Sleep Medicine Reviews xxx (2017) 1–17

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

CLINICAL REVIEW

A systematic review of variables associated with sleep paralysis

Dan Denis [a, b, c, *], Christopher C. French [d], Alice M. Gregory [d]

[a] Center for Sleep and Cognition, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA
[b] King's College London, MRC Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology, and Neuroscience, London, UK
[c] Department of Psychology, University of Sheffield, UK
[d] Department of Psychology, Goldsmiths, University of London, UK

ARTICLE INFO

Article history:
Received 2 August 2016
Received in revised form
29 May 2017
Accepted 30 May 2017
Available online xxx

Keywords:
Sleep paralysis
Isolated sleep paralysis
Parasomnia
REM sleep
Sleep disruption
Intruder
Incubus
Felt presence

SUMMARY

Sleep paralysis is a relatively common but under-researched phenomenon. While the causes are unknown, a number of studies have investigated potential risk factors. In this article, we conducted a systematic review on the available literature regarding variables associated with both the frequency and intensity of sleep paralysis episodes. A total of 42 studies met the inclusion criteria. For each study, sample size, study site, sex and age of participants, sleep paralysis measure, and results of analyses looking at the relationship(s) between sleep paralysis and associated variable(s) were extracted. A large number of variables were associated with sleep paralysis and a number of themes emerged. These were: substance use, stress and trauma, genetic influences, physical illness, personality, intelligence, anomalous beliefs, sleep problems and disorders (both in terms of subjective sleep quality and objective sleep disruption), symptoms of psychiatric illness in non-clinical samples (particularly anxiety symptoms), and psychiatric disorders. Sleep paralysis appears to be particularly prevalent in post-traumatic stress disorder, and to a less degree, panic disorder. Limitations of the current literature, directions for future research, and implications for clinical practice are discussed.

© 2017 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Introduction

Sleep paralysis involves a period of time at either sleep onset or upon awakening from sleep during which voluntary muscle movements are inhibited. Ocular and respiratory movements remain unaltered and perception of the immediate environment is clear [1]. These episodes are frequently associated with a variety of hallucinations, such as a sense of an evil presence (known as intruder hallucinations), pressure felt on the chest (incubus hallucinations), and illusory feelings of movement (vestibular-motor (V-M) hallucinations) [2]. Sleep paralysis is a global phenomenon, with terms for sleep paralysis existing in over 100 cultures [3]. In many places, sleep paralysis experiences are interwoven with a culture's folklore [4,5]. Episodes of sleep paralysis have been suggested as an explanation for supposed paranormal phenomena such as witchcraft [6], demonic assault [7], and space alien abduction [8,9]. Fear and distress are typically associated with episodes [2], though feelings of bliss are sometimes reported [10].

A review of lifetime prevalence rates of sleep paralysis in the general population estimated prevalence to be approximately 8%, though individual study estimates greatly vary from 2 to 60% [11]. The lack of a 'gold standard' measure of sleep paralysis is likely part of the reason for this [3] and the problem is amplified by the fact that the precise phrasing used to ask about sleep paralysis has been shown to affect the reported prevalence rate [12]. Averaging over multiple studies, no effects of age have been found and sex differences show mixed results [11]. Finally, there are slightly higher lifetime prevalence rates of sleep paralysis in non-Caucasian compared to Caucasian groups [11].

In addition to limitations with the measures used to assess sleep paralysis, another outstanding problem is the lack of consistency with terminology. Sleep paralysis is a common symptom of narcolepsy, a neurological disorder. Narcolepsy is characterised by excessive daytime sleepiness, cataplexy (sudden, brief, bilateral losses of muscle tone in response to strong emotions such as laughter or anger [13]) and disturbed nocturnal sleeping patterns [14]. Therefore, the term *isolated sleep paralysis* is preferred when sleep paralysis is present in the absence of a diagnosis of narcolepsy [3]. Some authors also use the term *fearful isolated sleep paralysis* to indicate cases where episodes are causing clinically significant fear

* Corresponding author. Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Avenue, Boston, MA, 02215, USA.
E-mail address: ddenis@bidmc.harvard.edu (D. Denis).

http://dx.doi.org/10.1016/j.smrv.2017.05.005
1087-0792/© 2017 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

and/or distress [3]. Finally, when episodes occur repeatedly, the term *recurrent (fearful) isolated sleep paralysis* can be used, though there is no agreement on exactly how often episodes need to occur in order to be considered recurrent [15,16].

The causes of sleep paralysis are likely to be multifactorial [17]. It is therefore important for clinicians and researchers to understand the factors that may influence the frequency and intensity of episodes. Here, we systematically review the available literature regarding variables associated with sleep paralysis. It is hoped that this review will provide a resource to both clinicians, who shall have a better understanding of this common experience, and also researchers interested in formulating new research questions.

Methods

A literature search was performed using OvidSP on the following databases: *Ovid MEDLINE* (1946 – September 2015); *PsycINFO* (1806 – September 2015); *Journals@Ovid Full Text* (– September 2015); and *PsycARTICLES Full Text* (– September 2015). The following search terms were used: "sleep paralysis", "isolated sleep paralysis", "parasomnia not otherwise specified", "hypnagogic", "hypnopompic", "parasomnia", "sensed presence", and "incubus".

