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Prevalence, incidence, evolution and associated factors of sleep paralysis in a longitudinal study of the US general population

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ABSTRACT

Background and objective: Sleep paralysis is a common phenomenon which causes and consequences are seldomly studied. The aim of this study was to investigate the incidence and prevalence of sleep paralysis (SP) in the American adult population and its evolution on a 3-year period.
Methods: This longitudinal study was conducted between 2002 and 2015 and included a representative sample of the US general population. A total of 12,218 subjects were initially interviewed (W1) and 10,931 were re-interviewed three years later (W2). The subjects participated in telephone interviews using the Sleep-EVAL expert system. Each interview lasted for about 1 h. SP episodes were assessed according to their frequency and duration.
Results: At W1, 9.7% (95%CI: 9.1%–10.3%) reported having ≥1 episode of SP in the previous year. At W2, 15.1% (95%CI: 14.4%–15.8%) reported SP. A total of 29.9% of subjects with SP at W1 still reported episodes at W2. The 1-year incidence was 2.7% (95%CI: 2.4–3.0%). After adjusting for age and sex, prevalent SP (i.e., present at W2) was predicted by age and race and the following factors present at W1: major depressive disorder, pain, hypersomnolence, cataplexy, hypnagogic and hypnopompic hallucinations, posttraumatic stress disorder, a reduction in sleep duration of ≥60 min, and the use of analgesic/antipyretic medication. Incident SP (i.e. new cases at W2) had similar predictive factors.
Discussion: Episodes of SP are frequent in the general population. Its persistence is predicted by several factors associated with narcolepsy like hypersomnolence and cataplexy but also by other factors like posttraumatic stress disorder or pain.

1. Introduction

Sleep paralysis (SP) is a parasomnia disorder that results in the unpleasant experience of being unable to move at sleep onset or upon awakening. It occurs when Rapid Eye Movement (REM)sleep intrudes in the wake state. In REM sleep, there is a muscle safety lock which prevents an individual of acting his dreams. An episode of SP is sometimes accompanied with vivid hypnagogic (at sleep onset) or hypnopompic (upon awakening) hallucinations which are also REM intrusions into the wake state [1]. SP can last for several minutes, and sometimes a person can stop the attack by making eyeball movements [2,3]. SP is also a common phenomenon in narcolepsy and it was considered until recently as one of its associated symptoms. The disorder can also occur in isolation and is called isolated SP1.

Few epidemiological studies in the general population have been performed on SP but the question of its incidence has never been addressed [4,5]. Various studies have been conducted to investigate the prevalence of SP in specific population like student samples [4,5]. The first epidemiological study conducted by Ohayon et al. on SP found about 6% of the general German and Italian population had experienced SP at least once in their lifetime [6]. A systematic review of 35 different studies found that the lifetime prevalence of SP in the general population was estimated at approximately 8%, ranging from 2% to 60% [5]. However, one of the reasons for this wide range is the lack of standardized measures for identifying and measuring sleep paralysis.

Various types of risk factors for SP have been identified which include, among else, daily alcohol use, smoking, stress and trauma, urbanization, hereditary factors, etc. SP can be associated with hypertension [5], epilepsy [7] and with psychological factors such

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as decreased overall sleep, fear, severe anxiety, or fear of imminent death [8,9]. However, in other studies the association between many of these risk factors and SP was not observed [4].

Therefore, more epidemiological studies in the general population are needed to identify the factors affecting SP. Identifying these risk factors can play an important role in designing interventions to improve mental health and quality of life of affected populations. To date, there is no longitudinal information on SP. Its incidence is unknown. This study aims to determine the prevalence and incidence of SP in the US general population as well as the risk factors associated with its occurrence using a longitudinal design study of the general population.

2. Methods

2.1. Data source and population

Study data were collected over telephone using an AI-empowered survey system Sleep-EVAL [10,11] on a comprehensive list of topics, aiming to evaluate sleep, pain, psychiatric disorders and medical conditions. The study was approved by the Institutional Review Board of Stanford University.

