fragmented REM sleep.

In light of this, the selective increase in REM arousal density (together with the almost significant reduction of REM%) observed in the IS group after the TFP has strong theoretical grounding: a REM system that is already vulnerable and prone to fragmentation represents a critical point through which a stressor can interfere with nocturnal emotional regulation processes. Failure in the proper emotional processing of the stressor may in turn amplify the distress elicited by the stressor and therefore the associated arousal state, which would ultimately negatively impact sleep, thus concretizing the vicious cycle repeatedly described in the theoretical section of this dissertation.

In this case, post-hoc comparisons on the main effect (that is, the stress-induction intervention) were examined to ensure that the effects observed in the IS group after TFP, relative to the GS group, were not attributable to the GS group simply not being influenced by the video, especially considering that, in the validation experiment, we did not have the possibility to include an objective sleep measure. These analyses show that TFP produced a significant deterioration of sleep continuity and architecture in the entire sample, leading to a significant increase in WASO, NAWK, and REM arousal density, along with an increase in light sleep (N1%), as well as a significant reduction in TST, SE%, spindle density in N2, and the amount of N2% and REM%. The observed effects are consistent with a large body of literature documenting that acute stress robustly alters both sleep continuity and architecture, increasing nocturnal arousal and fragmentation. Indeed, the 2007 review by Kim and Dimsdale, which examined observational and experimental studies assessing the impact of stressors on objective sleep, reported that

experimentally induced stress was associated with reductions in slow-wave sleep and, similar to our findings, reductions in REM sleep, worsened SE%, and increased NAWK (Kim & Dimsdale, 2007). Moreover, the greater instability of REM sleep and the increase in micro-arousals observed after TFP reflect what has been reported in studies showing that stress impacts the quality and stability of REM sleep (Germain & Nielsen, 2003).

These results indicate that in the two nights immediately following stress induction, the video was effective in disrupting the sleep quality of the entire sample, at both the macro- and microstructural levels. They therefore strengthen the conclusion that the group differences highlighted by the group × time interaction reflect a different degree of resilience of the sleep system, rather than a different susceptibility to the stimulus in the two groups.

Finally, analyses of the main group effect produced findings consistent with the subjective data. The two groups did not differ significantly during the two baseline nights, with the exception of REM arousal density. This result may suggest that, having already a higher number of REM arousals under neutral conditions, the IS group exhibited a particular vulnerability to the subsequent introduction of emotional stimuli, such as the stress-inducing video. The fact that no other differences emerged may be due to the fact that, exactly as seen in the subjective data, participants in the IS group exhibit sleep characterized by marked instability, typical of ID (Buysse, 2010), and that two days are therefore not sufficient, in a pre-clinical group, to detect differences from the control group. Such differences might instead emerge when considering a wider temporal window, as is the case for subjective indices when examining comparisons across the whole pre-week.

In the two nights post TFP, however, the differences between the two groups are substantial: the IS group shows a worse sleep profile than the GS group in terms of SE%, TST, REM%, WASO, SL, and REM arousal density, reflecting a global deterioration of objective sleep that affects both sleep continuity and the quality of sleep architecture.

In summary, our findings show that individuals with ID symptoms exhibit a markedly reduced resilience of the sleep–wake system when exposed to stress, reflected in both subjective and objective disruptions of sleep continuity and architecture. These results support the view that heightened SR represents a core vulnerability factor in ID, already detectable at a preclinical level.

6.3.2. How Attention Shapes Sleep Response to Stress

The main objective of our work was not only to investigate how the same stressor affects the sleep system differently in individuals with ID symptoms (pre-clinical stage) compared to good sleepers, but also to explore more specifically the role of the attentional domain in modulating the impact of the stressor in these two subgroups. The results obtained indicate that the role of attention in modulating the sleep response to stress is more complex than what strictly behavioural models would suggest.

First, the absence of differences in attentional tasks between GS and IS indicates that ID, at least in its pre-clinical phase, is not associated with objective or generalized deficits in attentional abilities. This finding aligns with the literature showing that attentional alterations in ID do not necessarily manifest as poorer performance on "cold" cognitive tasks, but rather as a dysfunctional use of cognitive resources, characterized by hypermonitoring, difficulties in disengaging, and inflexible attentional control (Harvey, 2002; Espie et al., 2006). However, our study differs from the classical literature based on Espie's model, since our investigation focused less on sleep-specific attentional mechanisms and more on general attentional functioning. We hypothesize that ID is not driven exclusively by hyperattention or hypermonitoring directed at sleep, but may instead reflect a broader alteration in attentional functioning.

