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Significance and phenomenology of non-rapid eye movement (NREM) parasomnias. Data from a representative population survey, YouTube video collection, and clinical experiences

PhD thesis

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List of abbreviations

AASM: American Academy of Sleep Medicine

ACEs: Adverse Childhood Experiences

ACR: Absolute Rating Category

AI: Artificial Intelligence

BF: Basal Forebrain

CAP: Cyclic Alternating Pattern

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CI: Confidence Interval

CPG: Central Pattern Generator

DSM: Diagnostic and Statistical Manual of Mental Disorders

DOA: Disorders of Arousal

EEG: Electroencephalography

GABA: Gamma-Aminobutyric Acid

HSD: Hypersynchronous Delta

ICSD: International Classification of Sleep Disorders

LC: Locus Coeruleus

LDT: Laterodorsal Tegmental Nucleus

NREM: Non-Rapid Eye Movement

OR: Odds Ratio

ORX: Orexin

OSAS: Obstructive Sleep Apnea Syndrome

PLMS: Periodic Limb Movement Disorder

PPT: Pedunculopontine nucleus

REM: Rapid Eye Movement

RBD: REM Sleep Behavior Disorder

RLS: Restless Legs Syndrome

SCN: Suprachiasmatic Nucleus

SRED: Sleep-related Eating Disorder

SSRIs: Selective serotonin reuptake inhibitors

SW: Sleepwalking

SWA: Slow Wave Activity

TCAs: Tricyclic Antidepressants

TMN: Tuberomammillary Nucleus

TTFL: Transcription-translation Feedback Loop

VLPO: Ventrolateral Preoptic Area

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1 Introduction

NREM parasomnias, also termed disorders of arousal (DOA), constitute a curious and mysterious group of conditions manifesting undesired behaviors during non-rapid eye movement sleep. These conditions challenge our notion about sleep; the episodes present with parallel sleep-like and wake-like phenomena, partial awareness, and partial sleep (1). While there is ongoing research and an abundance of literature trying to understand these conditions, there is much data on the prevalence, facilitating factors, and consequences (often injurious) of DOA, as well as on the impact of genetic/psychological/environmental factors (2); the data remain contradictory.

The different kinds of study populations (sleep laboratory and sleep clinic patients, psychiatry in- and outpatients) (3), heterogeneous data-collection methods (retrospective analyses, questionnaires, telephone interviews, targeted web-based surveys, mostly involving persons affected in some way, etc.) (4, 5). This may have caused inconsistencies in the results. Population surveys rely purely on the affected persons' self-reports, with little or no recollection of their episodes. In those studies, usually, no historical data or medical diagnoses are provided (6). In most of the surveys, people may be unwilling to disclose sensitive or confidential data to a generally non-medical questionnaire. Sleep clinics, and especially sleep laboratory patients, represent selected populations; many of those persons seek medical help for the high frequency or injurious consequences of the episodes; they represent, by no means, the parasomnia population's average (7). YouTube and other community videos on sleep-related activities suffer another bias of non-representativeness, displaying those potentially extraverted persons happy to appear in public, still selecting non-humiliating, non-embarrassing videos, e.g., on sexual behaviors. In many countries, having access to the internet is another selection bias. In addition, this method provides just phenomenology (semiology) features without the support of medical histories, collaterals' comments, or investigations. Therefore, I aimed to overcome those limitations as much as possible by applying each method, criticizing them, and finally, trying to combine the results to come to conclusions.

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1.1 Sleep Physiology in a Nutshell

Sleep is a naturally occurring, reversible state of reduced responsiveness to external stimuli, characterized by distinct neurophysiological patterns, including specific electroencephalographic (EEG) changes, altered muscle tone, and reduced movements (8). It consists of two main phases — rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep — each with specific functional, behavioral, and neurophysiological properties (9). Those sleep phases have multiple distinct roles (10) in memory and emotional regulation, bodily and mental restoration, metabolism, and maintenance of normal functioning (11). Two main regulatory forces determine the timing, quantity, and "depth" of sleep:

1. **Homeostatic regulation to maintain a steady state** (from ancient Greek ὅμοιος (hómoios) = "similar, the same", and stasis *στάσις (stasis)* = "standing, state") (12).

   Sleep homeostatic power is described in Borbély's two-process model as process S. It accumulates during wakefulness and decreases during sleep, regulated by prior wake duration and degree of activity or exhaustion (13).
2. **Circadian regulation to maintain daily rhythm** (from Latin: *circa = about,* and *dian = day*).

   It is described in Borbély's two-process model as process C. It follows a roughly 24-hour cycle (14), synchronizing biological activities with the regular day-night alternation, such as sleep-wake patterns, hormone production, metabolism, and cellular rhythms (15). The master clock, localized in the suprachiasmatic nucleus (SCN), receives direct signals from the retina that detect light levels, driving the expression of clock genes (16). It also generates a self-sustained oscillatory rhythm through the cyclic expression of clock genes in a transcription-translation feedback loop (TTFL) (17). The SCN then sends signals to the rest of the brain and body to synchronize all daily processes (18). Among many other functions, the SCN regulates the activity of the pineal gland (19), which secretes melatonin in darkness (about two hours before bedtime in people with regular sleep patterns). Melatonin is a marker and an essential participant in the circadian system, fine-tuning its regulation and synchronizing

it with the external light cycle (20). Conversely, to melatonin, cortisol levels rise throughout the night, peak in the morning, and decline during the day (16), and other hormone secretions are also under circadian regulation (21).

1.1.1 Sleep Architecture

Sleep duration depends on genetic determination, age, prior sleep deprivation, medication, and general health (22). It occurs in regular, about 90-minute cycles of non-rapid eye movement (NREM) and rapid eye movement (REM) phases, constituting sleep macrostructure (22). The recent AASM classification (23) defines three sleep stages, N1, N2, and N3, based on the proportion of slow waves (N3 is consistent with stages S3 and S4 of Rechtschaffen and Kales (24)). The first sleep phase between wakefulness and superficial sleep lasts 1-7 minutes, accounting for 2-5% of total sleep time (25). It can be easily disturbed by noises or artificial light (26). Stage 2 sleep lasts 10-25 minutes during the first cycle and extends in subsequent cycles, constituting 45 to 55 percent of night sleep. More potent stimuli are needed to wake up someone from stage 2 sleep than from stage 1 (22). Stage 3 takes 15–25% of healthy adults' sleep time and is characterized by a high slow wave power on the EEG. An adult sleeps 7–8 hours per night, with stage 3 sleep lasting 60–120 minutes. Deep sleep dominates the first sleep cycle and tends to decrease later during the night (22). REM sleep (constituted by phasic and tonic REM phases) is characterized by desynchronized EEG activity resembling wakefulness as well as striated muscle atony, vivid dreams, and bursts of rapid eye movements (27). In the first sleep cycle, the REM period is 1-5 minutes long, and it lengthens gradually overnight across sleep cycles (10). Many new data highlight the sophisticated and fine-tuned regulation of NREM and REM sleep, including the orexin (hypocretin) system's roles (10,28), and a widespread sleep-wake regulation system extended to several regions and pathways of the brain.

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1.1.2 The Regulation of Sleep and Wakefulness

Complex interactions of homeostatic and circadian processes regulate sleep and wakefulness, driving the mutual interactions of arousing and sleep-promoting forces. (12). Wakefulness is regulated by the cholinergic ascending reticular arousal system, comprising specific cell groups and neurotransmitters in the brainstem, hypothalamus, and basal forebrain (BF) (12). This system involves the upper brainstem, specifically the cholinergic pedunculopontine (PPT) and laterodorsal tegmental nuclei (LDT) (29). Ascending further, it stimulates the cerebral cortex through cell groups in the upper brainstem and caudal hypothalamus, including the noradrenergic locus coeruleus (LC), serotonergic raphe nuclei, the dopaminergic ventral periaqueductal grey, and the histaminergic tuberomammillary nucleus (TMN) (30). Neurons of the ventrolateral preoptic area (VLPO) in the anterior hypothalamus promote sleep (29).

Sleep regulation follows 'FLIP-FLOP' patterns with the cyclic alternations of sleep-wakefulness and REM-NREM sleep (31). The brainstem centers activate the cerebral cortex, leading to wakefulness or REM sleep, determined by the combination of chemical and electrical signals (32).

Micro-oscillations and rhythms constituting the microstructure of sleep build up each sleep cycle (10). Its elements are the bistable ~1Hz slow waves, K-complexes, sleep spindles, and the cyclic alternating pattern (CAP) (33), as well as microarousals (arousals without awakening). There are two types of inherent microarousals: some of them (termed anti-arousals) enhance sleep (protect and promote NREM sleep by boosting slow waves) (34), and default or traditional arousals, which promote wakefulness, with EEG desynchronization (36). Whether an arousing stimulus evokes a sleep-promoting anti-arousal or "traditional" arousal depends on the level of homeostatic pressure and the arousal's position within the sleep cycle (35). Intrinsic rhythms govern the dynamic sleep process, while internal and external stimuli influence the arousal levels, adjusting them to immediate needs (35, 36).

Sleep patterns undergo significant ontogenetic changes across human life, influenced by age-related physiological alterations and lifestyle variations (37). Understanding these

differences is crucial for optimizing sleep health and addressing age-specific sleep challenges.

- **Infancy and Childhood**: Circadian rhythms appear early, around the first month of infancy, with increased nocturnal sleep in the second month, as well as melatonin and hormone cycles by the third month (10). Sleep architecture is driven by neuronal pathways involved in sleep regulation (26). One study found that children appear to have distinct but shorter sleep cycles and a higher proportion of rapid eye movement (REM) sleep than adults (38). However, children spend more time in stage 3, deep slow-wave sleep, and have longer REM sleep latencies than adolescents (22).

- **Adolescence:** Changes in neurotransmitter systems, particularly dopamine and serotonin pathways, affect sleep regulation, delaying sleep onset (39). Sleep latency progressively declines, paralleling pubertal development, leading to a mismatch in internal biological clocks (40) and an important reduction in SWS (22).