There was no restriction made on the age of articles included in this review. For all identified articles published since 2000, reference lists were also scanned to see whether we had missed any articles suitable for inclusion in this review. Furthermore, manual searches were conducted of all journals containing more than five of the studies identified via the database searches. Where this happened, only articles published since 2000 were scanned for reasons of feasibility. These steps allowed us to reduce the possibility of non-indexed studies being missed.

For inclusion in this systematic review, studies were required to meet the following criteria:

1) Results are presented as a full original research paper published in a peer-reviewed journal.
2) Sleep paralysis is clearly defined, and it is clear that sleep paralysis has been identified in the sample, and is not another phenomenon (e.g., night terrors, hypnagogic/hypnopompic hallucinations with no paralysis, nightmares).
3) The presence of sleep paralysis in the sample was measured either via a continuous measure of frequency or a binary sleep paralysis present/absent measure.
4) An association has been explored between sleep paralysis and other variable(s). Either a comparison between groups (e.g., sleep paralysis vs no sleep paralysis) or an association between sleep paralysis frequency/intensity and the variable(s) under study.
5) Single case studies were excluded.
6) Review, commentary, or opinion articles not including any original data were excluded (but reference lists from such papers were checked, in line with the search strategy).
7) Only English language articles were included.

In total, 35 articles were identified that matched all the inclusion criteria. One article was found by searching article reference lists, and one was obtained through personal correspondence. Furthermore, an additional five articles that also matched all the inclusion criteria were recommended by a reviewer. Therefore, a total of 42 articles were included in this report. The process for selecting studies is displayed in the Supplementary materials. From the included studies, the following data were extracted: Sample size and study site, sex and age of participants, sleep paralysis measure, and results of analyses looking at the relationship(s) between sleep paralysis and associated variable(s).

Results

The articles included in the review can be found in Table 1. The studies identified come from a wide range of different research groups, providing a highly international and cross-cultural sample.

Studies looking at associations between sleep paralysis and other variables are found in Table 2. Associations between sleep paralysis and variables specifically related to sleep-related factors are displayed in Table 3, with associations with other sleep disorders shown in Table 4. Table 5 shows associations between sleep paralysis and symptoms of psychiatric illness. Finally, Table 6 shows associations with psychiatric disorders and medication. Overall, the majority of studies looked at sleep paralysis frequency alone, with fewer studies examining variables associated with the intensity and/or vividness of sleep paralysis and associated hallucinations.

Demographics

Relationships between demographic variables and sleep paralysis are shown in Table 2. Generally, studies that have investigated age differences in sleep paralysis prevalence have found no significant effect of age [17–21]. Similarly for sex differences the majority of studies found no significant effects [17–19,22–27]. Two large-scale surveys of Asian adolescents did find significant sex differences, with higher prevalence in females though the reported differences were very small. Munezawa and colleagues found an 8.2% prevalence for males and 8.4% for females. Ma et al. found a prevalence of 6.1% for males and 7.4% for females [24,28]. In a study of Hmong immigrants, higher odds of experiencing sleep paralysis were found in male participants (odds ratio (OR) = 1.61), though the exact prevalence rates for both sexes were not reported [21]. Finally, a study in an American sample found that males were more likely to have experienced lifetime isolated sleep paralysis than females, but there were no differences in lifetime fearful or recurrent fearful isolated sleep paralysis episodes [29]. With regards to ethnicity, evidence is also mixed. One study found African American individuals experienced a higher incidence of sleep paralysis compared with Caucasians [30]. A second study found that non-Caucasians had a higher prevalence of fearful and recurrent fearful isolated sleep paralysis compared to Caucasians [29]. Two other studies however found no significant effect of ethnicity [23,31].

Presence of sleep paralysis was shown to be higher in rural compared to urban areas in a survey of Chinese adolescents [28]. Having a higher amount of available money had a small but significant association with the presence of sleep paralysis (10.9% of participants with ¥5000 or more available to them experienced sleep paralysis compared with 7.3% who had less than ¥5000 available) in one Japanese sample, as did regularly eating breakfast, with those who ate breakfast everyday reporting a lower incidence of sleep paralysis (7.5%) compared to those who ate breakfast occasionally (10.8%) [24], though findings are inconsistent [21,32]. It is unclear why these variables should be associated with sleep paralysis. Some research suggests that food timing may be important in the synchronisation of internal circadian clocks [33]. It is possible that by eating breakfast only occasionally, circadian clocks are disrupted leading to an increased incidence of sleep paralysis.

Drinking alcohol, smoking, and substance use

Relationships between substance-use variables and sleep paralysis are shown in Table 2. It is unclear whether sleep paralysis is associated with general substance use. In two large nationwide samples in China (N = 11,754) and Japan (N = 90,081) it was found that those who reported drinking at least one alcoholic drink per day over the last month were significantly more likely (9–12%) than

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 1
Summary of articles included in the review.