The picture becomes clearer when attentional performance is analysed not as a stable trait but as a dynamic modulator of the sleep response to stress, an approach made possible by the use of GLMMs. In this perspective, robust and theoretically meaningful effects emerge for the ANT task, specifically for the Executive Control and Orienting components.

With regard to the subjective indices obtained in the short-term window, the two perceived sleep quality measures (depth and restorativeness), which had previously been influenced by group, stressor, and their interaction, both showed a significant time by executive control interaction. Since higher Executive Control scores reflect poorer performance, the estimated models indicate that participants with less efficient executive functioning experience a greater deterioration in perceived sleep quality after stress induction, independently of group membership. Although these are estimated effects, they suggest that poor executive functioning may represent a vulnerability factor for sleep satisfaction in both pre-clinical ID individuals and healthy subjects. This result fits well

within the theoretical framework proposed by Wells in the CAS hypothesis, where reduced executive efficiency may reflect difficulty interrupting monitoring and disengaging from intrusive content, which in this case could intuitively relate to the stress induction (Wells, 1995; Wells and Matthews, 1996). The involvement of two satisfaction-related indices supports this interpretation, since difficulty disengaging from negative thoughts and images during the pre-sleep phase could influence the individual's entire perception of the night.

Even more interesting are the results concerning Orienting performance. A significant time by group by orienting interaction emerged for subjective SE% in the short-term window. In GS participants, better Orienting performance was protective, being associated with more stable subjective sleep efficiency estimates. Orienting also significantly modulated the effect of stress on both subjective and objective sleep efficiency. In IS participants, however, the opposite pattern was observed: better Orienting performance corresponded to worse post-stress estimates, whereas poorer performance corresponded to better estimates. The same pattern was found for objective SE%, indicating a robust interaction between Orienting and both subjective and objective sleep quality.

In GS, this relationship suggests that a greater ability to shift attention rapidly toward relevant stimuli and disengage from irrelevant ones may support more effective downregulation of pre-sleep arousal, thereby promoting a more adaptive response to stress. In contrast, the paradoxical pattern observed in IS participants suggests that high Orienting performance may not be used adaptively but instead it seems to amplify attention toward internal states, undesirable content, and arousal sensations. This intensifies reactivity to the stressor and fits well within the CAS framework. Supporting this interpretation is the positive correlation between SR (FIRST scores), which were significantly higher in IS, and orienting performance. Individuals who are more reactive to sleep-related stress appear to be characterized by an attentional style oriented toward vigilance and monitoring. These individuals perform well in orienting and maintaining attention, but unlike GS participants, appear to direct their attention toward negative rather than neutral stimuli, which can hinder normal sleep onset, particularly following negative or stressful events.

An alternative explanation, also consistent with previous literature, is that good or relatively preserved attentional performance can coexist with compromised sleep quality. In patients with ID, Liu and colleagues reported that the absence of marked cognitive decline, despite sleep disturbance, may reflect a compensatory function of hyperarousal on cognitive performance (Liu et al., 2014). In other contexts, better sustained attention performance has been associated with poorer sleep quality, as in children with higher tablet use (Chiu et al., 2022). Although this finding aligns with our hypotheses, it should be interpreted cautiously, given the differences in population and context.

The analysis further revealed that REM arousal density was significantly affected by a three-way interaction involving time, group, and executive control. In GS, better executive abilities were associated with fewer estimated REM micro-arousals after stress, suggesting efficient regulatory processes. In IS, however, the opposite pattern was observed: individuals with better executive control appeared more susceptible to increased REM fragmentation after stress. This finding recalls research on REM sleep as a privileged window for emotional downscaling. In individuals vulnerable to stress, better executive control may reflect an active but ineffective attempt for cognitive regulation, interfering with natural offline emotional processing and promoting microstructural instability.

The inconsistency between the role of executive control in subjective sleep satisfaction and in REM arousal density may reflect a dissociation between perceived sleep quality and microstructural sleep regulation. Successfully redirecting attention away from negative content may improve the subjective experience of falling asleep, without guaranteeing adequate physiological regulation. While attentional disengagement reduces monitoring and rumination during pre-sleep, thereby improving subjective perception, it may also reduce opportunities for natural emotional processing, which in vulnerable individuals relies on cognitive and affective reprocessing. As a result, part of the emotional load associated with the stressor remains unresolved and is instead processed during REM sleep, where increased residual activation manifests as higher arousal density.

Taken together, these results suggest something important and partly unexpected for therapeutic interventions targeting ID. Treatments should not be limited to reducing behavioural hyperarousal or correcting dysfunctional beliefs. They should also include

components specifically aimed at modulating attentional processes and executive control. The dissociation observed between subjective satisfaction and microstructural sleep stability indicates that good attentional disengagement may improve the conscious experience of falling asleep, but is not sufficient, in vulnerable individuals, to support full physiological emotional regulation. In fact, most findings suggest that enhancing attentional capacities, particularly in their more basic domains, may paradoxically be harmful for individuals with ID or those vulnerable to it.