The target population was all non-institutionalized individuals aged 18 years and older living in the USA. A representative sample of the general population was drawn from 8 States (California, New York, Texas, Pennsylvania, Arizona, Colorado, Oregon, and Idaho). The study period (April 2002–June 2015), included two waves of recruitment and data collection: the first interview wave (W1) was from April 2002 to June 2012, and the follow-up interview (W2) started in January 2006 and finished in June 2015. Participants lost in follow-up were two times more likely to be younger than those who participated in the second wave (OR:2.0 95% CI:1.7–2.4; $p < 0.001$). Presence of a medical condition or a psychiatric disorder were not related to lost in follow-up with the exception of Major Depressive Disorder (OR:1.3; 95% CI:1.1–1.7; $p < 0.05$).

2.2. Sampling method

A two-stage design was used to draw the sample. First, the population was divided into geographic strata based on official census data. Telephone numbers were subsequently randomly pulled according to this stratification. Second, a controlled selection method was applied to limit the within-sampling unit non-coverage error. Therefore, one member per household was selected for interview using the Kish procedure [12].

An add-a-digit sampling, that is, increasing the last digit of a number by one, was employed to avoid the bias created by unlisted phone numbers [13]. Subjects who do not want to participate upon the first contact were called back a second time. Those who state upon the first contact that they do not want to be called back or do not want to participate in any form of inquiry were discarded and tabulated as refusal. Selected phone numbers were dialed for at least ten times at different hours of the evening and on different days, including weekdays and weekends, before being replaced on account of no answer.

2.3. Exclusion criteria

Interviewers first explained the goals of the study to potential participants before requesting verbal consent. Respondents were given the opportunity to call a member of the research team if they wanted further details before deciding to participate. Subjects who were younger than 18 years of age, who did not speak English or Spanish, who suffered from a hearing or speech impairment that precluded the possibility of an interview, or who had an illness (physical or mental like Alzheimer's or dementia) that precludes the possibility of an interview, were excluded from the study.

2.4. Survey system

Interviewers used the Sleep-EVAL knowledge-based expert system [10,11]. This artificial intelligence tool is specially designed to administer questionnaires and conduct epidemiological studies. The system is composed of a non-monotonic, level-2 inference engine, two neural networks, a mathematical processor, the knowledge base, and the base of facts. The interview began with a series of questions asked of all the participants. It includes, in order of: sociodemographic information, sleep/wake schedule, sleeping habits, sleep disturbance symptoms, medical and paramedical consultations and hospitalizations in the last 12-month period, medical conditions, use of prescribed and non-prescribed drugs, health quality assessment scale, alimentation, fatigue scale, pain questionnaire, vital statistics (e.g., height and weight). Once this information was collected, the system began the diagnostic exploration of sleep and/or mental disorders. On the basis of responses provided by the subject, the system formulated an initial diagnostic hypothesis that it attempted to confirm or reject by asking supplemental questions or by deductions. Concurrent diagnoses were allowed in accordance with the DSM-IV text revision [14] and the International Classification of Sleep Disorders (ICSD-2) [15]. The system terminated the interview once all diagnostic possibilities were exhausted. On average, interviews lasted on for 1 h 04 (±32.47 min), with a minimum of 19 min and a maximum of 3 h 50. Interviews could be completed in two or more calls.

2.5. Key variables

Items related to sleep paralysis included, frequency, age when the first episode occurred and moment when the episode occurred (upon awakening or at sleep onset). Participants were asked if they ever found themselves unable to move (like if they were paralyzed) when 1) falling asleep for the night of for a nap; 2) waking up in the morning; 3) waking up from a nap. For each situation, the participants answered on a 8-point frequency scale ranging from never to every day.

Hypersomnolence was defined as periods of excessive sleepiness despite a normal sleep duration occurring at least three times per week for at least 3 months.