- **Adulthood:** Aging may bring sleep timing earlier, reduce the duration of nocturnal sleep, and facilitate daytime napping (10). The quantity of slow-wave sleep (SWS) decreases further, leading to more frequent awakenings due to lower arousal thresholds and short arousals near REM sleep transitions (41).

- **Elderly:** Difficulties with sleep initiation and maintenance are typical (42). The number of SWS decreases (43,44), and stage 1 sleep increases while REM sleep decreases. Older adults experience reduced melatonin levels, increased sleep latency, and fragmented night sleep. External cues' inconsistency contributes to poorer sleep quality (42). Aging alters brain structures involved in sleep regulation; vascular changes, cortical thinning, and white matter degeneration may occur (45). Growing evidence links age-related sleep changes to adverse health effects, especially cognitive decline (46). Basic sleep alterations after age 60 include shifts in sleep timing, disrupted sleep maintenance, and decreased sleep spindle activity (47). Interestingly, sleep deprivation in older adults causes less cognitive impairment than in the young, reducing vulnerability to prolonged wakefulness (43,44).

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1.1.3 Local and Mosaic-like Sleep

The recognition of the protective alternating half-hemisphere sleeping of big aquatic mammals and birds of long passage (48) has highlighted that sleep is not ‘global’; there is a biological possibility and need for the simultaneous occurrence of sleeping and waking in different brain parts (49). This is seen in a mitigated form in human sleep as well, where different sleeping-waking depths and states normally co-occur, justifying the term "mosaic-like sleep-wakefulness" or local sleep (50). Sleepy persons typically manifest micro-sleeps during waking: sleep-like local slow waves consistent with ‘sleep isles’ intrude into wakefulness, as shown by electroencephalography (EEG). Therefore, sleepiness may lead to transient lapses in attention and cognitive performance (53, 54). In other words, local slow waves appear in the waking EEG (52). An inverse example of sleep dissociation is seen in NREM sleep, where slow-wave sleep coexists with local patches or columns exhibiting faster, wake-like EEG activities; isles of more superficial sleep or wakefulness intrude upon slow-wave sleep (50).

The phenomenon of local sleep is closely related to the homeostatic drive (52). Longer-lasting or more intensive ‘use’ (activity) of a cortical region leads to synaptic down-regulation (53) or ‘exhaustion’, resulting in deeper subsequent sleep (more slow waves on EEG) occurring in line with other, less ‘used’ cortical regions remaining in shallower sleep (54). Thus, in NREM sleep, there are cortical columns in deeper sleep, as shown by local slow waves (52), coexisting with other ones, “awake” or in superficial sleep. Due to the homeostatic power, the longer a cortical region is ‘awake’, the higher the probability of its falling asleep (34,55,56). Understanding local sleep is essential for sleep research and treatment of sleep disorders, especially NREM parasomnias, where this normal mosaic-like sleep-waking pattern is augmented during the episodes (54); a part of the brain is awake or near to it, while another one remains in deep sleep (50).

1.1.4 Central Pattern Generators: A Neuroethology Approach

Central Pattern Generators (CPGs) are neural brain networks that can produce rhythmic outputs without external input, essential for survival. CPGs orchestrate ancient motor behaviors such as chewing and swallowing, flying, swimming, walking, respiration, and reproduction (57); we “know” these movements and behaviors without learning them. The sophisticated cellular mechanism of how CPGs control those movements has been nicely elaborated in the paper of Marder and Bucher (58). In higher primates, including humans, CPGs are under neocortical control (59). The neuroethological theory of Tassinari states that these fixed ancient motor movements emerge in the movements of epileptic seizures and parasomnia episodes, presenting those innate motor patterns (60). Tassinari's work also highlights the shared features of these conditions, likely involving the same neuronal networks (61).

1.2 Parasomnias

1.2.1 Definitions

‘*Parasomnias are undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal from sleep. Parasomnias may occur during non-rapid eye movement sleep (NREM), rapid eye movement sleep (REM), or during sleep-wake transitions’* (62, 63).

NREM parasomnias are defined as conditions (1) not secondary to psychiatric disorders; (2) not secondary pathologic brain condition or head injury; (3) linking to absent or minimal cognitive functioning; (4) no memory for the episode; and (5) often triggered by arousing stimuli such as sound or touch (63).

The sleep-related episodes of NREM parasomnias typically occur during deep NREM sleep of the first few hours of night sleep, in the first or second sleep cycles, at the turning point of the descending (deepening) and ascending (arousing) slopes of NREM sleep (64). Sleep-disturbing conditions and factors such as leg movements in periodic

leg movement disorder, apneas in sleep apnea syndrome, bruxism, external noises, a distended bladder, or sudden pain may precipitate the episodes (65).

While the typical onset of NREM parasomnias is childhood, they may start or persist into adulthood (62). The adult variants are somewhat different compared to the childhood ones. Those DOA episodes in adults arise from shallower NREM sleep than in children; they are associated with less consistent amnesia and more conscious experiences than in childhood (66). In adult cases, more violence occurs, sometimes leading to physical injury and forensic issues due to aggressive behaviors. Psychological distress may be associated (67). In addition, based on a twin study on NREM parasomnias, the concordance rate of childhood parasomnias is significantly higher than it is in adults (68), marking less impact of genetics in adults than in children. Based on the above, the question seems justified: Do childhood and adult variants represent distinct entities or just two ends of a DOA spectrum (7,69–71)?

The classical definition states that most patients are unaware and have amnesia of the episodes; while some individuals can partially recall them in the morning, others just infer from moved or utilized items or injuries (72). Challenging this idea, recent research has revealed frequent conscious (hallucinatory or real) experiences associated with NREM parasomnia episodes (73) and found that about 60% of adult parasomnia patients (versus just 30% of childhood cases) have some recall of their episodes (34).

Parasomnia episodes may present with dangerous activities, including violence during sleep (typically in adulthood), raising forensic issues with questions of responsibility (74). Activities like sitting up, leaving the bed, and walking during sleep are typical; risky behaviors such as handling sharp objects, electricity, running out of the house, or driving have been described (74). Seeking medical help is typically motivated by challenging or traumatic sleep events (75). Risk management begins with identifying the potential dangers associated with the individual's sleep-related actions.

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1.2.2 Classification of NREM parasomnias

a. Confusional arousals
b. Sleepwalking
c. Sleep terrors
d. Sleep-related eating disorder
e. Sexsomnia
f. Sleep talking
g. Parasomnia due to medical conditions

The 5th edition (revised) version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5TR) classifies NREM parasomnias as “sleepwalking” with subsequent specifiers such as “sleep-related eating”, “sleep-related sexual behavior (sexsomnia)”; and “sleep terror” (77).

a) Confusional arousals often occur due to forced awakenings, particularly in individuals lacking sufficient sleep. Sedative hypnotics can contribute to these episodes. These typically brief events may involve sleep-talking and simple motor actions, usually not remembered later by the sleeper (79). Their frequency diminishes with age; persistence into adulthood may indicate underlying health conditions or the effect of medication (76).

b) Sleepwalking (somnambulism) means ambulation in sleep. It is often linked to other complex behaviors (64). During the episodes, the affected individuals may have blank expressions and be unresponsive to external stimuli (80). Sleepwalking persons sometimes engage in half-adequate or inadequate dialogues during their episodes (81).

   This is the most prevalent non-rapid eye movement NREM parasomnia, sometimes posing risks of injury to the individual or others (82). It has a 13-15% (or even higher) prevalence in childhood (83), diminishing across increasing age, with a prevalence of ~4% in adulthood and becoming more seldom in the elderly (68).

c) Sleep terrors manifest behaviors reflecting sudden and intense fear, escape, panic, and agitation during sleep (83) in the absence of any frightening dream or event (84). The affected persons do not wake up and, upon awakening, typically have no memory of the episode (85). Usually, no injuries occur (86).

d) Sleep-Related Eating Disorder (SRED) is featured by episodes of preparing and consuming food and drink while sleeping or partially awakening from NREM sleep. Typically, there is no awareness or recollection of the events. Individuals with SRED may consume unusual or normally inedible foods such as raw meat, deep-frozen stuff, soap, and even poisonous or dangerous substances, experiencing significant health risks, including choking, weight gain, or poisoning (87). Interestingly, this condition is a typical comorbidity of restless legs syndrome (RLS) (89). Females are affected more often than males (3).

e) The DSM-5 and the ICSD-3 recognize sexsomnia as a performance of sex during sleep (78). It is considered rare, with less than 150 cases documented in the literature by 2024 (88), but it might be much more prevalent. DSM-5 lists sexsomnia under the diagnosis “NREM sleep arousal disorders” (89); and one paper (90) has raised the issue that, in some cases, it may represent REM parasomnia (REM Sleep Behavior Disorder – RBD rather than DOA. People with sexsomnia may exhibit masturbation, sexual vocalizations, or even intercourse, often without awareness or memory of the events upon waking (91). This condition can lead to conflicts in relationships and may have legal implications if sexual activity without consent is considered ‘molestation’ or ‘raping’ by the sex partner (92).

f) ICSD-3 recognizes sleep talking (somniloquy) as a standard variant or isolated symptom associated with both NREM and REM parasomnias (77). It is classified within the broader context of sleep-related phenomena and is often considered benign unless it accompanies other sleep disorders (76). It can range from simple sounds or mumbles to coherent speech (93). While common and harmless, excessive or disruptive sleep talking may warrant further evaluation to exclude other underlying sleep disorders or medical conditions, including epilepsy (94).

g) *Parasomnia due to medical conditions* may evolve when those conditions affect sleep architecture and regulation (95).

Because of the high frequency of such associations, a careful patient history, preferably with eye-witness accounts, collateral experiences, and home video records, is essential in cases of sleep-related activities, considering symptoms, frequency of episodes, their night distribution, comorbidity, treatments, and family history as well (96).