This suggests that it is not the amount of attention or cognitive control that determines adaptive outcomes, but rather the way in which these resources are used. In individuals vulnerable to sleep-related stress, more efficient attentional capacities may be deployed rigidly, in a hypercontrolling manner, or directed toward negative content, thereby contributing to the maintenance of the CAS rather than interrupting it. From this perspective, therapeutic interventions should not aim to strengthen attention in its basic components, but instead promote a more flexible and less perseverative use of cognitive resources.

Approaches incorporating metacognitive components, such as reducing internal monitoring, training attentional disengagement, promoting a more detached relationship with pre-sleep thoughts and sensations, or strategies that limit excessive voluntary regulation, may be particularly effective. These strategies closely reflect the principles of Wells's Metacognitive Therapy (Wells, 2009). Such interventions act not only on mental contents but also on the way attention and executive control are recruited, preventing these functions from being used in a hypercompensatory manner that interferes with the natural emotional downscaling processes occurring during sleep.

6.3.3. Limits and Future Directions

The study presents several limitations, some of them due to our limited resources, that future research could address.

One of the main limitations is certainly related to the sampling strategy and sample size: the sample of 40 participants was obtained through a power analysis based on previous studies and on the primary outcome of investigating the differential impact of stress on sleep in the two subsamples analysed. However, the conclusions that can be drawn regarding the more complex analytical models involved are limited by the modest

number of participants included in the two subgroups. This may also have contributed to the lack of emergence of effects that were indeed present but not statistically significant due to the small sample size.

It should also be noted that the sample consisted mostly of university students or people connected to them (as recruitment flyers were distributed almost exclusively within university facilities), or socially related individuals who learned about the study through word of mouth. This resulted in a sample largely drawn from a medium–high socio-cultural background and composed, to a significant extent, of individuals with a (professional or personal) interest in psychology or sleep medicine.

In the future, it would therefore be interesting to replicate the protocol with a larger and more various sample, both in terms of age and socio-cultural background, to draw more robust conclusions from more complex analytical models.

Additionally, we were primarily interested in assessing the pre-clinical phase of the disorder, as the etiopathogenetic mechanisms of ID were of particular interest to us. However, it would also be valuable for future studies to examine the consequences of experimentally inducing stress in individuals with a clinical diagnosis of chronic ID.

Finally, our sample was selected to exclude individuals with psychiatric comorbidities, particular vulnerability to trauma due to past experiences, and sleep profiles that were not borderline between the presence of symptoms and good sleep quality. While this allowed for greater experimental control and minimised potential confounding factors, it also reduced ecological validity, for example because, as highlighted several times in the introduction, psychiatric comorbidities are frequently present in ID. It therefore becomes relevant to ask whether, in this subgroup of individuals, one might expect a different sleep response to trauma or a different role of attentional performance compared to individuals without comorbidities. Moreover, ID has been described as a disorder with a fluctuating course, which could be reflected in intermediate SCI scores; alternatively, these participants might represent a healthy subgroup with poor sleep hygiene or another form of sleep disturbance. It would also be valuable to investigate whether the induced stressor has a different impact, and to what extent, on individuals with a history of similar past trauma, given that a well-known finding in trauma literature is that previous exposure to a traumatic event is a risk factor for experiencing subsequent trauma, making these individuals particularly vulnerable (Breslau et al., 2008).

The use of a portable device such as the SP introduced the most significant limitations of this study. While it offered ecological advantages, it also presented several methodological drawbacks. First, a large portion of the sample, regardless of group, reported some discomfort caused by the device. This is likely the most significant limitation, as it raises questions about the extent to which device-related discomfort may have affected participants' sleep, particularly in the group with symptoms. Indeed, a recent study by Pieroni and colleagues showed that ID symptoms and the level of SR (both measured through self-report questionnaires) are correlated with a phenomenon known as sensory-processing sensitivity, which describes a heightened sensitivity to internal and external stimuli (Pieroni et al., 2014), which may have made participants in the IS group more susceptible to being disturbed by the device. Second, although the device is a validated EEG tool, a PSG setup would have allowed for a greater number of measures of sleep microstructure, specifically those related to topographical evaluations. In the future, it would be highly valuable to replicate the protocol using a full PSG setup.

Regarding the attentional test battery, we believe that the chosen Go/No-Go task likely resulted in a ceiling effect. This also meant that the PES index, potentially yielding interesting results, was available only for a small subsample of 11 participants, which is insufficient for drawing meaningful conclusions. In future work, it may therefore be useful to employ a more challenging attentional test battery that, given the domains identified as most relevant, can also assess them in more depth.