Study	N (% female)	Country	Mean age (y), SD (age range)	Measure of sleep paralysis	Associated variables
Abrams et al. (2008) [35]	263 (72)	USA	22, 6 (18–52)	WUSEQ	CSA, PCL-C, CES-D, DES-II, TAS
Andlauer et al. (2012) [58]	171 (43)	International	31, 3 (nr)	Questionnaire (self-made)	Narcolepsy, CSF hypocretin-1 concentration
Bassetti & Aldrich (1997) [60]	109 (60)	USA	35, nr (nr)	Single item	Idiopathic hypersomnia, mono-symptomatic narcolepsy, narcolepsy with cataplexy
Bell et al. (1984) [38]	108 (52)	USA	31, nr (nr)	Interview (Bell)	HRSRS, SSADS, MCMI
Bell et al. (1986) [40]	25 (76)	USA	25, nr (nr)	Interview (Bell)	Family history, life stress, panic attacks
Cheyne (2002) [53]	5799 (65)	International	27, 10 (nr)	WUSEQ	Sleep position, sleep timing
Dahlitz & Parkes (1993) [41]	64 (44)	Not reported	nr	Single item	Cataplexy symptoms, ESS, family history, HLA gene expression
Denis et al. (2015) [17]	862 (66)	UK	25, 2 (22–32)	Single item	PSQI, anxiety symptoms, MFQ, threatening life events, substance use, genetics
Denis & Poerio (2016) [39]	1928 (53)	International	34, 14 (18–82)	WUSEQ	Lucid dreaming, SCI, DDFS, PCDD, DES-II, MAAS, PSI-Q, MFQ, STAI, PSS, GCBS, PBS
Dodet et al (2015) [46]	159 (40)	France	37, 14 (nr)	Single item	Narcolepsy, with and without cataplexy
Fukuda et al. (1998) [54]	235 (53)	Canada and Japan	20, nr (nr)	Questionnaire (self-made)	Sleeping position
Girard & Cheyne (2006) [55]	348 (71)	International	31.3, 10 (16–69)	WUSEQ	Sleep timing
Hinton et al. (2005a) [65]	100 (55)	Cambodian refugees	55, 9 (nr)	Interview (Hinton)	Panic attack, PTSD
Hinton et al. (2005b) [66]	100 (68)	Cambodian refugees	49, 5 (nr)	SPQ	Panic attack, PTSD
Hsieh et al. (2010) [18]	107 (19)	Taiwan	52, 14 (15–88)	Questionnaire (self-made)	ESS, PSQI, P-SF36, M-SF36
Kotorii et al. (2001) [51]	8162 (nr)	Japan	nr	Questionnaire (self-made)	Employment type, sleep schedule
Ma et al. (2014) [28]	11,764 (49)	China	nr	Single item	Substance use, subjective sleep quality, sleep timing, GHQ
McNally & Clancy (2005) [36]	84 (76)	USA	43, 13 (nr)	SEQ	TAS, DES, BDI
Mellman et al. (2008) [37]	441 (68)	USA	40, 13 (nr)	Questionnaire (self-made)	Traumatic life events, depressed mood, panic attacks, SCID-IV/CAPS
Mume & Ikem (2009) [43]	91 (36)	Nigeria	37, 12 (16–72)	Questionnaire (self-made)	Musculoskeletal illness, multiple somatic complaints, HARS
Munezawa et al. (2009) [32]	916 (38)	Japan	nr	Single item	PSQI, ESS, lifestyle, physical health, GHQ
Munezawa et al. (2011) [24]	90,081 (50)	Japan	nr (6–15)	Single item	Subjective sleep quality, ESS, GHQ, nightmares, substance use, lifestyle
Ohaeri et al. (1989) [26]	164 (27)	Nigeria	24, 2 (20–37)	Questionnaire (Bell)	SRQ, EPQ, major life events
Ohaeri et al. (1992) [27]	95 (39)	Nigeria	nr 17–32	Questionnaire (Bell)	SRQ, EPQ, major life events
Ohayon et al. (1999) [20]	8085 (nr)	Germany and Italy	nr	Interview (SLEEP-EVAL)	HH, EDS, subjective sleep quality, sleep disorder, physical disorder, psychiatric disorder, medications, substance use, BMI
Otto et al. (2006) [23]	61 (44)	USA	43, 13 (nr)	SEQ	PD, social anxiety disorder, GAD
Paradis et al. (1997) [30]	129 (68)	USA	nr	Questionnaire (Bell) adapted	ADIS-R
Paradis et al. (2009) [31]	208 (83)	USA	22, nr 18, 59	USEQ	Anxiety disorders
Ramswah et al. (2008) [19]	72 (73)	USA	26, 9 (nr)	ISPQ	ADIS-IV-L, ASI, LES, PBS
Sharpless (2015) [47]	211 (71)	USA	20, 2 (nr)	FISPI	Exploding head syndrome
Sharpless et al. (2010) [29]	133 (67)	USA	39, 13 (nr)	FISPI	ADIS-IV, ASI, brief bodily sensations, BMI
Simard & Nielsen (2005) [63]	45 (62)	Canada	22, 2	ISPQ	LSAS, BDI, FSS-II, SDDQ
Solomonova et al. (2008) [64]	193 (67)	Canada	32, 13 (18–87)	WUSEQ – adapted	LSAS, OEQ7, psychiatric diagnosis, nightmare distress
Spanos et al. (1995) [22]	1798 (54)	Canada	nr	Questionnaire (self-made)	Psychopathology, imaginativeness, headache/ migraine, sleep problems
Szklo-Coxe et al. (2007) [56]	866 (47)	USA	nr	Single item	Depressed mood
Takeuchi et al. (1992) [50]	16 (50)	Japan	Nr (18–21)	Experimental induction	Sleep interruption
Takeuchi et al. (2002) [42]	13 (33)	Japan	21, 2 (nr)	Experimental induction	Multi-phase sleep/wake schedule, KSS, SFQ, MACL
Vernet et al (2009) [61]	75 (64)	France	34, 13 (nr)	Single item	Idiopathic hypersomnia with either long or short sleep time
Vernet et al (2011) [57]	60 (27)	France	60, 10 (nr)	Single item	Obstructive sleep apnoea