1.2.3 Some viewpoints on the mechanism of NREM parasomnias

- **Sleep-wake dissociation:** Sleep-wake dissociation is considered the primary mechanism of parasomnias. Sleeping and waking or partially waking cortical regions co-occur in NREM parasomnias; elements of certain sleep stages emerge at inappropriate times or lack in their typical sleep phase in REM parasomnias. (2). Sleep-wake dissociation is a normal protective phenomenon in birds of passage and big aquatic mammals. In them, one brain hemisphere is sleeping, and the other hemisphere is awake, and sleeping-waking alternates between hemispheres (97). This pattern of alternating rest and alertness provides the animal with ongoing protective vigilance and the ability to control activity and breathing while sleeping (48). A regular mosaic-like sleep-wake pattern characterizes humans, too. Due to the homeostatic drive, different sleep depths (as measured by the amount of slow waves) occurring in cortical 'patches' reflect the duration and extent of those regions' activity during the previous day. This flexible regulation is termed "use-dependent homeostatic control" (34). In NREM parasomnias, the normal mosaic-like sleep dissociation is augmented. Those conditions arise due to incomplete arousals from deep sleep, consistent with sleep dissociation (a part of the brain sleeping, and another part is partially or fully awake) (98). In addition, a specific distribution pattern may be hypothesized in the different subtypes of parasomnias (e.g., in sleepwalking, the motor cortex would be awake within the overall sleeping brain, and in sleep-sex

or sleep-eating, the amygdala or other 'reward' regions would be awake while the rest of the brain is sleeping (99).

- **Central pattern generators:** The theory on central pattern generators of Tassinari gives another concept of the mechanism of parasomnia-related activities. Indeed, in many of those behaviors, innate ancient motor patterns can be recognized – e.g., walking, running, sexual activities, eating (59). Based on this theory, those activities would be driven by the outputs of CPGs that lose neocortical control during sleep. However, the CPG concept does not seem to explain all parasomnia-related activities. The ones that represent learned and complex behaviors, such as playing the piano, driving a car, packing and unpacking, etc., might have different origins, better explained by the sleep-wake dissociation concept (61).

- **Sleep-relations:** NREM parasomnia episodes occur at the "clash-points" of sleeping and waking, where deep sleep meets arousal, which can partially overcome deep sleep; therefore, sleep-wake dissociation may evolve. Genetic factors, sleep-deepening, and fragmenting factors have a role (79). The abundance of deep NREM sleep in children may account for NREM parasomnias' high prevalence, and one of the reasons why those conditions are less frequent in adults and the elderly may be related to the progressive decrease of deep sleep (22). In adult NREM parasomnias, night sleep is fragmented and more superficial than in childhood; the role of arousing forces is increased (38). Since the ratio of N3 sleep peaks in childhood when NREM parasomnias accumulate, it has been proposed that adults experiencing NREM parasomnias might have more N3 sleep than controls (100). Polysomnography studies have not confirmed this, but adult patients with DOA had more frequent arousals from N3 sleep and less N3 sleep compared to healthy controls (101). DOA episodes link to (partial) arousals and mostly occur during the first sleep cycle, at the turning point of deep NREM sleep, where sleep deepening (the descending slope of the sleep cycle) turns towards superficial sleep phases (to

the ascending slope) (71). This timing of the episodes, the increased arousal expressed by sleep fragmentation, and the related abnormal and specific dissociation features justify NREM parasomnias' recent name: disorders of arousal (DOA). NREM parasomnia and DOA are interchangeable terms; I use both across the thesis.

- **The relationship between Slow Wave Activity (SWA), Hypersynchronous Delta Activity (HSD), and Disorders of Arousal (DOA):** EEG Slow Wave Activity (SWA), built up by synchronous high-amplitude ≈ 1Hz delta waves, is a hallmark of NREM sleep, profound slow-wave sleep (SWS) (102). Hypersynchronous Delta Activity (HSD) refers to continuous, high-voltage delta waves exceeding 150 microvolts in the EEG, also occurring in SWS (103). A study by Suzuki in 2024 found that continuous delta waves after arousal emerge less frequently in older individuals, suggesting an age-dependency (104). Another study by Pressman (2004) found that individuals without a history of parasomnias exhibited frequent HSD and SWA with arousals, suggesting a link between these patterns (105). A case study by Kang (2023) reported an adult patient with NREM parasomnia who exhibited prolonged post-arousal HSD (106). Jaar (2010) found that adult sleepwalkers exhibited significantly lower overall SWA, particularly during the first NREM cycle, than controls. This reduction in SWA may have contributed to the instability of sleep (100). Pilon (2006) found more HSD in sleepwalkers than in healthy controls, suggesting a potential link between HSD and NREM parasomnias (103).

1.2.4 The Management and Treatment of DOA

Management of DOA involves safety measures, counseling against sleep deprivation, irregular sleep-wake patterns, and sleep-disrupting lifestyle issues. Alcohol and drug use need to be reduced, and it is necessary to address comorbid sleep disorders such as obstructive sleep apnea syndrome or periodic leg movement disorder (65). Techniques

of introducing regular sleep-wake patterns with scheduled sleep and awakenings may help. Psychotherapy, relaxation, and hypnosis are described as additional treatment options (75,107), but pharmacotherapy is often necessary for individuals experiencing frequent or risky episodes. Medications such as melatonin, benzodiazepines, as well as antidepressants - imipramine and paroxetine - are commonly used (101,108,109).

1.2.5 Differential Diagnosis

For both prognostic and therapeutic implications, DOA needs to be distinguished from other sleep disorders, e.g., from REM sleep behavior disorder (RBD) and, most importantly, from nocturnal (mainly frontal lobe) seizures (**Table 1**). Additionally, it is crucial to rule out medication- or substance/alcohol-induced nocturnal confusion before diagnosing DOA (71).

Unlike NREM parasomnias, which typically but not exclusively affect young people, RBD evolves preferentially in elderly males. While DOA episodes are linked to the early hours of sleep (96), episodes of RBD favor the second part of the night, which is dominated by REM sleep. Polysomnography reveals that RBD is underlined by REM sleep without atonia, allowing dream enactment, owing to the absence or just partial presence of the typical loss of muscle tone characterizing normal REM sleep (110). RBD may involve disruptive, abrupt-onset, and violent fragmentary movements during sleep (7), reflecting the dreams of the affected persons who may escape or fight accordingly (111). Those activities can harm or disrupt REM sleep (109) and cause injuries to the patient or bedpartner. Unlike sleepwalking, where the individual's eyes are open during the episodes, in RBD episodes, the eyes are typically closed (65).

RBD can remain isolated but may be associated years later with neurodegenerative conditions, especially synucleinopathies like Parkinson’s disease or dementia with Lewy bodies; it is potentially consistent with an early phase of those conditions (112). It manifests another type of sleep dissociation than in NREM parasomnias, where an element of the REM sleep pattern, such as the atonia of striated muscles, is absent or fragmented (111).

The differentiation of DOA episodes from nocturnal frontal lobe seizures is challenging, as also shown by the ongoing debate in the literature based on early and even recent studies (113). It has been raised whether the two conditions represent distinct entities or if they are consistent with two ends of a spectrum of arousal disorders, showing shared genetic, semiology, and sleep-distribution features, and even overlaps of brain localizations (114–117). A recent study raised discriminatory cognitive and psychiatric profiles in DOA and sleep-related hypermotor epilepsy patients (118). However, the classification of sleep-related episodes remains an issue of discussion (119). Discriminating night terrors from the so-called 3rd degree agitated hypermotor seizures, the two conditions have overlapping symptoms and shared features (84), making it especially hard. Video-EEG-polysomnography is the gold standard tool for differential diagnosis and may give a clue (120); however, sometimes it does not help either because frontal lobe epilepsies may go without interictal or even ictal electroencephalography signs on the scalp (121). Recent sophisticated EEG methods calculating spectral powers, aperiodic sleep-EEG components, and scalp gamma frequencies have revealed specific differences between episodes/seizures of DOA versus frontal lobe epilepsy; however, these methods are far from everyday clinical use (122).

In the present stage, clinical features can provide reasonably good discriminatory points. The stereotypy of the episodes (typically absent in DOA), tonic posturing, dystonic and dyskinetic features or jerks; an abrupt onset and short (<1 min) duration, as well as the accumulation of several seizures/night contrasting the rare or very rare DOA episodes (maximum 2/night or much more seldom), may support the diagnosis of frontal lobe seizures (117).

The differentiation of nocturnal frontal lobe seizures of sleep-related hypermotor epilepsy (SHE) (123), DOAs, and RBD is crucial for appropriate management. SHE is a form of epilepsy, while DOA and RBD are not epileptic but parasomnias. Below is a detailed comparative table highlighting the key differences between these conditions, emphasizing clinical features, diagnostic methods, and treatment approaches (**Table 1**).

Table 1. The differences between nocturnal epileptic seizures, episodes of DOA, and RBD

Feature	**Epileptic seizure**	**DOA**	**RBD**
Sleep Stage	Most seizures occur in the first third of night sleep, typically from deep NREM (124).	Episodes from deep NREM sleep transition to more superficial phases, typically in the first 2 hours of the night (41).	Episodes occur during REM sleep, typically later at night when REM sleep dominates (125).
Number of episodes/night	Typically, several/night	Maximum 2; typically, 1/night or much more seldom	1-2/night
Motor symptoms	From simple arousal-like phenomena (head lifting..) to convulsive, dystonic, or dyskinetic movements; < 1 minute duration; abrupt onset; tonic limb extension; cramping (126).	Subtle arousal movements through sleepwalking to uncoordinated and panic-like escape behaviors, typically lasting 5-20 minutes. (79).	Sudden, abrupt movements that may involve escaping, violent or aggressive actions (e.g., punching, kicking) (62).
EEG Findings	Epileptic signs on EEG, often during sleep; spikes or sharp waves in frontal regions. The scalp epileptic EEG	No epileptic signs. Hypersynchronous delta may occur before or after the episodes (106).	Polysomnography's hallmark is the lack or partial absence of striated muscle tone during REM sleep

	signs may be absent even during seizures (127).		(111).
Diagnosis	Confirmed or supported by overnight video-EEG monitoring or home video records. Clinical data are essential.	Primarily clinical. Home video, video-EEG-polysomnography, and history are supportive. A sleep study may reveal other comorbid sleep disorders.	Polysomnography reveals REM sleep without atonia (128).
Typical comorbidities	Parasomnias or other types of epilepsy (118).	Other sleep disorders include OSAS and RLS (priming or precipitating the episodes) and sleep-related hypermotor epilepsy (118).	Often preceding or associated with neurodegenerative conditions, such as Lewy body dementia and Parkinson's disease (112).
Treatment	Anti-epileptic with lifestyle modifications and sometimes surgery (129).	Safety measures, sleep scheduling, cognitive-behavioral therapy, and medication (e.g., benzodiazepines, antidepressants, Melatonin) (108).	Clonazepam, melatonin, and safety measures are also included (130).