In this context an additional limitation concerns the correlational nature of attentional measures. Although stress exposure was experimentally manipulated, attentional functioning was assessed observationally and not experimentally manipulated. It is worth noting that no significant group differences were observed at baseline in attentional performance or sleep measures, suggesting that the observed differences in stress-related sleep responses did not simply reflect pre-existing group differences.

Nevertheless, the present design does not allow definitive conclusions regarding the temporal or causal direction of the association between attentional functioning and sleep reactivity. Attentional differences may become functionally relevant only under conditions of stress, may reflect subtle vulnerabilities not detectable at baseline, or may interact with sleep processes through bidirectional pathways over time. Longitudinal designs and experimental manipulations of attention will be necessary to further clarify

these mechanisms and to strengthen causal and translational interpretations. It may also be valuable for future research to examine the impact of enhancing such attentional abilities on sleep in both the pre-clinical and chronic phases of ID, in order to evaluate the feasibility of including targeted cognitive-attention training techniques in treatment and prevention protocols for ID.

Another aspect that we were not able to investigate in depth, but which would certainly be of great interest, is dreaming. We chose not to include a Dream Diary in the protocol, as the experiment was already highly complex for the volunteers; however, future studies could benefit from exploring this dimension. In light of the well-established central role of dreaming in the processing of emotional memories, it would be useful to examine how the stressor impacts dreams (in terms of both frequency and characteristics) in the two subgroups, and whether this effect might also be modulated by attentional performance.

6.3.4. Conclusion

The present thesis had two main objectives. The first was to experimentally evaluate how a stressful event it is able to influence subjective and objective sleep in a sample of healthy adults, differentiated on the basis of the presence or absence of ID symptoms, thereby investigating the role of SR as a possible early vulnerability factor in the development of the disorder. The second objective aimed to explore whether and how attentional abilities might modulate the sleep system's response to stress, and whether such modulation would take on a different meaning in the two subgroups.

The results collected showed that the TFP impact negatively on participant's sleep across the entire sample, confirming the effectiveness of the stimulus. However, it emerged quite strongly that IS exhibited greater vulnerability compared to GS, both in terms of sleep quality. The overall picture fits coherently within SR theory, according to which some individuals possess an intrinsically more sensitive sleep–wake system that is more prone to destabilisation in response to stressful events. It is relevant that such differences emerge in a preclinical sample, suggesting that this vulnerability is already fully operative before the onset of clinical ID.

The analysis of the attentional functions further enriches this interpretation by providing a meaningful account of individual differences and of the mechanisms underlying the trait-like nature of SR. Executive control and orienting performance

proved capable of modulating the impact of stress on sleep, but in different ways across the two subgroups. In GS, better attentional functioning appeared to constitute a protective factor, being associated with greater sleep stability after the stress induction. In IS, by contrast, the same attentional efficiency seemed to be associated with a more dysfunctional response, amplifying the impact of stress on sleep quality both subjectively and objectively. And it is meaningful that REM microstructure is involved, considering the role of REM sleep in emotional processing. This involvement may help explain how the vicious cycle that leads to symptom chronicisation unfolds: a stressful event occurs; the individual, characterised by high SR, shows attentional performances that are adaptive during wakefulness but become maladaptive in the context of sleep. These patterns induce hyperarousal, which in turn disrupts sleep, particularly by impairing REM sleep efficiency. This, in turn, disrupts the processing of the stressful event and leaves its emotional load unresolved. Together with the individual's attentional strategies, this unresolved emotional charge then gives rise to a renewed state of arousal that further disrupts sleep, and so on. The asymmetry, indeed, suggests that in individuals vulnerable to sleep-related stress, attentional resources may be employed in a rigid, over-controlling manner, contributing to the maintenance of internal monitoring and rumination processes typical of Wells' CAS. From this perspective, attentional efficiency would not constitute a protective factor per se, but could instead fuel cognitive processes that hinder the natural course of sleep by increasing the individual's arousal state.

The value of these findings must also be interpreted in light of two innovative aspects of the present work. This study represents one of the few attempts to investigate in a controlled manner the relationship between stress and sleep using an experimental paradigm that includes both subjective and objective measures, recorded over several consecutive nights directly in the participants' home environment. The employment of a portable and self-applicable PSG made it possible to obtain EEG data, including microstructural indices, without sacrificing ecological validity. Moreover, the stressor was not selected from an existing set, but was instead preceded by a validation experiment through which we also assessed its ability to affect subjective sleep, considerably strengthening the paradigm's robustness. The second element of originality lies in the fact that this is the first study to test the role of attention not in the components typically investigated in the context of ID, such as bodily monitoring or selective attention toward

sleep-related content, but as a neuropsychological domain. This approach made it possible to reveal a pattern of sleep modulation by attention that is more complex than what we hypothesised and potentially relevant for understanding the cognitive mechanisms that contribute to sleep vulnerability to stress.