Cataplexy was defined as episodes of muscle weakness triggered by a sudden strong emotion.

Chronic pain was defined as a pain occurring nearly every day for at least three months.

2.6. Analyses

A weighting procedure was applied to compensate for disparities between the initial sample (W1) and the standard population with respect of age, gender and geographic distributions. Results are presented with weighted percentages and 95% confidence intervals when appropriate. Bivariate analyses were performed using chi-squared or ANOVA tests depending on variable types. Predictors for sleep paralysis were identified through logistic regressions. Variables entered into the models were chosen according to their relevance and their significance in bivariate analyses. The cut-off point of entry (PIN) was set at .15 and the cut-off point of exclusion (POUT) at 0.10. IBM SPSS Statistics, version 27 was used to performed the analysis.

3. Results

A total of 10,931 individuals answered to the first interview and

to the follow-up about three years later. The sample was comprised of 53.7% of women. Caucasian represented 70.9%. The mean age for the sample at the first interview was 52.13 years (SD ± 17.21) and the mean Body Mass Index (BMI) was 27.87 kg/m^2 (SD ± 5.84). At follow-up, the mean age for the sample was 54.78 years (SD ± 17.22) and the mean BMI was 28.18 kg/m^2 (SD ± 5.95). About half of the sample (57.8%) was married or living with a domestic partner at the initial interview (57.3% at follow-up). Most of the participants finished high school (93.2% at the initial interview and 93.9% at follow-up).

3.1. Prevalence, incidence and evolution of sleep paralysis

At the initial interview, 9.7% (95% Confidence Interval (C.I.) 9.1%–10.3%) of the sample (n = 1060) reported at least one episode of SP in the previous year. Episode occurred weekly for 0.6% (n = 66) of the sample and monthly for 2.7% (n = 295). At follow-up, 15.1% (95% C.I. 14.4%–15.8%) of the participants (n = 1651) had at least one episode in the previous year. Weekly episodes were reported by 0.7% (n = 77) and monthly episode by 4.0% (n = 437).

The one-year incidence was 2.7% (95% CI: 2.4%–3.0%) in the sample.

Overall, 29.9% of the participants with SP at the initial interview still reported episodes at follow-up (2.9% of the sample): 6% reported episodes occurring at least once a week and another 31.4% reported episodes occurring at least once a month but less than weekly. Overall, 52.9% did not reported a change in frequency of the episodes, 21.3% reported a decrease in the number of episodes at follow-up and 25.8% reported an increase of episodes at follow-up.

As seen in Table 1, prevalence of SP was comparable between men and women and was unrelated to the education level at both interviews. Prevalence significantly differed with age being lower among the 55 years and older at the initial interview. At follow-up, the prevalence peaked among participants aged between 35 and 54 years. Sleep paralysis was higher at both interviews among individuals who were Black, of other race alone and of 2 or more races compared to Caucasians.

3.2. Description of the sleep paralysis episodes

At the initial interview, 7.2% (95% C.I. 6.7%–7.7%) of the participants reported that SP occurred in the morning, 4.0% (95% C.I. 3.6%–4.4%) at sleep onset and 4.3% (95% C.I. 3.9%–4.7%) upon awakening from a nap. At follow-up, SP when awakening in the morning was found for 8.1% (95% C.I. 7.6%–8.6%) of the sample, when falling asleep in 7.0% (95% C.I. 6.5%–7.5%) and when awakening for a nap in 3.4% (95% C.I. 3.1%–3.7%).

Episodes of SP started during childhood for 15.1% of cases, during adolescence for 50.1%, between the age of 18–40 years for 19.5% and after 40 years of age in 15.3% of cases.

The last episode occurred within the last 24 h prior the interview in 20.5% of the cases, within the last week in 27.9% of the cases, within the last month in 22.0% of the cases, within the last year for 3.5% and more than one year ago for 26% of the cases.