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1.3 Predisposing, Priming, and Precipitating Factors of NREM parasomnias

The features and conditions promoting NREM parasomnia episodes can be classified as predisposing, priming, and precipitating (triggering) factors that interact (131) and create the 'ideal' premises for the development of those sleep-related episodes (132). These factors have cooperative roles and participate with different weights in promoting the episodes (133). Without their variable combinations, the occurrence of NREM parasomnia episodes is unlikely (62,134). Typically, those factors increase homeostatic sleep pressure, e.g., in childhood or disrupt slow-wave sleep in adulthood, having the same homeostatic effect. High sleep pressure meets arousal forces, constituting "collisions" between deep sleep and arousal.

1.3.1 Predisposing Factor: genetics

A substantial family accumulation of NREM parasomnias is well known. The probability of NREM parasomnia in a child was reported at 22% if neither parent had NREM parasomnia, 45% if one parent had it, and 60% if both parents were affected (83). One study suggested that developing NREM parasomnia shows an autosomal dominant trait (62). In a twin-study study of childhood sleepwalking, the concordance rate was 1.6 times greater in monozygotic vs. dizygotic twin pairs (133); this effect was significantly stronger in childhood cases than in adults (68). Compared to the general population, a person had 10-fold odds of having DOA if there was a DOA case in the family (134).

Sleepwalking, sleep terrors, and confusional arousals have been associated with the human leucocyte antigen (HLA) system, particularly with the HLA DQB1*05:01 locus (135). The study was conducted in a Caucasian sample, where more than 30 percent of sleepwalkers carried this HLA-type, compared to 13.3% of controls (136). This HLA allele was found in 41% of those with familial cases. Licis and colleagues found a single genetic locus for sleepwalking at chromosome 20q12-q13 in a large family with

somnambulism (137). Further research on genetic factors influencing this field is actively conducted (131,138).

1.3.2 Priming Factors

Priming factors make arousal from sleep more difficult by deepening sleep (134). If arousing (wake-promoting) forces occurring in deep sleep are relatively weak, they may partially surmount sleep-promoting forces. Such a scenario favors sleep dissociation and NREM parasomnia episodes (97). Priming factors deepening sleep and increasing sleep inertia include sleep deprivation, certain medications, stress, hypothyroidism, migraine, variable brain conditions, regular alcohol use, etc. (4,134).

- **Sleep deprivation:** It increases sleep pressure and promotes the subsequent deep NREM sleep, e.g., in shift-working or due to irregular sleep-wake patterns (139). Insufficient and disrupted sleep seems to be the most significant DOA-priming factor; in many cases, the sole intervention of regulating sleep and providing sufficient sleep may "cure" NREM parasomnias (134).

- **Medication:** Several case reports have documented NREM parasomnia episodes primed by medications, Non-benzodiazepine (Zolpidem) (203), Benzodiazepines (Temazepam, Alprazolam, Diazepam) (204), Antidepressants (Citalopram, Amitriptyline) (205), Antipsychotics (Quetiapine, Risperidone, and Olanzapine) (206,207), and mood stabilizers (Lithium) (140,141). Zolpidem has been described as associated with car driving during sleep, leading to forensic consequences in a series of eight patients (142). Zolpidem and Quetiapine are known to prime sleep-eating (2,143). Methodological issues limit research on this topic since studies involving patients with poly-therapies can skew results. Non-benzodiazepine sedatives may be more likely to cause sleepwalking in psychiatric patients, possibly due to interactions with other drugs (134). There is a lack of controlled randomized trials specifically studying medication-induced NREM parasomnias. The actual mechanism is not known, but there are some hints: antagonism of dopamine transmission (Z

drugs as stimulators of GABA, which can inhibit dopamine cell bodies directly) (208). As dopamine is important for the transition between sleep stages (and particularly the transition into REM, a delay in this by antagonism of dopamine could keep the person in NREM longer) (209). Some of the medications implicated in the treatment and potential induction of NREM parasomnia found in the literature are shown in **Table 2**.

Table 2. Proposed mechanism of medications implicated in the treatment and potential induction of NREM Parasomnias by drug classes

Drug Classes	Proposed Mechanism
Sedative-hypnotics (Benzodiazepines and Z drugs)	It is known that the action of these drugs on the cortex produces high-amplitude slow waves, stage 3–4 sleep, may fragment sleep architecture, and cause incomplete arousals (204).
Antidepressants (SSRIs and TCAs)	Increase sleep fragmentation; SSRIs can deepen sleep initially, but fragment REM/NREM architecture (205).

- **Alcohol:** In non-alcoholic persons, acute alcohol consumption shortens the latency of sleep and improves the quality (expressed by delta power) and duration of NREM in the first hours of night sleep; however, sleep becomes fragmented in the subsequent parts. Regular alcohol users, both during drinking and abstinent periods, experience multiple types of sleep issues, such as severe insomnia, excessive daytime sleepiness, and abnormal sleep structure (144). Because chronic alcohol use can modify sleep chronically, it can prime disorders of arousal (145). Studies show that 92% of NREM parasomnia patients who drink regularly experience more parasomnia episodes and more fragmented sleep; their likelihood of experiencing NREM parasomnia episodes

is higher than in parasomnia patients who do not regularly consume alcohol (134).

- **Stressful or traumatic events:** There is a clear link between stressful or traumatic events and the onset or worsening of NREM parasomnias (134). Lecendreux and colleagues identified stressful life events that preceded sleepwalking in 56.7% of their patients. (136)

Adverse Childhood Experiences (ACEs) are defined as potentially traumatic events occurring in childhood (0-17 years), e.g., experiencing physical and emotional abuse, including sexual abuse, or growing up in an unstable household (146, 147). ACEs are not isolated episodes but series or collections of experiences shaping a child's circumstances and sense of safety. These events and circumstances can significantly impact the development in childhood and may have long-lasting effects on physical, emotional, and mental well-being (148). Examples include emotional, physical, or sexual abuse, parental separation or divorce, substance abuse within the household, or exposure to domestic violence (149). ACEs can significantly alter children's behavioral and physiological development (150), affecting their health even in adulthood. These experiences evoke stress responses that precondition the body for fight-flight (151). Prolonged exposure to stress can disrupt the body's regulatory systems, leading to lasting and maladaptive physiological dysfunctions (152). Therefore, early-life adversities, including stress or even infections, can set the stage for health issues in adulthood (146). ACEs, together with genetic predisposition, set the stage for NREM parasomnia episodes to happen (62). Lacey and colleagues' theory on “immune programming” is grounded: early stress or trauma may influence the immune system over time (153). The biological mechanisms likely involve changes in the hypothalamic-pituitary-adrenal (HPA) axis, increased levels of cortisol, and altered cellular immune functioning (154). The prolonged activation of stress-related systems may prepare the ground for different conditions manifesting years or decades later (142, 146).

- **Other priming factors:** Based on the literature, cardiovascular disease (155), diabetes mellitus, menopause (156), and vitamin D deficiency (157) can prime parasomnias as well.

1.3.3 Precipitating Factors

Precipitating factors are the immediate triggers of an episode in someone predisposed or primed to NREM parasomnias:

- **Episodes of other sleep disorders and conditions:** Microarousals due to excessive movements of periodic limb movement disorder (PLMD) can elicit episodes of NREM parasomnias. (62). Similarly, sleep apnea or, based on a case report, laryngospasm related to gastroesophageal reflux (158), can be precipitators of DOA episodes by arousals from N3 sleep (159). A distended bladder or any pain may have a similar effect (63,77,78). To identify underlying sleep disorders and conditions, it is necessary to take a thorough past medical history and a medical check-up. If a medical disorder is the precipitant of DOA, it is diagnosed as P*arasomnia due to a medical disorder* (23). In such cases, treating the underlying condition is likely to treat DOA as well.

- **Environmental Influences:** External factors often trigger arousals or partial awakenings (e.g., noise and temperature changes). Noise can induce parasomnia episodes in susceptible individuals, especially when sleep-deprived (39).

- **Alcohol:** Alcohol intoxication has been removed from the list of triggering factors by both ICSD-3 and DSM-5 (63,77); however, it has remained an important priming factor, chronically transforming the structure of sleep (134).

In the literature, episodes of NREM parasomnias could be precipitated in patients with genetic predisposition and the presence of priming factors, but not in controls,

highlighting that the combination of predisposing, priming, and triggering factors is necessary for the emergence of those events (62).

1.4 Artificial Intelligence (AI) video analysis for diagnosing sleep disorders

Researchers at Mount Sinai Hospital have developed AI algorithms that analyze video recordings of sleep-related episodes to find identification points of different sleep-related behaviors, e.g., REM Sleep Behavior Disorder (RBD) (160). Utilizing an AI-powered algorithm assessing 3D video recordings on sleep behavior, 92% accuracy was achieved in diagnosing RBD (160). While this research focused on RBD, AI technology can potentially identify other sleep-related behaviors, including NREM parasomnias and seizures.

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2 Study Objectives

I aimed to collect data on the epidemiology, phenotypes, contributory factors, and injurious consequences of adult NREM parasomnias.

I had three approaches: (A) a cross-sectional representative population survey, (B) collecting and analyzing YouTube videos, and (C) retrospectively reviewing the database of Semmelweis University's Sleep clinic to investigate the following questions:

1. What is the prevalence of NREM parasomnias in Hungary?
    a. What is the proportion of different subtypes?
2. What is the rate of potentially dangerous activities during NREM parasomnia episodes?
3. How often is there a family accumulation of NREM parasomnias?
4. Do ACEs influence the occurrence of NREM parasomnias?