Taken together, these results hold clinical relevance. In a historical period in which stress is a constant component of daily life, understanding why some individuals develop ID after a stressful event while others remain resilient is crucial. From a metacognitive perspective, the results suggest that merely reducing arousal or correcting dysfunctional beliefs may not be sufficient to effectively address ID symptoms: it may be essential to promote a more flexible, less perseverative, and less controlling use of cognitive resources. Studying these early phases, thus helps clarifying the mechanisms underlying a highly impairing and widespread disorder such as ID and contributes to outlining new directions for both prevention and the development of more targeted and potentially more effective therapeutic interventions.

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Appendix A - Diaries

Sleep Diary: English version

The diary must be completed every morning upon waking, within 10 minutes, for 14 consecutive days. It is essential that this is done soon after waking because memories and impressions related to sleep tend to fade very quickly.

IMPORTANT NOTE: When answering, you must refer only to your own subjective sensations and memories of the night just passed. Specifically:

Do not consult others to obtain information about your sleep (e.g., a bed partner or roommate).

Do not refer to a clock (or any device showing the time) in order to be more precise; it is important that you rely on your own estimation skills only.

You must answer all questions and remember to submit the form by pressing "Submit survey".

1. *Subject identification code:* _____
2. *Date of complention:* dd/mm/yyyy
3. *What time did you go to bed last night (this refers to the moment when you physically lay down)?* hh:mm
4. *What time did you stop all activities (e.g. reading, using your phone, watching Television, etc.) while you were already in bed and closed your eyes to try to fall asleep?* hh:mm
5. *How many minutes later did you fall asleep?* _____ minutes
6. *Did you spend time half-asleep before finally falling asleep?*

   ☐ Yes ☐ No
7. *How hard was it to fall asleep?*

   1 2 3 4 5 6

   Very easy Very difficult
8. *How many times did you wake up during the night?* _____
9. *If you woke up during the night, how many minutes did you stay awake in total?*

   _____ minutes

10. *How difficult was it to go back to sleep after waking up?*

1	2	3	4	5	6
Very easy					Very difficult

11. *Did you spend time half-asleep at night?*

☐ Yes ☐ No

12. *What time did you definitely wake up?* hh:mm

13. *How many hours did you sleep in total?* _____ hours

14. *After your final awakening, how many minutes did it take you to get out of bed this morning?* _____ minutes

15. During the night did you have any kind of experience, sensation, dream?

- ☐ *Sensation*
- ☐ *Thought*
- ☐ *Dream*
- ☐ *Nothing*
- ☐ *Other:* _____

16. *Evaluate what your sleep was like overall:*

1	2	3	4	5	6
Very light					Very deep

17. *Evaluate what your sleep was like overall:*

1	2	3	4	5	6
Not very restful					Very restful

18. *Evaluate what your sleep was like overall:*

1	2	3	4	5	6
Very calm					Very agitated

19. *How did you wake up this morning?*

- ☐ Alarm Clock
- ☐ Spontaneously
- ☐ Other: _____

20. *Compilation end time (you must use a watch):* hh:mm

Intrusion Diary: English version

An intrusion is a thought or mental image that arises in a pervasive and involuntary way, is associated with a negative experience, and often causes a certain level of distress. This form is intended to track any intrusions that may occur in the days following the viewing of the videos. Participants are asked to complete this form each time they experience an intrusion during the week after watching the video.

1. *Subject identification code*: _____
2. *Date of complention*: dd/mm/yyyy
3. *Time of complention*: hh:mm
4. *Have you experienced one or more intrusions since the last alarm received?*

   ☐ Yes ☐ No

   If the answer is yes, please answer the following questions.

   If you have had more than one intrusion, complete a separate form for each, keeping the first three responses identical.

5. *Briefly describe the intrusion (indicate the context, what you saw/thought, how it manifested, its duration, and sensations felt):*

   _____

   _____

   _____

   _____

   _____

6. *Specify in what form the intrusion occured:*
   - Thoughts
   - Images
   - Thoughts and images
7. *Rate the vividness of the intrusion:*

   1 2 3 4 5 6 7

   Not vivid at all Extremely vivid

8. *Rate the level of discomfort caused by the intrusion:*

1 2 3 4 5 6 7

Not uncomfortable at all Extremely uncomfortable

9. *Rate how much you felt in control of the intrusion:*

1 2 3 4 5 6 7

Not under control at all Completely under control

10. *Rate how spontaneous the intrusion felt*:

1 2 3 4 5 6 7

Not spontaneous at all Completely spontaneous

11. *Which video is this intrusion related to:* _____

Appendix B - Video Validation Questionnaires

Mood Questionnaire: English version

Please indicate on the scales below the score that best represents how you feel right now.