(*continued on next page*)

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 1 (*continued*)

Study	N (% female)	Country	Mean age (y), SD (age range)	Measure of sleep paralysis	Associated variables
Wing et al. (1994) [25]	603 (42)	China	21, 1 (17–32)	Questionnaire (self-made)	Family history, cataplexy symptoms, EDS
Yeung et al. (2005) [67]	194 (nr)	China and USA	46, 14 (nr)	SPQ	Psychiatric disorder
Young et al. (2013) [21]	747 (38)	Hmong immigrants	40, 13 (18–86)	Questionnaire (self-made)	Sleep problems, socio-demographics, acculturation, traditional beliefs, general health

Note: N = number of participants, SD = standard deviation, nr = not reported.
Measures of sleep paralysis: FISPI = fearful isolated sleep paralysis inventory, ISPQ = isolated sleep paralysis questionnaire, SEQ = sleep experiences questionnaire, SPQ = sleep paralysis questionnaire, USEQ = Unusual sleep experiences questionnaire, WUSEQ = Waterloo unusual sleep experiences questionnaire.
Associated variables: ADIS-IV = anxiety disorders interview schedule IV, ADIS-IV-L = anxiety disorders interview schedule IV – lifetime, ADIS-R = anxiety disorders interview schedule – revised, ASI = anxiety sensitivity index, BDI = Beck depression inventory, BMI = body mass index, CAPS = clinician administered PTSD scale, CES-D = center for epidemiologic studies depression scale, CSA = childhood sexual abuse, CSF = cerebral spinal fluid, DDFS = daydreaming frequency scale, DES = dissociative experiences scale, DES-II = dissociative experiences scale – revised, EDS = excessive daytime sleepiness, EPQ = Eysenck personality questionnaire, ESS = Epworth sleepiness scale, FSS-II = fear survey schedule II, GAD = generalized anxiety disorder, GCBS = generic conspiracist beliefs scale, GHQ = general health questionnaire, HARS = Hamilton anxiety rating scale, HH = hypnogogic/hypnopompic hallucinations, HRSRS = Holmes-Rahe social readjustment scale, KSS = Kuansei-gakuin sleepiness scale, LES = life experiences survey, LSAS = Liebowitz social anxiety scale, MAAS = mindful attention awareness scale, MACL = mood adjective checklists, MCMI = million clinical multiaxial inventory, MFQ = mood and feelings questionnaire, M-SF36 = mental health short form 36, OEQ7 = other experiences questionnaire 7-item social imagery subscale, PBS = paranormal beliefs scale, PCDD = positive constructive daydreaming scale, PCL-C = PTSD checklist, civilian version, PD = panic disorder, P-SF36 = physical health short form 36, PSI-Q = Plymouth sensory imagery questionnaire, PSQI = Pittsburgh sleep quality index, PSS = perceived stress scale, PTSD = post-traumatic stress disorder, SCI = sleep condition indicator, SCID-IV = structured clinical interview for DSM-IV, SDDQ = sleep and dreaming disorders questionnaire, SFQ = subjective fatigue scale, SRQ = self-reporting questionnaire, SSADS = Saafir stress-anxiety diagnostic scale, STAI = state-trait anxiety inventory, TAS = Tellegen absorption scale.

others (6–7%) to report experiencing sleep paralysis [24,28]. However, in a large (N = 862) UK sample where self-reported weekly alcohol consumption in terms of units of alcohol was recorded, alcohol intake over the past week did not predict sleep paralysis independently of anxiety symptoms, depressed mood, threatening events, and sleep quality [17].

Evidence for the association between smoking behaviour and sleep paralysis is also mixed – with increased odds of experiencing sleep paralysis in those who smoked (defined as at least one cigarette a day) (15%) compared with those who did not (8%) [24]. This relationship was not found in two other studies [17,28]. Of note, none of the studies on this topic have provided a quantitative estimate of amount of nicotine consumed. Given the negative impact caffeine can have on sleep [34], it was surprising that there was no evidence of a significant association between caffeine intake and sleep paralysis [17,28].

Stress and trauma

Relationships between stress and trauma, and sleep paralysis are shown in Table 2. A confirmed or unconfirmed history of childhood sexual abuse (CSA) was found to be significantly related to frequency of sleep paralysis episodes [35]. This study defined a confirmed case of CSA as either having a person other than the victim and/or abuser confirm the abuse, or that other people confronted the abuser, or that the abuser was charged in connection with the abuse. An unconfirmed history of CSA was defined as having vivid recollections of abuse in the absence of anyone being aware of it/no one being confronted or charged as the abuser. The frequency and intensity of intruder and incubus hallucinations were significantly greater in both CSA groups as compared to those who did not report sexual abuse [35]. No differences in terms of V-M hallucinations were found between groups. Another study found the reported prevalence of sleep paralysis did not differ significantly between groups of participants who reported remembering their CSA (47%) with those who believed they had experienced CSA but possessed no autobiographical memories of it (44%). Sleep paralysis prevalence was significantly higher in both CSA groups compared to a control group who reported not having experienced CSA (13%) [36].