2.1 Ethics

The Semmelweis University consented to the studies on numbers 248/2022, 29/2023, and 1/2024, respectively. Informed consent was obtained from all participants, except for the persons in the YouTube videos. Videos on YouTube channels are published under a Creative Commons Attribution License (CC BY), allowing their use for non-commercial purposes, with no modifications or adaptations being made. Research use is allowed after proper citation (161).

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3 Methods of the three studies

A) Data representing the Hungarian population in 2023

A community-based representative survey was conducted between February 28 and March 8, 2023, targeting adults aged 18 and above in Hungary. A paid professional pollster company, Závecz Research, selected the participants using the random walk method (162). This method provides a systematic approach to approximating random household selection by starting at a randomly chosen point within a defined area and proceeding along a predetermined route while selecting units at fixed intervals (162,202). By combining operational simplicity with basic probabilistic principles, the random walk method helps to minimize selection bias, together with the multicriteria weighting procedure, computer-assisted personal interviewing (CAPI) method (202).

They selected 1000 respondents in a non-probability quota sample. For data collection, they applied the "bus" method: two or three different topics were targeted simultaneously, which allowed two or three parallel surveys to be performed; the participants responded to questions on each topic. After getting the participants' written informed consent, the non-medical questioners of Závecz Research interviewed the participants personally, using a computer-assisted method via laptops or tablets.

The survey encompassed independent sociodemographic variables such as age group (19-30, 31-49, or ≥50), biological sex, residence (urban or rural), and education level (primary, secondary, or graduated), stratified by regional demographics from the last census. This approach ensured the representativeness of data (163). After collecting sociodemographic data, participants were queried about their sleep-related activities, family history of parasomnias, whether they had experienced ACE and its type, and their potential engagement in risky behaviors during their episodes by the following questions:

1. Have you ever (or have you ever been told that you) got out of bed and sleepwalked or performed other sleep-related activities? (Yes/No/do not know)
2. What activities did you perform during sleep? (open text)
3. Do you have any family members who have experienced sleep-related activities/parasomnias? (Yes/No/do not know)
4. Have you performed any dangerous or injurious activity during sleep? (e.g., climbing or jumping out through the window, leaving the house, falling from the bed, driving in sleep, etc.) (Yes/No/do not know)
5. Have you experienced any physical or mental adverse/traumatic experiences in your childhood? (Yes/No/do not know)
6. Would you like to describe them? (open text)

Statistical analysis

Microsoft Excel (164) was used for computing descriptive statistics, including mean ± standard deviation for quantitative variables and percentages for qualitative variables. We implemented a multicriteria-weighting procedure for correcting sample bias based on key sociodemographic variables. Pairwise marginal distributions for gender, age, education, and municipality type were utilized for weighting (165). Chi-squared tests assessed the relationship between responses and survey dates to identify response changes over time. Binary logistic regression assessed the association between parasomnias and variables, with chi-square tests and odds ratios (ORs) reported with 95% confidence intervals (CIs). Two scorers coded missing data and "do not know" responses ‘conservatively’ as "No". Logistic regression analyzed associations between sociodemographic characteristics and binary responses. A Hungarian-speaking sleep specialist (AS) coded the open responses using categories to standardize and enhance the reliability of the analysis. We coded activities as '1' for present (yes) and '0' for absent (no). Binary logistic regressions explored associations between activity types (e.g., sleepwalking) and explanatory variables. ORs were calculated for each explanatory variable (e.g., sex, age groups), representing the odds of specific sleep

behaviors in a group compared to the reference group. More significant disparities from an OR of 1 indicated stronger associations between variables. The finalized data were imported into SPSS version 27 for analysis. The STATA statistical package computed ORs and 95% CIs for each activity type. (StataCorp. 2019. Stata Statistical Software: College Station, TX: StataCorp LLC) (166).

Limitations of the survey methodology:

- The data were reliant on self-reports without medical validation or comprehensive data assessment, while patients may have amnesia of their episodes.
- Another issue is the high level of subjectivity in the definition of ACEs; however, the fact that someone considers something in their past as stressful or traumatic gives weight to those subjectively interpreted events or situations.

B) Data collection from YouTube videos on sleep-related behaviors likely to represent NREM parasomnias

Although often criticized, YouTube and other social media, as tempting data sources, have been increasingly used in medical education and research (167–170). Two investigators (VMC and AS) searched the terms “sleepwalking,” “somnambulism,” “sleep eating,” “sleep sex,” “sleep terror,” and “sleep talking” on YouTube (https://www.youtube.com) between January and July 2022 in six languages (English, French, German, Hungarian, Portuguese, and Russian), after the approval of Semmelweis University's Ethical Committee. We also searched for videos used in YouTube-based medical education. Some records contained more than one type of sleep-related activity, and we scored each activity type individually in these cases. We had to estimate the biological sex and age of those persons in the videos. We set up two sexes (M/F) and three age groups (child or adolescent ≤18 years; adult between 19 and 50 years; and older adults over 50 years).

A video record was excluded if the two investigators disagreed on the biological sex, age, video quality, type of record, and likely diagnosis. We excluded those videos we considered ‘bad’ or ‘poor’ quality, or apparently faked. We also excluded advertisement or entertainment videos, as well as the ones likely showing RBD or epileptic seizures, as assessed by good/reasonable discriminatory points provided by the literature. The differentiation between frontal lobe seizures and parasomnias may be challenging without historical information or the support of electroencephalography (123,171,172). We excluded those videos raising any suspicion of epilepsy: convulsive, dystonic, or dyskinetic movements; < 1 minute duration; abrupt onset; tonic limb extension; cramping, screaming; or non-coherent vocalizations (126). Evaluating a video record as one showing NREM parasomnia rather than an epileptic seizure is also supported by the prevalence difference between the two conditions. In an unselected sample of sleep-related motor events, as in a YouTube search, the chance of capturing a DOA episode is about a thousand times higher than capturing a frontal lobe seizure. This is due to the prevalence of frontal lobe epilepsy being assessed at 1.8/100,000 (173) versus about 2-4/100 of adult DOA episodes (4,113,120).

We excluded apparent RBD cases based on the violence, abruptness, and fragmentary character of movements: punching, kicking, jumping, and falling out of bed; screaming or laughing. Unlike NREM parasomnias, individuals with RBD have their eyes closed, may wake up quickly without confusion, and may vividly remember the enacted dreams (111). The types of sleep activities in each video were coded as ‘yes’ or ‘no’ for statistical analysis. We utilized binary logistic regression to assess the relationship between sleep-related behaviors and other variables, scaling and applying it to activity types. Some records showed more than one type of activity; each one was classified as either present or absent based on our self-made list of activities, as follows:

1. Ambulation (Sleepwalking)
2. Sitting in the bed, handling objects, getting dressed, packing, etc.
3. Dangerous activities, e.g., leaving the house or driving, handling electricity or sharp objects, etc.

4. Emotional behaviors, e.g., being scared, laughing, or crying
5. Talking
6. Eating in an apparent sleeping state
7. Engagement in sexual activities
8. Intense panic-like escape behaviors with motor and autonomic signs (possible sleep terror episodes)

Statistical analysis

Binary logistic regression assessed the association between NREM parasomnia activities, age, and sex groups. The odds ratio (OR) was calculated for each variable and represented the probability of specific sleep behaviors in a group compared to the reference group. We coded sleep behaviors as '1' for present and '0' for not present for each explanatory variable (e.g., sex, age groups). The OR value above 1 indicated the associations between variables analyzed by the STATA statistical package and reported with 95% confidence intervals (CIs) for each activity type (StataCorp, 2019, Stata Statistical Software: College Station, TX: StataCorp LLC) (166).

Limitations of the YouTube videos methodology:

- The phenomenology approach only, with no support from clinical reports, history, or investigations.
- Non-representative data, with selection bias as well.
- Cannot replace PSG-confirmed data for diagnostic or prevalence conclusions.

C) Experiences on adult parasomnias seen in the Sleep Clinic of the Institute of Behavioral Sciences, Semmelweis University, between 2018 and 2023.

A retrospective analysis of the patient records in the Seep Clinic of Semmelweis University database was performed using descriptive statistics. I present two illustrative

cases of sexsomnia, highlighting the phenomenology of this sleep disorder, problems of management, and diagnostics.

Limitations of the Sleep Clinic cases:

- Few cases of PSG-confirmed data for diagnostic or prevalence conclusions.
- Lack of patient follow-up**.**

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4 Results of the three studies

A) Data representing the Hungarian population in 2023

The data collection by the random walk method, quota criteria based on the participants' address cards, is shown in **Table 3**.

Table 3. Results of the survey participants' selection using a random walk method in a non-probability quota sample, N=1000

Survey population (random walk method)	**N**
Total of visits	1978
Total number of people who did not meet the random walk method criteria to represent the adult population of Hungary (this number was deducted from the total visits)	733
Total number of people who did not agree to the interview (this number was deducted from the total of visits)	245

Of the 1,000 participants, 70% were urban dwellers, and 30% lived in rural areas. The mean age was 48 years (SD: 16.75, range 18–90 years), 53% were females, and 50% had primary education (**Table 4**).

Table 4. Sociodemographic features versus NREM parasomnia-like activities in the population

	Population N=1000, n (%)	**NREM parasomnia- activities N=27, n (%)**

Gender		
Men	469 (46.9)	15 (3.2)
Women	531 (53.1)	12 (2.3)
Age		
19-30	181 (18.1)	10 (5.5)
31-	356 (35.3)	7 (2.0)
≥50	463 (46.3)	10 (2.2)
Education		
Primary	508 (50.8)	12 (2.3)
Secondary	315 (31.5)	9 (2.9)
Graduated	177 (17.7)	6 (3.4)
Residence		
Urban	695 (69.5)	9 (1.3)
Rural	305 (30.5)	18 (5.9)

The types of activities in the twenty-seven participants who reported apparent NREM parasomnia episodes are shown in **Table 5**.