3.3. Predictive factors for sleep paralysis at W2

Predictive factors entered into the models were age, gender, race, BMI, alcohol intake, fatigue, presence of medical conditions, pain, insomnia disorder, hypersomnolence, cataplexy, hypnagogic and hypnopompic hallucinations, mood disorders, anxiety disorders, medication intake by class of medication (antidepressants, anxiolytics, hypnotics, antihypertensive medications, analgesics, etc). Two multivariate models were calculated: one was to assess

Table 1
Prevalence of sleep paralysis at least once in the previous year by sociodemographic characteristics.

	N	Prevalence at W1	N	Prevalence at W2
		% (95% CI)		% (95% CI)
Gender				
Male	5057	9.0 (8.2–9.8)	5057	15.1 (14.1–16.1)
Female	5874	10.3 (9.5–11.1)	5874	15.1 (14.1–16.1)
Age groups				
18-24	625	9.4 (7.1–11.7)	435	6.2 (3.9–8.5)
25-34	1172	13.3 (11.4–15.2)	915	13.9 (11.7–16.1)
35-44	2170	10.8 (9.5–12.1)	1923	22.4 (20.5–24.3)*
45-54	2252	12.3 (10.9–13.7)	2302	22.6 (20.9–24.3)*
55-64	1836	7.6 (6.4–8.8)*	2039	14.6 (13.1–16.1)
≥65	2876	6.8 (5.9–7.7)*	3317	20.2 (18.8–21.6)
Marital status				
Single	2285	11.6 (10.3–12.9)	2459	16.0 (14.6–17.4)
Married/domestic partner	6318	9.4 (8.7–10.1)	6263	16.0 (15.1–16.9)
Separated/Divorced	1126	9.8 (8.1–11.5)	874	20.1 (17.4–22.8)
Widow	1202	8.1 (6.6–9.6)*	1334	11.6 (9.9–13.3)*
Education level				
Less than high school	743	9.5 (7.4–11.6)	667	11.8 (9.4–14.2)
High school	2394	9.4 (8.2–10.6)	2263	12.1 (10.8–13.4)
Some college, no degree	3432	11.3 (10.2–12.4)	3673	18.6 (17.3–19.9)
Graduate	3236	8.7 (7.7–9.7)	3028	16.7 (15.4–18.0)
Postgraduate	1126	9.2 (7.5–10.9)	1301	14.8 (12.9–16.7)
Race				
American Indian	77	18.2 (9.6–26.8)	77	9.1 (2.7–15.5)
Asian	557	12.9 (10.1–15.7)	557	20.2 (16.9–23.5)
Black	1137	19.4 (17.1–21.7)*	1137	23.2 (20.7–25.7)*
Other race alone	1071	13.3 (11.3–15.3)*	1071	19.7 (17.3–22.1)*
2 or more races	339	14.8 (11.0–18.6)*	339	24.6 (20.0–29.2)*
White alone	7750	9.0 (8.4–9.6)	7750	13.4 (12.6–14.2)
Total	10931	9.7 (9.1–10.3)	10931	15.1 (14.4–15.8)

W1: Initial interview; W2: follow-up interview.
*p < 0.001.

relative risks of having SP at follow-up (Table 2); the other model was to estimate the relative risks of having incident SP at follow-up (i.e., present only at follow-up) (Table 3).

As seen in Table 2, prevalent SP at follow-up was predicted by age (being aged between 25 and 34 y.o.) and race (being black, other race alone or two or more races). Among sleep-related predictors, hypersomnolence at the initial interview (remitted) and hypersomnolence reported at both interviews (persistent). Cataplexy (incident and persistent) were strong predictors of prevalent SP. As seen in Table 2, 63% of individuals with persistent cataplexy and 36.8% of those with incident cataplexy reported SP. Hypnagogic hallucinations at follow-up (incident) or persistent hypnagogic hallucinations, hypnopompic hallucinations (remitted, incident and persistent) and a sleep duration shortened by at least 1 h at follow-up also predicted SP. Medical conditions were not predictive of SP but incident and persistent chronic pain as well as the presence of remitted, incident and persistent cataplexy were predictive of prevalent SP. Among psychiatric disorders, only major depressive disorder and posttraumatic stress disorder (PTSD) at the initial interview predicted SP.