Other experiences of threatening/traumatic events also appear to be related to sleep paralysis. In a sample of Hmong immigrants living in the USA, stressful experiences during the Vietnam war (e.g., "I was exposed to chemical warfare", "I lost family, close relatives or friends") were related to increased odds of experiencing sleep paralysis [21]. General experiences with potentially traumatic events (such as assault, death of a loved one, disasters, etc.) were found to be related to sleep paralysis in terms of the occurrence of a traumatic event [37]. Also a link was found between increasing numbers of traumatic events experienced and sleep paralysis [17,37]. Relatedly, self-report levels of life-stress showed similar associations with sleep paralysis [19,38,39]. In studies of Nigerian doctors and nurses, the authors did not find a relationship between sleep paralysis and threatening events [26,27]. This may be due to a smaller sample size or could be because of the specific demographic targeted.

Hereditary factors

Relationships between hereditary factors and sleep paralysis are shown in Table 2. A familial association has been reported for sleep paralysis [25,40,41]. One study by us used a twin modelling approach to disentangle genetic from environmental effects. There appeared to be moderate genetic influences (estimated at 53%) on variation in presence of sleep paralysis [17]. A number of specific genes involved in circadian cycles were also examined and specific polymorphisms of the PER2 gene were associated with sleep paralysis although the association was no longer significant when adjusting for multiple testing [17]. Another study employing a very small sample (N = 44) found no association between sleep paralysis and narcolepsy genes HLA DR2 or HLA DQ1 [41].

Physical health

Relationships between physical health and sleep paralysis are shown in Table 2. General physical health problems appear to be associated with sleep paralysis when using general physical health quality of life scales [20,42,43], though this was not found in one study [18]. Little research however has linked any specific physical health problems with sleep paralysis. For example, body mass index (BMI) was not found to be related to whether someone reported having experienced sleep paralysis during their lifetime [32] or to vary with frequency of episodes [20]. However, in another study, higher BMI was related to both lifetime and recurrent episodes of fearful isolated sleep paralysis but not related to lifetime non-fearful isolated sleep paralysis [29]. In an experimental study,

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 2
Associations between sleep paralysis episodes and other variables.

Associated variable	Association	No association
Section 1 – Frequency/occurrence of sleep paralysis episodes		
1. Demographics		
Age		[18] – Age of those with SP vs those without SP: 49.9 y vs 52.7 y ns [19] – Age of those with SP vs those without SP: 24.9 y vs 27.6 y ns [17,20,21] – Ns in a multiple predictor model
Sex	[21] – Increased odds of SP in males. OR = 1.61 (males) [28] – Females with SP vs males with SP: 7.4% vs 6.1%, OR (CI) = 1.24 (1.07–1.45) [24] – Females with SP vs males with SP: 8.4% vs 8.2% [29] – Males more likely to experience SP than females, but not fearful or recurrent fearful SP	[17] – Ns in a multiple predictor model [18,22] – No association [19] – No sex differences in those with SP vs those without SP [23] – Prevalence of SP in females (29.6%) vs prevalence of SP in males (11.8%) ns [32] – Females with SP vs males with SP: 8.0% vs 6.7% ns [25] – No significant difference between females (39.7%) and males (35%) [26] – No significant difference between females (30.6%) and males (19.5%) [27] – No significant difference between females (40.5%) and males (46.5%)
Ethnicity	[29] – SP more common in non-Caucasian individuals, $r = 0.21$. [30] – SP more common in African Americans with panic disorder (59.6% vs 7.5%), other anxiety disorders (11.1% vs 0%), and controls (23% vs 6%) compared to Caucasians	[23] – Inclusion or exclusion of non-Caucasian groups did not lead to differences in SP prevalence [31] – African American with SP vs non-African Americans with SP, 31% vs 24% ns
Community	[28] – Prevalence of SP in those from rural areas vs those from urban areas, 7.3% vs 6.2%, OR (CI) = 1.17 (1.01–1.37)	
Lifestyle/SES	[24] – Prevalence of SP in those with ¥5000 or greater vs prevalence of SP in those with less than ¥5000, 10.9% vs 7.3%, OR (CI) = 1.08 (1.01–1.15) [24] – Prevalence of those who eat breakfast only occasionally vs those who eat breakfast everyday, 7.5% vs 10.8%, OR (CI) = 1.06 (0.97–1.15)	[21] – Years living in the USA and tradition vs Western diet both ns predictors of SP
2. Smoking, drinking alcohol and substance use		
Alcohol intake	[24] – Prevalence of SP in those who drink 1 + drink per day over the past month) vs those who do not drink, 12.2% vs 7.1%, OR (CI) = 1.23 (1.16–1.32) [28] – Prevalence of SP in those who drink 1 + drink per day over the past month) vs those who do not drink, 9.0% vs 6.3%, OR (CI) = 1.84 (1.23–2.74)	[17] – Ns in a multiple predictor model. OR (CI) = 1.12 (0.95–1.32)
Smoking behaviour	[24] – Prevalence of SP in those who smoke 1 + cigarette per day over the past month) vs those who do not smoke, 15.3% vs 7.8%, OR (CI) = 1.16 (1.02–1.32)	[17] – No association with SP. OR (CI) = 1.15 (0.99–1.33) [28] – Prevalence of SP in those who smoke 1 + cigarette per day over the past month) vs those who do not smoke, 6.8% vs 6.7%, OR (CI) = 1.05 (0.78–1.42). Ns
Caffeine intake		[17] – No association with SP. OR (CI) = 1.08 (0.93–1.25) [32] – Prevalence of SP in those who drink tea daily vs those who occasionally/never drink tea, 7.1% vs 8.0%.ns. Prevalence of SP in those who drink coffee daily vs those who drink coffee occasionally/never, 7.0% vs 9.3% ns.
3. Stress and trauma		
Threatening/traumatic life events	[17] – Significant predictor of SP in a multiple predictor model, OR (CI) = 1.29 (1.08–1.54) [21] – Significant predictor of sleep-onset SP in a multiple predictor model, OR = 1.69. Also a significant predictor of during sleep SP in a multiple predictor model, OR = 1.80 [37] – Prevalence of experienced traumas in those with SP vs those without SP, 79% vs 57%. Number of specific traumas higher in those with SP vs those without SP, M (SD) = 3.0 (2.6) vs 1.4 (1.8)	[26] – Negative life events score in those with SP vs those without SP, M (SD) = 5.3 (1.9) vs 4.9 (1.7) ns [27] – Negative life events score in those with SP vs those without SP, M (SD) = 4.79 (1.92) vs 4.87 (1.56) ns
Childhood sexual abuse	[36] – Prevalence of SP in those who have experienced CSA vs those who have not, 44.6% vs 13%	[22] – No association with SP
Life stress	[19] – Higher life stress scores in those with SP vs those without SP, M (SD) = 25.0 (13.4) vs 18.9 (10.0) [38] – In outpatient subjects, significant correlation with SP $r = 0.40$ [39] – Significant predictor of SP in a multiple predictor model	
4. Hereditary factors		
Family history	[41] – Family history of SP reported in 13 out of 22 participants [40] – Out of 64 family members, 33 reported SP [25] – Family history of SP reported in 20% of participants	
Genetic influences	[17] – Moderate genetic influences on SP (53%)	