Table 5. The proportion of sleep-activity types among parasomnias (N=27) and the whole population of 1000 persons representing population-prevalence values (N=1000)

Parasomnia types	**N=27 (%)**	**Population, N=1000, n (%)**
Sleepwalking	22 (81.5)	2.2
Sleep eating	1 (3.7)	0.1
Sleep talking	4 (14.8)	0.4
Sleep terror	-	-
Sleep sex	-	-

The types of adverse childhood experiences reported as open text are shown in **Table 6.**

Table 6. Types of reported adverse childhood experiences among the 27 DOA cases, N=9; and in the whole population of 1000 participants, N=92

Childhood adverse experiences	**Among DOA cases N=27, n (%)**	**In the population N=1000, n (%)**
Emotional and physical abuse (including sexual)	9 (33)	57 (62)
Dysfunctional household conditions (parental loss/divorce, orphanage, etc.)	0	35 (38)

Five persons (18%) of the 27 DOA participants reported having performed dangerous behaviors during the episodes. Types of dangerous activities during reported NREM parasomnia-like activities are shown in **Table 7**.

Table 7. Types of dangerous activities during reported NREM parasomnia-like activities, N=5 (18.5% of 27 DOA participants)

Dangerous activities	**N=5, n (%)**
Went to the window or the street	4 (80)
Eating in sleep (carrying the risk of consuming inappropriate or inedible substances)	1 (20)

A significant age difference in our binary logistic regression results (OR = 0.3, 95% CI [1.10-0.80], p = .03) showed that the probability of sleep-related activities likely consistent with DOA was lower in older than in younger adults. To check the age groups' decreasing 'age-slope', I calculated the prevalence percentages by age-decades as shown in **Table 8**.

Table 8. Prevalence of NREM parasomnias in Hungary by age groups (decades)

Age groups	Population by age groups, N:1000, n	NREM parasomnia, N:27 n (% of the age groups)
19-29	148	9 (6.1)
30-39	197	4 (2.0)
40-49	176	3 (1.7)
50-59	176	5 (2.8)
60	199	6 (2.0)

Twenty-six percent of those reporting sleep-related activities reported a family occurrence of DOA. The probability of DOA was also significantly higher (OR = 7.1, 95% CI [2.72–18.72], p < .001) in those reporting family cases of sleep-related behaviors compared to people with no DOA cases in the family.

The association of adverse childhood experiences was six times higher (OR: 6.2, 95% CI [2.53–14.96], p < .001) in people with reported DOA compared to those without.

B) Data collection from YouTube videos on sleep-related behaviors likely to represent NREM parasomnias

After excluding 551 videos (17 apparent RBD- and epilepsy cases, 534 poor quality-, likely faked, entertainment and advertisement records), 207 records have remained, likely consistent with NREM parasomnias (102 women, 68 children, 116 adults, and 23 older adults). Regarding the types of sleep activities among individuals included **(Table 9)**, the two scorers found a satisfactory level of agreement: 0.68 Cohen's Kappa coefficient at 0.39 to 0.96 (95% CI), SE 0.14.

Table 9. Ratios of sleep activity-types, likely consistent with NREM parasomnias, based on YouTube videos, N:207

Sleep activities*	N =207, n (%)
Sleepwalking	66 (31.8)

Sleep eating	25 (12.1)
Sleep talking	125 (60.4)
Sleep sex	3 (1.4)
Sleep terror	2 (0.9)

* If someone had multiple types of activity during the episodes (e.g., sleepwalking and sleep talking), those were calculated separately; this is why our sum is 221

Only two videos were found on sleep terror episodes, and we identified three videos related to sexual activity; these videos were parts of educational materials uploaded to YouTube.

The binary logistic regression results show that individuals over 50 exhibited significantly lower odds of sleepwalking than younger adults and children. Consequently, younger adults had significantly higher odds of performing dangerous activities than those over 50 years (**Table 10**).

Table 10. Results of logistic regression analysis to assess the associations between age and sleep activity types based on the 207 parasomnia-persons data in the YouTube collection (comparison age groups were compared to the reference group category: over 50 years old)

Activity during sleep	**Comparison age-groups**	**OR**	**95%CI**	**p-value**
Ambulation (SW)	0-16 y 18-50 y	5.14 6.33	1.10-23.9 1.39-28.7	0.037 0.017
Talking	0-16 y 18-50 y	0.42 0.35	0.05-1.12 0.71-1.69	0.08* 0.19*
Dangerous activities	18-50 y	8.14	1.04-64.1	0.046
SRED-like activities**				
Engagement in sex**				
Sleep terror**				

*Non-significant result **No statistical analysis due to the small number of cases

Although the small number of SRED cases did not allow for associating SRED with age or biological sex in this study, 75.8% of dangerous activities during NREM parasomnia-like activities were found to be SRED (**Table 11**).

Table 11. The distribution of dangerous activity types in the group of 33 persons performing dangerous activities (15.9% of the 207 videos) and in the whole parasomnia population of 207 persons (YouTube)

Dangerous activity-types	**'Dangerous' group, N=33, n (%)**	**Whole parasomnia population, N=207, n (%)**
The person went to the window or street (we saw a kid jumping and falling off the balcony while sleepwalking)	8 (24.2)	8 (3.86)
Ate in sleeping (possibly inappropriate substances)	25 (75.8)	25 (12.1)

C) Experiences on adult parasomnias seen in the Sleep Clinic of the Institute of Behavioral Sciences, Semmelweis University, between 2018 and 2023.

Over 5 years, between 2018 and 2023, 45 parasomnia cases were seen, and 36 of them were likely consistent with NREM parasomnias, based on clinical data and, in several but not all cases, video-polysomnography. Sleepwalking, sexsomnia, and sleep eating were noted. The small non-representative group does not allow drawing conclusions that would be valid for the population; however, we found the occurrence of parasomnia types in this highly selected patient group interesting (**Table 12**). Seven out of the 36 patients (19.5%) reported a family accumulation of DOA, and 13 (36.1%) of the

patients reported dangerous activities among NREM parasomnia-like activities (**Table 13**).

Table 12. Age groups versus types of NREM parasomnias in the Sleep Clinic of Semmelweis University between 2018 and 2023, N=36

Age groups	**19-39**	**≥50**	**N** (%)
Sleepwalking	25	6	31 (86.1)
Sleep eating	0	3	3 (8.3)
Sleep sex	1	1	2 (5.5)
Sleep terror	0	0	0
Total	27 (75)	9 (25)	

Table 13. The types of dangerous activities among NREM parasomnia-like activities in the sleep clinic population, N=13 (36.1% of the 36 cases)

Dangerous activity types N=13 (36.1%)	**N (%)**
Injury from breaking things: glass door, window	4 (30.8)
Hit someone	3 (23.1)
Fell out of the window	3 (23.1)
Ate inappropriate substances	3 (23.1)

A study of sleep clinic patients (176) had partially similar results on adult NREM parasomnias. In that study of 39 patients, the average age was 32.7 (17-56 years) at the first clinical consultation, and the occurrence of self-reported psychological trauma was 15%. In the Semmelweis Sleep-clinic, the first clinical consultation's age was similar (35 ±15.7) (19-89 years), while we had no data on ACE in our group. The same study's

reported frequency of sleep behavior types versus our clinic findings is shown in **Table 14.**

Table 14. Comparison of literature results and Semmelweis clinical data in percentages

Behavior	**Banerjee, D., & Nisbet, A. (176)**	**Semmelweis clinic**
Physical aggression toward someone	36 %	8.3%
Ate inappropriate substances (one fainted and had a shoulder dislocation)	5%	8.4%
Sexual activities	13%	5.5%

Case reports

I present two illustrative cases of sexsomnia, highlighting the typical behavior in a sexsomnia episode (case 1), as well as a case where alcohol use may have had a priming role in sexsomnia in a genetically predisposed person who had recovered from his childhood sleepwalking. Both patients had an irregular sleep-wake pattern with disturbed sleep.

Case 1

A 52-year-old married woman, a hospital nurse living with her husband and children, had been treated for high blood pressure. There was no DOA in her family.

She worked alternating day/night shifts, which resulted in irregular, disrupted sleep. She came to the clinic for recent symptoms (the last 2 years) of difficulty falling asleep and sleepwalking, associated with sexual activity occurring 2-3 times/week, about three hours after falling asleep. She screamed and slapped, sometimes fought, and scolded her husband and even her teenage son, using obscene words. Her husband reported that it was difficult or impossible to wake her up during the episodes, and she did not

remember them either, but sometimes she had some vague memory patches. Essentially, her husband informed her about the sleep-related sexual symptoms. After the episodes in the morning, she felt exhausted, unfocused, and in a depressed mood. She feared that this phenomenon could lead to the deterioration of her marriage by calling her previous partner's name in the episodes; also, she was humiliated and embarrassed by molesting his son. Still, she refused to have a polysomnography sleep study, possibly due to the awkwardness of her symptoms and fear of having them in the sleep lab. An EEG, after a whole night's sleep deprivation, has not revealed any epileptic symptoms. Her MRI brain scan revealed small vascular lesions consistent with vascular encephalopathy (Fazekas-grade-2). For finance-related issues, she refused to reschedule her working pattern but tried clonazepam 1 mg before bedtime. This had proved practically useless during the about 2-month follow-up period.

This late-onset case has demonstrated the complexity of dealing with such conditions, needing a multilateral, biological, and psychological approach, including her son's safety. Our case with potential marital consequences and frustration is similar to those in the literature (177). The irregular sleep-wake pattern and the vascular encephalopathy of our patient might have primed her events by deepening and disorganizing her sleep and decreasing her bearing capacity. Unfortunately, she refused further exploration (for clarifying an underlying sleep disorder, e.g., sleep apnea syndrome). Regular sleep could have helped, but she also refused this for financial reasons. The combination of sleepwalking, sleep talking, and sexual activities in her sleep might suggest the diagnosis of parasomnia overlap disorder (159). The late age of onset and the difficulty waking her up from the episodes have raised the distant possibility of an epileptic disorder, unsupported by the EEG after sleep deprivation.