Incident SP had mostly the same predictive factors than prevalent SP with some variations: only remitted hypersomnolence, remitted and persistent cataplexy and incident and persistent hypnopompic hallucinations predicted incident SP. PTSD at the initial interview was no longer significantly associated with SP while insomnia disorder at the initial interview appeared to be a protective factor for incident SP (Table 3).

4. Discussion

Sleep paralysis is a common type of parasomnia occurring at sleep onset or upon awakening. The main results of this study are summarized as follows:

The one-year incidence of sleep paralysis was 2.7% in the population. Risk factors for both the prevalence and incidence of sleep paralysis include younger age, black race, chronic pain, major depression, and lower body mass index. In the present study, both sexes showed almost the same pattern for both the prevalence and incidence of sleep paralysis.

The prevalence of sleep paralysis in the first and second measurements were 9.7% and 15.1%, respectively, which is higher than previous studies in the general population. In a meta-analysis of 18,330 people out of the general population, the prevalence of sleep paralysis was 7.6% [5]. The prevalence was also higher than in Germany and Italy study. However, the severity of sleep paralysis in the Ohayon et al.'s study was approximately the same as in the present study [6]. There are several reasons for this discrepancy. Race and ethnicity are one of the factors affecting the prevalence of sleep paralysis. In the present study, nearly 20% of the participants were black. Black or African American race has been reported in some studies with a higher a higher prevalence of sleep paralysis [16,17]. Perhaps the reason for these differences is due to their experience with sleep paralysis and cultural or spiritual beliefs. More than half of African Americans surveyed by Friedman et al. [18]. shared experiences of sleep paralysis with spiritual or cultural beliefs (such as being a Christian and being angered by the devil). Other influential factors include stress levels and socioeconomic status. Some African Americans may experience long-term stressors due to a low socioeconomic status [19,20]. One of the earliest studies sleep paralysis among African Americans found that environmental stressors and genetic could be associated with the frequency and severity of the disorder [21]. It seems that the higher prevalence of hypertension in the population can also be a factor in the different prevalence of sleep paralysis in African Americans. In their study, Ohayon et al. [6] reported an association between

Table 2
Prevalence and adjusted relative risks for prevalent sleep paralysis at W2.