(*continued on next page*)

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 2 (*continued*)

Associated variable	Association	No association
PER2 gene polymorphism	[17] – PER2 SNP rs2304672 associated with SP in additive (OR (SE) = 1.88 (0.45)) and dominant (OR (SE) = 1.84 (0.45)) models	
HLA gene polymorphism		[41] – No association
5. Physical health-related variables		
General physical health	[20] – Significant predictor of SP in an multiple predictor model, OR (CI) = 1.55 (1.06–2.25) [42] – Higher physical complaint scores in those with SP vs those without SP, M (SD) = 4.68 (2.07) vs 3.02 (1.21) [43] – SP prevalence in patients with orthopaedic complaints (44%), SP prevalence rates in patients with multiple somatic complaints (56%). Control group 28%	[18] – No association in a multiple predictor model
Body mass index	[29] – Significantly correlated with lifetime (r = 0.21) and recurrent (r = 0.23) fearful SP	[20] – No association [32] – No difference in SP prevalence in individuals with a BMI < 20 (7.3%) vs BMI => 20 (7.1%)
Blood pressure	[42] – Higher diastolic blood pressure in those with SP vs those without SP, M (SD) = 71.88 (5.23) vs 66.28 (7.68). Lower systolic blood pressure in those with SP vs those without SP, M (SD) = 104.14 (9.61) vs 112.53 (11.45)	
Chronic pain	[21] – Significant predictor of SP in multiple predictor model, predicting SP episodes when going to sleep (OR = 1.66), SP episodes occurring during sleep (OR = 1.80), and SP episodes occurring during wake (OR = 1.72)	
6. Personality, intelligence, and anomalous beliefs		
Personality		[26] – Mean score on personality scales in those with SP vs those without SP. M (SD) score on extraversion/introversion scale, 12.77 (4.4) vs 13.5 (3.3), psychoticism scale, 4.5 (2.2) vs 4.1 (2.3), and neuroticism scale, 8.4 (2.2) vs 7.6 (4.1). All ns [27] – Mean score on personality scales in those with SP vs those without SP. M (SD) score on extraversion/introversion scale, 13.41 (3.64) vs 13.51 (3.05), psychoticism scale, 4.79 (2.65) vs 4.12 (2.31), and neuroticism scale, 8.67 (4.43) vs 7.64 (3.79). All ns [22] – Mean score on personality scales in those with SP vs those without SP. M (SD) score on extraversion scale, 14.61 (4.37) vs 14.60 (4.61), and psychoticism scale, 4.27 (2.82) vs 4.24 (2.97). Both ns [38] – No association
Dissociative experiences	[36] – Higher mean scores in those with SP vs those without SP, M (SD) = 23.3 (17.0) vs 14.1 (11.9) [39] – Significant predictor of SP in a multiple predictor model	
Absorption		[36] – Mean score in those with SP vs those without SP. M (SD) = 19.8 (7.50) vs 17.0 (7.60), ns
Imaginativeness	[22] – Higher imaginativeness factor score in those with SP vs those without SP, M (SD) = 1.99 (6.05) vs −1.97 (5.47)	
Mindfulness		[39] – Ns in a multiple predictor model
Sensory imagery		[39] – No association with SP (r = 0.05)
Daydreaming frequency		[39] – Ns in a multiple predictor model
Daydreaming style		[39] – Ns in a multiple predictor model
Hypnotizability	[22] – Mean scores in those with SP vs those without SP. M (SD) score on objective hypnotizability, 3.15 (1.94) vs 2.65 (2.06), and subjective hypnotizability 8.36 (4.39) vs 6.56 (4.54)	
IQ	[29] – SP (r −0.22), fearful SP (r −0.29), recurrent fearful SP (r −0.28) associated with lower IQ	
Paranormal beliefs	[19] – Mean spiritual beliefs score in those with SP vs those without SP. M (SD) = 11.8 (3.9) vs 10.0 (3.7)	[19] – Mean score in those with SP vs those without SP. M (SD) = 73.8 (15.6) vs 69.5 (16.6) ns [22] – Paranormal beliefs factor score in those with SP vs those without SP, M (SD) = 0.08 (0.26) vs 0.04 (0.18) ns [39] – Ns in a multiple predictor model
Conspiracist beliefs		[39] – No association with SP (r = 0.04)
Section 2 – Intensity of sleep paralysis episodes/associated hallucinations		
1. Stress and trauma		
Life stress	[22] – Associated with SP episode intensity	[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.
Childhood sexual abuse	[35] – SP hallucinations in those with a history of CSA vs those with no history of CSA. M for intruder, 5.67 vs 2.91, for incubus, 4.78 vs 2.58 hallucinations	[35] – SP hallucinations in those with a history of CSA vs those with no history of CSA. M for vestibular-motor hallucinations, 4.05 vs 3.07 ns
Physical abuse	[22] – Associated with SP episode intensity	
2. Personality, intelligence and beliefs		
Dissociative experiences	[35] – Dissociative experiences correlated with intruder (r = 0.35), incubus (r = 0.31), and vestibular-motor	[39] – Ns predictor of incubus hallucinations in a multiple predictor model.