1. *How anxious/stressed do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

2. *How happy do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

3. *How afraid do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

4. *How much do I feel in control right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

5. *How helpless do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

6. *How sad do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

7. *How angry do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

8. *How content do I feel right now?*

1	2	3	4	5	6	7	8	9
Not at all								Extremely

Film Response Questionnaire: English version

Please indicate how you felt while watching the video by selecting the number that best represents your experience on each scale.

1. *How would you rate your overall experience of watching the video?*

   -4 -3 -2 -1 0 1 2 3 4

   Very unpleasant Very pleasant

2. *To what extent did you feel physiologically activated during the video (e.g., sweating, feeling warm, increased heart rate)?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

3. *To what extent did you feel paralyzed or unable to move while watching the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

4. *To what extent did you feel anxious or stressed during the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

5. *To what extent did you feel disgusted during the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

6. *To what extent did you feel embarrassed during the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

7. *To what extent did you feel emotionally involved while watching the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

8. *To what extent did you pay attention during the video?*

   1 2 3 4 5 6 7 8 9

   Not at all Extremely

9. *Did you look away from the screen at any point during the video?*

   ☐ Yes ☐ No

10. *Had you already seen the video or parts of it before?*

☐ Yes ☐ No

Appendix C - Tables of Experiment 1

Theme		Valence (M ± SD)	Arousal (M ± SD)	Immobility (M ± SD)	Distress (M ± SD)	Disgust (M ± SD)	Embarrassment (M ± SD)	Involvement (M ± SD)
Physical	PT1[a]	-3.5 ± 0.7	5.9 ± 2.2	4.4 ± 2.8	5.6 ± 2.4	7.7 ± 1.7	2.8 ± 2.0	5.3 ± 2.4
	PT2[b]	-3.0 ± 1.3	4.8 ± 2.2	3.3 ± 2.3	4.8 ± 2.2	6.1 ± 2.3	2.1 ± 2.0	4.9 ± 2.4
	PN[c]	1.0 ± 1.4	2.1 ± 1.4	1.4 ± 1.0	1.3 ± 0.8	1.1 ± 0.4	1.1 ± 0.4	3.1 ± 1.9
	PP[d]	2.0 ± 1.8	2.6 ± 2.0	1.1 ± 0.3	1.2 ± 0.6	1.5 ± 1.2	1.7 ± 1.5	4.2 ± 2.4
	Fr	103.4**	80.7**	68.1**	97.0**	110.5**	33.7**	24.7**
		a, b > c, d	a, b > c, d	a, b > c, d	a, b > c, d	a, b > c, d	a > c, d; b > c	a, b > c; d > c
Relational	RT1[a]	-3.5 ± 0.7	4.9 ± 2.4	4.3 ± 2.5	5.4 ± 2.5	7.7 ± 1.6	4.5 ± 2.7	5.3 ± 2.3
	RT2[b]	-3.6 ± 0.8	5.6 ± 2.1	4.1 ± 2.6	5.9 ± 2.1	7.8 ± 1.7	4.1 ± 2.7	5.4 ± 2.3
	RN[c]	1.4 ± 1.3	2.3 ± 1.7	1.3 ± 0.6	1.3 ± 1.0	1.1 ± 0.3	1.4 ± 1.0	3.5 ± 2.4
	RP[d]	1.1 ± 1.8	3.8 ± 2.2	1.4 ± 0.7	1.7 ± 1.2	1.3 ± 0.9	2.2 ± 1.9	3.5 ± 2.1
	Fr	102.6**	54.2**	80.6**	92.2**	111.0**	58.8**	27.2**
		a, b > c, d	a, b > c, d; d > c	a, b > c, d	a, b > c, d	a, b > c, d	a, b > c, d	a, b > c, d
Traffic	TT1[a]	-2.9 ± 1.4	4.6 ± 2.2	3.2 ± 2.1	4.9 ± 2.3	3.2 ± 2.4	1.6 ± 1.1	5.1 ± 2.1
	TT2[b]	-2.6 ± 1.2	4.8 ± 2.1	3.2 ± 2.2	4.9 ± 2.1	1.6 ± 1.1	1.5 ± 1.2	5.9 ± 2.1
	TN[c]	0.0 ± 0.9	1.6 ± 1.1	1.2 ± 0.5	1.3 ± 0.7	1.1 ± 0.2	1.1 ± 0.3	2.1 ± 1.7
	TP[d]	1.3 ± 1.9	2.7 ± 1.9	2.0 ± 1.7	2.2 ± 1.6	1.4 ± 1.0	1.5 ± 1.2	3.6 ± 2.0
	Fr	97.3**	74.8**	56.13**	80.3**	53.6**	12.8*	70.4**
		a, b > c, d	a, b > c, d; d > c	a, b > c, d	a, b > c, d	a > c, d	b > c	a, b > c, d; d > c