Participants Characteristics	N	%	Prevalent Sleep Paralysis		
			Adjusted RR	95% CI for RR Lower-Upper	Sig.
Age Groups at W1					
18-24	625	16.5	1.10	0.82–1.46	0.536
25-34	1172	19.6	1.48	1.17–1.87	**0.001**
35-44	2170	17.1	1.15	0.94–1.41	0.162
45-54	2252	16.6	1.04	0.86–1.27	0.681
55-64	1836	13.2	1.03	0.84–1.26	0.767
≥ 65	2876	11.6	ref		
Race					
American Indian	77	9.1	0.88	0.31–2.54	0.814
Asian	557	20.2	1.42	0.94–2.18	0.097
Black	1137	23.2	2.13	1.67–2.71	**0.0001**
Other race alone	1071	19.6	1.46	1.16–1.85	**0.001**
2 or more races	339	24.6	1.93	1.50–2.47	**0.0001**
White alone	7750	13.4	ref		
Chronic Pain Status					
Never had pain	5077	11.1	ref		
Pain at W1 only	2684	14.0	1.13	0.96–1.33	0.130
Pain at W2 only	1655	21.0	1.61	1.33–1.93	**0.0001**
Pain at W1 & W2	1515	24.3	1.61	1.35–1.92	**0.0001**
Hypersomnolence					
No somnolence	6966	12.9	ref		
at W1 only	1475	19.1	1.46	1.23–1.74	**0.0001**
at W2 only	1406	16.4	1.01	0.84–1.23	0.887
at W1 & W2	1084	22.1	1.28	1.05–1.56	**0.016**
Insomnia disorder at W1					
Absent	9335	14.9			
Present	1596	16.2			n.s.
Major depressive disorder at W1					
Absent	10374	14.7	ref		
Present	557	19.8	1.69	1.28–2.24	**0.0001**
Cataplexy					
No cataplexy	10348	14.5	ref		
at W1 only	298	11.4	0.60	0.39–0.92	**0.019**
at W2 only	238	36.8	1.68	1.16–2.43	**0.007**
at W1 & W2	47	63.0	5.55	2.56–12.05	**0.0001**
Hypnagogic hallucinations					
None	9277	13.7	ref		
at W1 only	794	15.7	1.11	0.88–1.40	0.361
at W2 only	625	26.5	1.47	1.17–1.84	**0.001**
at W1 & W2	235	39.3	1.72	1.22–2.44	**0.002**
Hypnopompic hallucinations					
None	9210	13.4	ref		
at W1 only	970	17.2	1.36	1.11–1.68	**0.004**
at W2 only	523	29.6	2.09	1.65–2.65	**0.0001**
at W1 & W2	228	42.3	3.80	2.78–5.20	**0.0001**
Sleep duration at W2 vs W1					
About the same	3223	14.0	ref		
Shorter >60 min	1048	22.3	1.65	1.33–2.04	**0.000**
Shorter 16–60 min	2784	15.4	1.00	0.84–1.19	0.980
Longer 16–60 min	2831	15.7	1.20	1.02–1.42	**0.031**
Longer >60 min	1049	15.1	1.06	0.84–1.33	0.646
Posttraumatic stress disorder at W1					
Absent	10592	14.9	ref		
Present	339	17.7	1.54	1.02–2.34	**0.041**
Analgesic/antipyretic at W1					
Absent	10439	14.7	ref		
Present	492	21.7	1.73	1.33–2.25	**0.0001**
Body mass index			0.82	0.76–0.89	**0.0001**

W1: initial interview; W2: follow-up interview.
RR: Relative risk; CI: Confidence interval.
Non-significant variables adjusted model: Gender, Alcohol intake at W1, medical conditions at W1*, anxiety disorder (other than posttraumatic stress disorder) AT W1, fatigue at W1, antidepressant, anxiolytic or hypnotic medications at W1.
* Medical conditions included: Cerebrovascular diseases, diabetes, heart diseases, malignant neoplasm, hypertension, diseases of the central nervous system, disorders of the lipoprotein metabolism, diseases of the digestive system, diseases of the blood and blood-forming organs, disorders of the genital tract, lower respiratory tract diseases, upper respiratory tract diseases, disorders of the kidney and ureter, diseases of the musculoskeletal system and connective tissue, disorders of the thyroid gland, diseases of the urinary system.

Table 3
Incidence and adjusted relative risks for sleep paralysis at W2.