Case 2

This 28-year-old metalworker came to the sleep clinic to discuss his legal issue about performing sex without the consent of his partner during sleep. His girlfriend, for being “raped” at night while he claimed to be asleep, reported him to the police. He had

amnesia from the episodes. To his knowledge, his episodes, witnessed by his girlfriend, started with leaving the bed, getting dressed, and turning on the light. Based on his girlfriend's report, his facial expression differed from usual. His activity continued sometimes with masturbation and full sex. Typically, the episodes occurred when he went to bed late and was tired. He had been sleepwalking as a teenager, and, for the last 4-5 years, at least 2-3 times a year, he had these episodes of sexsomnia. In the year he came to the clinic, it happened eight times: 2-3 hours after falling asleep, he "raped" his girlfriend in his sleep without ever being aware. He worked 12-hour days, drank alcohol regularly, and smoked. His medical history showed no head trauma, and he was on no medication. He slept well and woke up rested most of the time. There was sleepwalking in his family (his father). His polysomnography revealed mildly fragmented sleep; no parasomnia episode was captured. His non-enhanced MRI brain scan was normal. To treat his episodes, he decreased alcohol and tried to keep a regular sleep-wake pattern; in addition, he started taking Melatonin 2 mg, increasing it up to 6 mg. He had no more episodes during the 33-month follow-up time.

In this case, a sleepwalking person with a positive family history of DOA has experienced the reappearance of his childhood parasomnia in a modified form, possibly related to regular alcohol use and over-exhaustion. This symptom extension highlights the overlap and potential severity order of different types of DOAs, pointing also to the difference between childhood and adulthood manifestations. Lifestyle change and melatonin seemed to help during the nearly 3-year follow-up period. We have no information about the legal outcome of his case.

In both patients, parasomnias' priming factors - irregular sleep-wake pattern, alcohol, and abnormal brain imaging in one of them - were recognized. The interplay between genetic predisposition, shift work, lifestyle choices, emotional stress, and organic brain conditions can create a complex set for individuals experiencing sexsomnia.

D) Synopsis of results

The prevalence of NREM parasomnias, types, dangerous activities, and ACEs by the three data collection methods is shown in **Table 15**. We found an 'age slope' for children, adults, and older adults, with a decreasing prevalence of DOA, especially sleepwalking, by each method.

Table 15. Comparison of results by the three methodologies (N, percentages of DOA-types, and the occurrence of dangerous activities)

	Population survey	**YouTube-videos***	**Clinic data***
DOA persons (N)	27	207	36
Sleepwalking	22 (81.5 %)	66 (31.8 %)	31 (86.1 %)
Sleep eating	1 (3.7%)	25 (12.1%)	3 (8.3%)
Sleep talking	4 (14.8%)	125 (60.4%)	-
Sleep terror	-	2 (0.9%)	-
Sexsomnia	-	3 (1.4%)	2 (5.5%)
Dangerous activities	5 (18.5%)	33 (15.9%)	13 (8.3%)

*The data from these studies are not representative.

The logistic regression on the age slope on YouTube videos methodology was calculated for comparison with the survey methodology. The survey methodology did not include children in its database. The probability of sleep-related activities likely consistent with DOA was lower in older than in younger adults (OR = 0.3, 95% CI [1.10-0.80], p = .03), which is the same result found in the YouTube videos methodology. The age slope for NREM parasomnias was found in children (OR = 5.14, 95% CI [1.10-23.9], p = .037) and adults (OR = 6.33, 95% CI [1.39-28.7], p = .017) when compared with older adults. It means that both children and adults have an association with NREM parasomnias. In this case, a large sample (YouTube videos) provided more examples than a single sleep clinic could collect. In this view of potential alternative diagnoses (epilepsy, RBD, and videos that disagreed on the type by

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the two data collectors were excluded), it seems justified to believe that the remaining cases were really consistent with NREM parasomnias.

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5 Discussion

Due to the variability of examined patient populations, data collection methods, and population sizes, resulting in inconsistent data on NREM parasomnias in the literature and the many unanswered questions related to their mechanism, we felt that performing a study of DOA was justified. We used three data-collection methods to balance methodological issues and provide an accurate picture.

Because the random walk selection method in the population survey ensured that the 1000 interviewed participants represented the adult population of Hungary in terms of age, sex, education level, and habitat, to our knowledge, our study is the first representative survey on NREM parasomnia-like activities in Hungary.

We found a 2.7% prevalence of adult DOA, similar to 2-4% of international data (71,99). The most frequent DOA (by each method we used) was sleepwalking; its 2.2% prevalence we found was also similar to international studies (4,101), such as 1% in the USA (178), 1.7% in France (4), and 2.8% in Brazil (176).

Our single interviewing method did not allow for following up or scrutinizing other predisposing, priming, and precipitating factors, or the participants' outcomes. We could not gain more detailed data than the answers to the open-text questions. Possibly due to being interviewed by non-medical staff as well as due to the person's amnesia or patchy memory of the episodes, no people with sleep terror or sexsomnia episodes have been detected in the population survey. Similar is the case in the literature about sexsomnia, providing scarce prevalence data (179–181). A Norwegian population survey (4) selecting the interviewed people randomly (following the birthday method) found the lifetime prevalence of "sexual act during sleep" at 7.2%, with its current prevalence at 2.7%.

While a hint of disbelief has surrounded sexsomnia, its existence has been evidenced by both clinical and polysomnographic methods and experiences (179). It can lead to multilateral consequences, including forensic, psychological, and health-related ones. The challenging legal issue of sexsomnia as a defense in sex crimes is impressively detailed in the paper by Mohebbi A, Holoyda BJ, Newman WJ (182).

Sexsomnia needs to be differentiated from other automatic sexual activities such as ‘wet dreams’ (a normally occurring ejaculation during sleep, typically linked to erotic dreams) and sleep-related painful erections, as well as from the hyper-sexuality of Kleine-Levin syndrome and the rare cases of epilepsy, where seizures manifest sexual behaviors (89).

Cases in the literature, as well as our cases, call attention to the association of sexsomnia with other NREM parasomnias and the priming effect of sleep deprivation and alcohol (183). In addition to lifestyle changes, clonazepam and melatonin have sometimes proved helpful in treatment.

To our knowledge, there are just two surveys in the literature on the prevalence of SRED. One is a non-representative one for the population, targeting in- and outpatients with eating disorders, obese patients, depressed persons, college students, and psychiatric patients (184). In that study, the prevalence of SRED was 5%; 16% of inpatients with eating disorders and 8.6% of outpatients with eating disorders, suggesting a strong link between SRED and the severity of eating disorders. In another study using the ‘next birthday method’, the lifetime prevalence of SRED was 4.5%, and its current prevalence was 2.2% (4).

Our representative population survey found substantially fewer SRED cases in the global population - 0.1% -, but among DOA persons, it was similar to the literature, at 3.7 %. Highlighting the effects of methodologies, 12.1% of SRED cases were seen in our YouTube study (effect of curiosity? extraversion?), and SRED occurred in 5% of persons presenting with NREM parasomnias in the sleep clinic (complex and dangerous cases accumulating there).

SRED is often overlooked and underdiagnosed compared to eating disorders, while the relatively frequent co-occurrence of the two groups may suggest partially common roots. In addition, while overlooked or considered of subsidiary importance, the prevalence of SRED, even at 0.1% in our representative study (and much higher in other studies), approached the prevalence of bulimia nervosa (0.3%) and anorexia nervosa (0.6%) in adults (185). It is important to distinguish SRED from night eating syndrome (NES), which occurs in fully awake individuals who consume large amounts of calories (50% of the 24-hour intake, mostly carbohydrates) at night (87,186), while

those with SRED eat or drink while being fully or partially asleep. They may consume substances unfit for food (which never happens in NES), face dangers involved in food preparation, consumption of hazardous substances (187), poisoning, and unexplained weight gain (188). Certain medications, smoking, substances of abuse, or even withdrawal from addictive substances can prime it (3, 190); Zolpidem and Quetiapine are the best-known ones (190). Stopping those drugs may instantaneously 'cure' the condition.

Since little attention is given to SRED by patients and the medical community, collecting data on it may help call attention to, understand, and treat it. Presently, the first-line treatment of SRED is '*selective serotonin reuptake inhibitors' at 20-30 mg/day, or Topiramate at 100-300 mg/day, or Clonazepam at 0.5-2.0 mg/day'* (191). SRED may benefit from treating the associated sleep disorders, especially RLS (87).

The same dosage of clonazepam (0.5-2.0 mg/day) is also the first-line pharmacotherapy for sleepwalking and sleep terror, stimulating the GABA-A receptor, inhibiting dopamine cell bodies directly (210). As dopamine is important for the transition between sleep stages (particularly the transition into REM), a delay in this by dopamine antagonism could keep the person in NREM sleep longer, which can also cause NREM parasomnias in some patients (211). Dopamine antagonists could also upset the balance between muscle atonia in REM and the dream state (212). In the literature, some case reports have described successful treatments of NREM parasomnias with SSRIs due to the SSRIs' long-term serotonergic modulation, which has been proposed to stabilize brainstem circuits involved in sleep–wake transitions, potentially reducing incomplete arousals that contribute to NREM parasomnias (214).

This way, medications like benzodiazepines or certain antidepressants, which are prescribed to reduce episodes, may disrupt normal sleep architecture, suppressing the REM sleep phase, thereby increasing the likelihood of NREM parasomnias in some individuals, especially in cases of family history (205,215).

Our first research, the population survey, has also supported the family history (related to the hardly known genetic factors) on adult DOAs.

Here, 26% of those adults reporting DOA had a family history of NREM parasomnias, which contrasts with the literature that reports 10 times the risk of developing NREM parasomnia in kids having DOA in their family compared to controls (134). This inconsistency might be related to patient selection; child cases likely targeted by the Pressman study have more genetic determination than adult cases, as in our study.

Our results have revealed that a historic ACE increased the odds of DOA by six times (a statistically significant finding) compared to those not reporting ACEs: nine patients (33%) of the 27 persons reporting DOA reported emotional and physical abuse consistent with ACEs. This finding accords with the literature highlighting a correlation between ACEs and undesired sleep-related behaviors (148,192,193), suggesting that childhood traumatic experiences as potential priming factors might cause permanent psychological and, possibly, related neurobiological changes leading to NREM parasomnias (194,195).