Table 2 (*continued*)

Associated variable	Association	No association
	($r = 0.33$) hallucinations [39] – Dissociative experiences significant predictor of intruder and vestibular-motor hallucinations. All in multiple predictor models	
Imaginativeness	[22] – Associated with SP episode intensity	
Absorption	[35] – Absorption correlated with intruder ($r = 0.40$), incubus ($r = 0.33$), and vestibular-motor ($r = 0.38$) hallucinations	
Mindfulness		[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.
Sensory imagery	[39] – Vivid sensory imagery significantly predicted intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models	
Daydreaming frequency		[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.
Daydreaming style	[39] – Positive constructive daydreaming significantly predicted intensity of vestibular-motor hallucinations in a multiple predictor model	[39] – Ns predictor of intruder or incubus hallucinations in multiple predictor models
Social imagery	[64] – Distress felt during sensed presence hallucinations associated with dysfunctional social imagery ($r = 0.19$)	
Paranormal beliefs	[39] – Paranormal beliefs associated with intruder frequency and intensity, and vestibular-motor frequency. All significant in multiple predictor models	[39] – Ns predictor of incubus hallucinations in a multiple predictor model
Conspiracist beliefs		[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.

Note. CI = 95% confidence intervals, M = mean, ns = not significant, OR = odds ratio, SD = standard deviation, SNP = single nucleotide polymorphism, SP = sleep paralysis. Article reference numbers are shown in square brackets.

participants who experienced sleep paralysis in a laboratory environment showed higher diastolic and lower systolic blood pressure in sleep-onset REM periods (SOREMP; REM episodes that occur abnormally early in the sleep cycle, defined in this study as within 25 min of sleep onset) preceding a sleep paralysis episode compared to SOREMPs without sleep paralysis [42]. Finally, one study showed that complaints of chronic pain were linked to the presence of sleep paralysis experiences [21].

Personality, intelligence, and anomalous beliefs

Relationships with sleep paralysis are shown in Table 2. Measures of personality traits, such as the Eysenck personality questionnaire, do not appear to be related to sleep paralysis frequency [22,26,27,38]. Levels of waking state dissociative experiences, involving depersonalisation, derealisation, and amnesia, were found to be related to both sleep paralysis frequency [36] and the frequency/intensity of all three hallucination types [35]. The degree to which individuals become absorbed in their mental fantasy has been linked to hallucination frequency and intensity [35], but another study did not find absorption to be related to overall episode frequency [36]. It should be noted that these studies were conducted in groups containing individuals who had experienced CSA. However, another study in a more general sample found dissociative experiences to be linked to sleep paralysis frequency, and also to intruder and V-M hallucinations [39].

In a university sample, a composite measure of "imaginativeness" that comprised scales of absorption, fantasy proneness, magical thinking, imagery vividness, paranormal and mystic beliefs, perceptual aberration, and unusual sensory experiences, was related to sleep paralysis frequency and intensity [22]. It was unclear which of these components was driving the relationship. In the same sample, those reporting sleep paralysis compared to others showed greater hypnotisability assessed both subjectively and objectively [22]. Sensory imagery vividness was found to not be related to sleep paralysis frequency, but significantly predicted the intensity of all three hallucination types [39]. Frequency of daydreams was not related to sleep paralysis, but daydreaming style, particularly positive constructive daydreams, was related to V-M hallucinations [39].