Participants Characteristics	N	%	Incident Sleep Paralysis		
			Adjusted	95% CI for RR	Sig.
			RR	Lower-Upper	
Age Groups at W1					
18-24	625	11.4	0.89	0.64–1.24	0.506
25-34	1172	15.0	1.60	1.26–2.03	**0.0001**
35-44	2170	12.6	1.25	1.02–1.53	**0.031**
45-54	2252	13.2	1.08	0.88–1.32	0.461
55-64	1836	11.3	1.02	0.83–1.26	0.859
≥ 65	2876	10.8	ref		
Race					
American Indian	77	9.1	1.22	0.42–3.51	0.711
Asian	557	8.6	0.75	0.42–1.35	0.342
Black	1137	16.1	1.66	1.27–2.19	**0.0001**
Other race alone	1071	15.6	1.44	1.12–1.86	**0.005**
2 or more races	339	18.8	1.98	1.52–2.58	**0.0001**
White alone	7750	11.1	ref		
Chronic Pain Status					
Never had pain	5077	9.7	ref		
Pain at W1 only	2684	9.6	0.99	0.83–1.19	0.936
Pain at W2 only	1655	17.3	1.61	1.32–1.96	**0.0001**
Pain at W1 & W2	1515	19.5	1.60	1.33–1.93	**0.0001**
Hypersomnolence					
No somnolence	6966	11.1	ref		
at W1 only	1475	14.9	1.44	1.19–1.74	**0.0001**
at W2 only	1406	12.7	0.99	0.821.21	0.952
at W1 & W2	1084	15.0	1.11	0.87–1.37	0.467
Insomnia disorder at W1					
Absent	9335	12.4	ref		
Present	1596	10.7	0.79	0.65–0.96	**0.018**
Major depressive disorder at W1					
Absent	10374	12.0	ref		
Present	557	12.6	1.63	1.18–2.57	**0.003**
Cataplexy					
No cataplexy	10348	12.0	ref		
at W1 only	298	6.7	0.53	0.31–0.90	**0.018**
at W2 only	238	25.5	0.90	0.55–1.46	0.658
at W1 & W2	47	19.1	4.70	1.80–12.21	**0.002**
Hypnagogic hallucinations					
None	9277	11.4	ref		
at W1 only	794	11.7	1.05	0.81–1.36	0.724
at W2 only	625	20.9	1.55	1.21–1.98	**0.001**
at W1 & W2	235	20.9	1.60	1.06–2.40	**0.026**
Hypnopompic hallucinations					
None	9210	11.6	ref		
at W1 only	970	9.9	1.12	0.87–1.44	0.373
at W2 only	523	19.3	1.70	1.29–2.22	**0.0001**
at W1 & W2	228	31.3	3.50	2.50–4.96	**0.0001**
Sleep duration at W2 vs W1					
About the same	3223	12.0	ref		
Shorter >60 min	1048	19.2	1.69	1.36–2.10	**0.0001**
Shorter 16–60 min	2784	11.4	0.88	0.73–1.05	0.158
Longer 16–60 min	2831	12.5	1.17	0.98–1.39	0.082
Longer >60 min	1049	12.1	1.02	0.80–1.31	0.856
Posttraumatic stress disorder at W1					
Absent	10592	12.1			
Present	339	9.0			n.s.
Analgesic/antipyretic at W1					
Absent	10439	11.5	ref		
Present	492	20.6	2.02	1.54–2.64	**0.0001**
Body mass index			0.80	0.73–0.88	**0.0001**

W1: initial interview; W2: follow-up interview.
RR: Relative risk; CI: Confidence interval.
Non-significant variables adjusted model: Gender, Alcohol intake at W1, medical conditions at W1*, anxiety disorder (other than posttraumatic stress disorder) AT W1, fatigue at W1, antidepressant, anxiolytic or hypnotic medications at W1.
* Medical conditions included: Cerebrovascular diseases, diabetes, heart diseases, malignant neoplasm, hypertension, diseases of the central nervous system, disorders of the lipoprotein metabolism, diseases of the digestive system, diseases of the blood and blood-forming organs, disorders of the genital tract, lower respiratory tract diseases, upper respiratory tract diseases, disorders of the kidney and ureter, diseases of the musculoskeletal system and connective tissue, disorders of the thyroid gland, diseases of the urinary system.

hypertension and sleep paralysis. However, the results of the studies are contradictory, and some studies have not mentioned ethnicity as an associated factor. The present study does not consider the role of cultural factors and more studies are needed to consider cultural differences [20,22].