There are numerous aspects linking adversities, mood disorders, and sleep disorders. Barclay's research indicated that early-life adversity experienced by mothers was associated with depressive symptoms during pregnancy, which in turn were linked to reduced cortisol reactivity in infants, causing sleep disturbances (196).

Taken together, the 26% presence of family accumulation and the 33% of historic ACE in our DOA-reporting participants suggest that inheritance and early adversities may contribute to developing adult NREM parasomnias, neither factor having a determinant role. It could be raised whether familial and ACE-related cases represent the same phenotype (199).

Our population survey participants reported a remarkably high (18.5%) association with dangerous activities related to DOA, and about one-third of sleep-clinic patients suffered severe injuries by falling out of the window, running away at night, leaving the house, consuming cleansing products, etc. In the YouTube study, we found a record of a child jumping and falling out of the balcony in a sleepwalking episode. Given these data, we feel that the general belief that DOAs are harmless and “innocent” conditions, especially in adults, needs to be revised.

Each of our studies has confirmed the known age distribution- a decrease with age, the age-slope, of NREM parasomnias.

In children, DOA episodes occur when deep NREM sleep with peak slow-wave activity "meets" arousal forces (34,201), during the transition from deepening to rising of the first NREM sleep cycle (197). Based on a genetic predisposition and sometimes triggered by internal or external sleep-disturbing factors, this may lead to a dissociation between sleeping consciousness and waking motor activity (37).

In adults and the elderly experiencing somnambulism, external triggers seem more influential than genetic factors (198), as also suggested by our survey's relatively low proportion of adult familial cases. In these age groups, sleep is shallower and more fragmented; sleep-fragmenting factors elevate homeostatic sleep drive. Thus, sleep-wake transitions and 'collisions' of sleep and wake-promoting forces occur in more superficial sleep stages than in childhood (79).

This disparity of DOA episodes' position within sleep might involve potentially distinct mechanisms of sleep dissociation in children and adults. In turn, this may result in differing clinical manifestations of sleepwalking in children (typically harmless with amnesia) compared to adults/elderly (often violent, fierce, and accompanied by vivid dreams and partial recollection) (7, 204).

Therefore, while genetic factors are involved in the development of parasomnias (200,201), it is essential to consider priming and precipitating factors, especially in adult cases. Avoiding them may help prevent strategies and provide therapeutic approaches for those affected by DOA.

5.1 General Limitations

A limitation arises from the survey data without medical validation or comprehensive data assessment, while patients may have amnesia of their episodes. We could not check priming and precipitating factors in our population and YouTube studies. The YouTube methodology faces bias due to its use of a phenomenological strategy with video evidence only, devoid of demographic, polysomnographic, or historical context, which

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would help classify the episodes seen. A significant limitation was the small population size that characterized each methodology.

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6 Conclusions

Our study reveals that adult NREM parasomnias, as relatively frequent conditions, may be linked to lifestyle, medical, psychological, and substance (alcohol and drugs) – related issues. These conditions carry significant hazards and may compromise the quality of life of those affected.

In our study, which was conducted from a representative population survey in Hungary, the prevalence of adult sleepwalking was similar to that found internationally.

Our study seems to be one of the few representative surveys on the prevalence of SRED (affecting 0.1% of the population). As a usually forgotten or overlooked condition not included in the group of eating disorders, it needs awareness and attention. Clinicians need to know provoking drugs, especially psychoactive ones.

The remarkably high occurrence of sexsomnia in our retrospective clinical study highlights that this kind of DOA exists, and it may have a higher prevalence than expected. Naturally, identifying this sensitive phenomenon requires confidential medical exploration. It needs to be considered in certain forensic cases of sexual crime.

In our studies, the rate of dangerous behaviors was higher than in most international studies; we found significant risks of injury with each methodology. By acknowledging this and implementing appropriate precautions, affected individuals could be protected, and risks associated with sleep-related activities mitigated; our results underscore the importance of safety measures and risk management.

The 33% proportion of DOA-like individuals reporting adverse childhood experiences supports the possibility of traumatic experiences' long-term neurobiological impact on the genesis of NREM parasomnias.

The family accumulation affecting about one-quarter of adult DOA persons suggests that inheritance has less impact in this age group than in children.

Whether ACE-related and familial cases manifest the same phenotypes needs further study.

Our results have confirmed the known age-slope of sleepwalking; it is most prevalent in children and has a decreasing rate in adults and further in the elderly. This finding may be considered in theoretical considerations both on the mechanism and the potential

divergence of childhood and adult NREM parasomnias. We still need to make considerable progress in uncovering the causes of NREM parasomnias, including this age association.

Our new findings were the following:

- To our knowledge, this is the first representative study on adult parasomnias in Hungary, finding prevalence rates of sleepwalking similar to most international data.
- We found a considerably lower prevalence of SRED than the scarce international data did. We found its prevalence at 0.1% of the population, similar to the prevalence of anorexia nervosa. Therefore, SRED needs awareness and attention, as well as a potential side-effect of several psychoactive medications, especially zolpidem and quetiapine.
- The high occurrence (5.5%) of sexsomnia reported in our sleep clinic cohort is similar to that of other sleep clinics presented in the literature. It suggests a higher prevalence in the population than expected. It needs awareness and also to be considered as a medico-legal issue in certain sexual crimes as an NREM parasomnia-related automatism.
- We found a high occurrence of dangerous behaviors during sleep-related episodes in each study, highlighting that adult parasomnias carry important risks of injury.
- Having DOA was six times more probable (OR = 6.2; 95% CI [2.53–14.96], $p < .001$) in those reporting ACEs than those not, highlighting the potential harm of childhood adversities in developing adult parasomnias.
- The high occurrence of reported childhood adversities in the population survey requires attention in childcare.

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7 Summary

Adult NREM parasomnias present highly variable clinical forms and often cause injuries. Identifying typical phenotypes may help in the risk management of sleep behaviors that characterize parasomnias in different age and sex groups. This research has three parts. First, a cross-sectional representative population survey in Hungary was conducted on the frequency of NREM parasomnias and their types, DOAs' family accumulation, association with adverse childhood experiences (ACEs), and related risks. Secondly, a YouTube video-based study on sleep-related behaviors likely representing NREM parasomnias to find phenotypes in different age and sex groups. The last study was a retrospective clinical evaluation.

One of the most important findings was the association between NREM parasomnias and ACEs. The prevalence of NREM parasomnias in Hungary was 2.7%, including 0.1% of SRED, approaching the prevalence of anorexia nervosa. SRED was reported by 3.7% of DOA persons. The high (5.5%) occurrence of sexsomnia among sleep-clinic patients calls attention to this intriguing, dangerous, and frustrating condition with potential forensic implications. Currently, there are no large randomized controlled trials for managing NREM parasomnias. Any future studies should consider predisposing, priming, and precipitating factors to find effective treatments for preventing the episodes.

In summary, this thesis provides novel insights into the prevalence, phenomenology, promoters, family accumulation, and risks of injury of adult NREM parasomnias.

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9 Bibliography of the candidate's publications

Miranda Correa Vivian, Biresaw Mengesha Srahbzu, Vitrai Jozsef, Szűcs Anna

NREM parasomnia-related behaviors and adverse childhood experiences

SLEEP MEDICINE 121 pp. 365-369., 5 p. (2024)

Publication: 35162222 | Journal Article (Article) | Scientific

Scopus - Medicine (miscellaneous) Rank: Q1

IF: 3,8

Miranda Correa Vivian, Vitrai Jozsef, Szűcs Anna

Parasomnias manifest different phenotypes of sleep-related behaviors in age and sex groups. A YouTube-based video research highlighting the age slope of sleepwalking

JOURNAL OF CLINICAL NEUROSCIENCE 122 pp. 110-114., 5 p. (2024)

Publication: 34433970 | Journal Article (Article) | Scientific

Scopus - Medicine (miscellaneous) Rank: Q2

IF: 1,9

Miranda Correa Vivian, Arruda Giseli Cristina da Costa, Szűcs Anna

Parasomnias patients and risk of injury, a 16-years clinical data study

Sleep Epidemiology 3 Paper: 100057, 3 p. (2023)

Publication: 33950069 | Journal Article (Note, Short, Rapid communications) | Scientific

DOI:10.14753/SE.2025.3219

9.1 Publications not related to the thesis, co-authored by the candidate

Biresaw Mengesha Srahbzu, Vitrai Jozsef, Halasz Peter, **Miranda Correa Vivian**, Szűcs Anna

Changes in public attitude toward epilepsy in Hungary since 1994. A multicriteria weighting analysis

EPILEPSIA OPEN 9: 3 pp. 1042-1050., 9 p. (2024)

Publication: 34774937 | Journal Article (Article) | Scientific

Scopus - Neurology (clinical) Rank: Q2

Journal subject: Scopus - Neurology Rank: Q2

IF: 2,8

∑IF: 8.5

9.2 Congress Abstract Speaker

Miranda Correa Vivian, Szűcs Anna

Parasomnias in Hungary. A high association with adverse childhood experiences

INTERNATIONAL JOURNAL OF PSYCHOLOGY 59: Suppl. pp. 206-206. Paper: 4305, 1p. (2024), Publication: 36000942 | Journal Article (Abstract) | Scientific

Miranda Correa Vivian

Wearable Devices Could Reduce the Risk of Injury in Parasomnia Phenotypes

PALLIATIVE MEDICINE 37: Suppl.1, EAPC2023 Abstract Book pp. 76-76. Paper: P 1.042,1 p. (2023) Publication: 36001042 | Journal Article (Abstract) | Scientific

DOI:10.14753/SE.2025.3219

Miranda Correa Vivian, Szűcs Anna

THE PHENOMENOLOGY OF PARASOMNIAS BASED ON INTERNET DATABASES

SLEEP MEDICINE 100 pp. S190-S190., 1 p. (2022)

Publication: 35640060 | Journal Article (Abstract) | Scientific

Miranda Correa Vivian, Szűcs Anna

The phenomenology of parasomnias based on internet databases

JOURNAL OF SLEEP RESEARCH 31: Suppl 1 Paper: P032, 1 p. (2022)

Publication: 35640085 | Journal Article (Abstract) | Scientific

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