Table 8. *Comparison of Film Response Questionnaire scores between video categories within each theme from Friedman's Test. Valence scale goes from -4 (very unpleasant) to +4 (very pleasant).*
M = mean; SD = standard deviation; Fr = Friedman's chi-square statistic; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** $p < 0.001$.*

Theme		Valence (M ± SD)	Arousal (M ± SD)	Immobility (M ± SD)	Distress (M ± SD)	Disgust (M ± SD)	Embarrassment (M ± SD)	Involvement (M ± SD)
Physical	PT1[a]	-3.5 ± 0.7	5.9 ± 2.2	4.4 ± 2.8	5.6 ± 2.4	7.7 ± 1.7	2.8 ± 2.0	5.3 ± 2.4
	PT2[b]	-3.0 ± 1.3	4.8 ± 2.2	3.3 ± 2.3	4.8 ± 2.2	6.1 ± 2.3	2.1 ± 2.0	4.9 ± 2.4
Relational	RT1[c]	-3.5 ± 0.7	4.9 ± 2.4	4.3 ± 2.5	5.4 ± 2.5	7.7 ± 1.6	4.5 ± 2.7	5.3 ± 2.3
	RT2[d]	-3.6 ± 0.8	5.6 ± 2.1	4.1 ± 2.6	5.9 ± 2.1	7.8 ± 1.7	4.1 ± 2.7	5.4 ± 2.3
Traffic	TT1[e]	-2.9 ± 1.4	4.6 ± 2.2	3.2 ± 2.1	4.9 ± 2.3	3.2 ± 2.4	1.6 ± 1.1	5.1 ± 2.1
	TT2[f]	-2.6 ± 1.2	4.8 ± 2.1	3.2 ± 2.2	4.9 ± 2.1	1.6 ± 1.1	1.5 ± 1.2	5.9 ± 2.1
	F	45.4**	32.9**	22.9**	20.4**	158.2**	92.1**	13.0*
		a, c, d > b, e, f ; d > a, c	a > b, c, e, f; d > b	a, c > b, f	d > b, e, f	d > a, b, e, f; c > a, b, e, f; a, b > e, f	c, d > a, b, e, f a, b > e, f	f > b

Table 9. *Comparison of Film Response Questionnaire scores between traumatic videos from Friedman's Test. Valence scale goes from -4 (very unpleasant) to +4 (very pleasant).*
M = mean; SD = standard deviation; Fr = Friedman's chi-square statistic; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video).
** p < 0.05; ** p < 0.001.*

Theme		HR (bpm) (M ± SD)		HR (bpm) (M ± SD)
Physical	PT1[a]	72.8 ± 3.1	PT1[e]	72.8 ± 3.1
	PT2[b]	74.1 ± 3.2	PT2[f]	74.1 ± 3.2
	PN[c]	78.6 ± 3.1		
	PP[d]	78.8 ± 4.4		
	F	44.7**		
		a, b < c, d		
Relational	RT1[a]	70.9 ± 2.8	RT1[g]	70.9 ± 2.8
	RT2[b]	72.5 ± 2.9	RT2[h]	72.5 ± 2.9
	RN[c]	78.5 ± 2.9		
	RP[d]	79.0 ± 4.7		
	F	102.7**		
		a, b < c, d; a < b		
Traffic	TT1[a]	75.3 ± 3.7	TT1[i]	75.3 ± 3.7
	TT2[b]	75.8 ± 3.5	TT2[l]	75.8 ± 3.5
	TN[c]	78.7 ± 2.1		
	TP[d]	79.1 ± 5.8		
	F	17.3**	*F*	16.0**
		a, b < c, d		g < e, f, h, i, l; h, e,f < i, l

Table 10. *Comparison of heart rate (HR) between video categories within each theme (rmANOVA results, on the left) and comparison on HR between traumatic videos (rmANOVA results, on the right).*
HR = heart rate; bpm = beats per minute; M = mean; SD = standard deviation; F = Fisher's F ratio; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2

= Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** p < 0.001.*

Theme		CDA.AmpSum (µS) (M ± SD)	CDA.SCR (µS) (M ± SD)	CDA.ISCR (µS) (M ± SD)	CDA.Tonic (µS) (M ± SD)
Physical	PT1[a]	0.54 ± 0.32	0.12 ± 0.05	3.56 ± 1.54	4.71 ± 2.31
	PT2[b]	0.45 ± 0.25	0.10 ± 0.05	2.95 ± 1.29	4.89 ± 2.11
	PN[c]	0.33 ± 0.19	0.08 ± 0.03	2.04 ± 0.90	4.64 ± 2.01
	PP[d]	0.34 ± 0.02	0.07 ± 0.03	2.02 ± 0.84	4.65 ± 2.20
	F	77.9**	96.85**	133.5**	2.4
		a, b > c, d	a, b > c, d	a, b > c, d	
Relational	RT1[a]	0.56 ± 0.30	0.13 ± 0.05	3.56 ± 1.54	5.41 ± 1.99
	RT2[b]	0.53 ± 0.27	0.11 ± 0.04	2.97 ± 1.28	5.34 ± 2.30
	RN[c]	0.36 ± 0.20	0.08 ± 0.03	2.03 ± 0.84	5.09 ± 1.91
	RP[d]	0.33 ± 0.17	0.08 ± 0.03	2.04 ± 1.83	5.35 ± 2.18
	F	104.3**	89.3*	134.9**	1.1
		a, b > c, d	A > c, d	a, b > c, d	
Traffic	TT1[a]	0.42 ± 0.24	0.09 ± 0.04	2.69 ± 1.14	5.18 ± 2.47
	TT2[b]	0.34 ± 0.20	0.08 ± 0.03	2.01 ± 0.84	4.97 ± 2.13
	TN[c]	0.33 ± 0.19	0.08 ± 0.04	2.01 ± 0.87	4.94 ± 2.05
	TP[d]	0.26 ± 0.15	0.06 ± 0.07	1.51 ± 0.70	5.20 ± 2.77
	F	14.3*	47.4	28.1*	1.0
		a > c, d; b > d; c > d		a > c, d; b > d	

***Table 11**. Comparison of Continuous Decomposition Analysis (CDA) parameters (CDA.AmpSum, CDA.SCR, CDA.ISCR, and CDA.Tonic) between video categories within each theme (rmANOVA results). Data from 37 participants.*
µS = microsiemens; CDA = Conductance Data Analysis; AmpSum = amplitude sum of SCRs; SCR = Skin Conductance Response; ISCR = Integrated Skin Conductance Response; Tonic = tonic skin conductance level; M = mean; SD = standard deviation; F = Fisher's F ratio; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); PN = Physical Neutral Video; PP = Physical Positive Video; RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); RN = Relational Neutral Video; RP = Relational Positive Video; TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video); TN = Traffic Neutral Video; TP = Traffic Positive Video.
*** p < 0.05; ** p < 0.001.*

Theme		CDA.AmpSum (µS) (M ± SD)	CDA.SCR (µS) (M ± SD)	CDA.ISCR (µS) (M ± SD)	CDA.Tonic (µS) (M ± SD)
Physical	PT1[a]	0.54 ± 0.32	0.12 ± 0.05	3.56 ± 1.54	4.71 ± 2.31
	PT2[b]	0.45 ± 0.25	0.10 ± 0.05	2.95 ± 1.29	4.89 ± 2.11
Relational	RT1[c]	0.56 ± 0.30	0.13 ± 0.05	3.56 ± 1.54	5.41 ± 1.99
	RT2[d]	0.53 ± 0.27	0.11 ± 0.04	2.97 ± 1.28	5.34 ± 2.30
Traffic	TT1[e]	0.42 ± 0.24	0.09 ± 0.04	2.69 ± 1.14	5.18 ± 2.47
	TT2[f]	0.34 ± 0.20	0.08 ± 0.03	2.01 ± 0.84	4.97 ± 2.13
	F	69.2*	87.7*	115.5**	1.5
		a, c, d > b, e, f	a, c > b, e, f	a, c > b, d, e, f: b, d, e > f	

***Table 12**. Comparison of Continuous Decomposition Analysis (CDA) parameters (CDA.AmpSum, CDA.SCR, CDA.ISCR, and CDA.Tonic) between all negative videos across themes (rmANOVA results).*
µS = microsiemens; CDA = Conductance Data Analysis; AmpSum = amplitude sum of SCRs; SCR = Skin Conductance Response; ISCR = Integrated Skin Conductance Response; Tonic = tonic skin conductance level; M = mean; SD = standard deviation; F = Fisher's F ratio; PT1 = Physical Negative Video 1; PT2 = Physical Negative Video 2 (added video); RT1 = Relational Negative Video 1; RT2 = Relational Negative Video 2 (added video); TT1 = Traffic Negative Video 1; TT2 = Traffic Negative Video 2 (added video).
** $p < 0.05$; ** $p < 0.001$.*

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