Although most studies did not show significant differences for age and sex, in the present study, younger individuals reported a higher prevalence and incidence of sleep paralysis [4–6,8,23–25]. Not surprisingly, sleep paralysis may begin in adolescence and become less severe in middle age or disappear [26,27]. The reasons for this discrepancy may not be clear, but they can be attributed in part to the regular experience of sleep disorders. The under-40 age group is often an active population that may have an unhealthy lifestyle (including sleep disorders) that can itself be considered a risk factor for the severity and frequency of sleep paralysis [28]. The explanation could also be simply biological: as individuals are getting older, le CNS matures and parasomnias like sleep paralysis, sleep walking, hypnagogic and hypnopompic hallucinations are becoming less frequent and eventually stop. However, it should not be forgotten that younger people are more exposed to environmental stressors, which is another factor for the higher prevalence of sleep paralysis.

The present study showed that the symptoms of narcolepsy syndrome (Cataplexy, Hypersomnolence, Hypnagogic and Hypnopompic hallucinations) are strongly associated with the prevalence and incidence of sleep paralysis [22,29–32]. Narcolepsy is a sleep disorder characterized by REM sleep anomalies. Consequently, it is not surprising to observe a cluster of narcolepsy symptoms associated with sleep paralysis.

The results of our study show that the prevalence of sleep paralysis can be associated with PTSD in addition to narcolepsy. In this study, individuals with PTSD were 1.5 times more likely of reporting sleep paralysis. Sleep paralysis has a prevalence of 27.8–67% in people with PTSD [24,33]. People with PTSD often suffer from insomnia, nightmares, or sudden awakenings during the night [34]. Therefore, PTSD can be associated with the severity and frequency of sleep paralysis [24]. Similarly, intensity of the experienced stress is associated with a higher probability of experiencing sleep paralysis [35]. It has also been suggested that sleep paralysis could constitute a mode of reexperiencing the trauma associated with PTSD [36].

One of the interesting results of the present study was the relationship between chronic pain and the use of antipyretic and analgesic drugs with a higher prevalence and incidence of sleep paralysis. It has been shown that the use of antipyretic medications can have a negative effect on a person's sleep pattern [37]. For example, the use of NSAIDs can prolong the stage of wakefulness in people [37]. The relationship between pain and sleep problems is reciprocal, and even sleep disorders can lead to more severe or worsening pain in patients with chronic pain [38]. On the other hand, the use of analgesics can lead to delirium or sleep disorders. Therefore, the regular use of these medications may be associated with sleep paralysis [39].

This study has two important strengths: first, it was conducted for the first time on the general American population with a considerable sample size of 10,931 people randomly selected from eight states. Using the sampling class to distribute demographic characteristics, a representative sample of the US general population was collected. Using a longitudinal study design that did not exist in previous studies, and the results of this study for the first time examine the incidence of sleep paralysis in the general population.

Despite its strengths, this study has limitations, the most important being the use of self-report inherent to general population surveys.

5. Conclusion

This study reports the prevalence, incidence and evolution of sleep paralysis in the US general adult population. According to the results of the present study, sleep paralysis not only did not decrease but also increased in follow-up measurement. Several clinical and demographic factors were also associated with prevalence and incidence of sleep paralysis. Sleep paralysis remains strongly associated with the narcolepsy tetrad symptoms described by Gelineau in 1880 [40] and deserve more attention in the field of Hypersomnia disorders. Future studies should examine the role of these factors in the long term.

Data access

The principal author has full access of the data used in the analyses for this manuscript.

CRediT authorship contribution statement

M.M. Ohayon: contributed to conception, Conceptualization, design, and planning of the study, analysis of the data, Formal analysis, Data curation, interpretation of the results, and drafting of the manuscript, Writing – original draft. **A.H. Pakpour:** contributed to the interpretation of the results, and drafting of the manuscript, Writing – original draft.

Declaration of competing interest

Dr. Ohayon have consulting agreements with Novartis and Takeda non-related to this manuscript. Dr. Pakpour has nothing to disclose.

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