One study looked at intelligence in relation to sleep paralysis and found a significant negative association between intelligence quotient (IQ) and reports of lifetime isolated sleep paralysis, fearful isolated sleep paralysis, and fearful recurrent isolated sleep paralysis [29]. When levels of paranormal belief have been examined, mixed evidence has been found, with some studies finding no relationship [19,22], whilst others report links [39]. Paranormal beliefs have also been related to intruder and V-M hallucinations during sleep paralysis [39]. Levels of belief in conspiracy theories have been found not to relate to sleep paralysis [39].

Sleep-related factors

Relationships between sleep-related variables and sleep paralysis are shown in Table 3. Symptoms of automatic behaviour, the spontaneous production of purposeless verbal or motor behaviour without conscious control, an auxiliary symptom of narcolepsy [44], were found to predict sleep paralysis independently of age, physical health, sleep problems, mental health, and psychiatric medication [20]. In a specific sample of 747 Hmong immigrants living in the USA, sleep paralysis was higher in individuals who had a relative who had suffered sudden unexpected nocturnal death syndrome (SUNDS) (29% of the sample reported having a relative die of SUNDS) [21] as compared to those who did not have a relative die of SUNDS.

A number of 'anomalous' sleep experiences appear to be more common in people who also experience sleep paralysis. Experiencing nightmares appears to be common in those with sleep paralysis, as an association has been found in a number of studies [22,24,32]. Responses to whether participants have ever experienced a lucid dream, which is a dream in which the dreamer, whilst dreaming, is aware that they are in a dream [45], showed no relationship with sleep paralysis frequency in two studies [22,46]. However, in another study that used a continuous measure of lucid dreaming frequency, a significant association with sleep paralysis was found [39]. This was particularly true of sleep paralysis

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

ARTICLE IN PRESS

Table 3
Associations between sleep paralysis episodes and other sleep-related variables.

Associated variable	Association	No association
Section 1 – Frequency of sleep paralysis episodes		
Automatic behaviour	[20] – Significant predictor of SP in multiple predictor model, OR (CI) = 2.11 (1.42–3.13)	
Relative with sudden-unexpected nocturnal death syndrome	[21] – During sleep SP, OR = 1.68, during wake SP, OR = 2.18	
Nightmares	[22] – Mean nightmare factor score in those with SP vs those without SP, M (SD) = 0.52 (2.47) vs −0.62 (2.06) [24] – Prevalence of SP with nightmares: never (5.0%), seldom (8.4%), sometimes (15.2%), often (22.4%), always (41.8%). OR (CI) = 1.52 (1.40–1.65), 2.76 (2.57–2.96), 4.06 (3.67–4.49), 7.64 (6.68–8.75). All relative to no nightmares [32] – Prevalence of SP with vs without nightmares, 10.8% vs 5.9%. Significant in a multiple predictor model, OR (CI) = 1.90 (1.10–3.10)	
Lucid dreaming	[39] – Significant association, $r = 0.15$	[22] – No association [46] – Prevalence of SP in narcolepsy patients with lucid dreams vs prevalence of SP in narcolepsy patients without lucid dreams, 56.1% vs 66.7% ns.
Hypnopompic hallucinations	[20] – Prevalence of hypnopompic hallucinations in individuals with SP vs those without SP, 14.4% vs 5.75%, OR (CI) = 3.59 (1.55–8.28)	
Exploding head syndrome	[47] – Prevalence of EHS in individuals with fearful SP vs those without SP, 36.5% vs 13.5%	
Out-of-body experiences	[22] – Mean OBE factor score in those with SP vs those without SP, M (SD) = 0.19 (0.39) vs 0.08 (0.27)	
Sleep-onset REM periods	[50] – Five out of six induced SP episodes occurred following SOREMP [42] – Six out of eight induced SP episodes occurred following SOREMP	
Sleep deprivation	[50] – Six SP episodes elicited from 64 sleep interruptions (9.4%) [42] – Eight SP episodes elicited from 184 sleep interruptions (4.3%)	
EEG alpha activity	[50] – Abundant 8–13 Hz alpha EEG activity during SP episodes	
Amount of stage 1 sleep	[42] – Mins of stage 1 sleep in sleep periods with SP vs sleep periods without SP, M (SD) = 43.75 (9.26) vs 32.54 (10.23)	
Vigilance before sleep-onset REM periods	[42] – Reaction times (ms) before SOREMP with SP vs SOREMP without SP, M (SD) = 532.44 (249.99) vs 402.01 (102.52)	
Subjective sleep quality	[18] – Significant predictor of SP in a multiple predictor model, β (CI) = 2.72 (1.16–4.29) [17] – Significant predictor of SP in a multiple predictor model, OR (CI) = 1.28 (1.05–1.56) [24] – Prevalence of SP in those with good sleep vs prevalence of SP in those with bad sleep in SP, 6.3% vs 13.1%, OR (CI) = 1.22 (1.15–1.29). Significant in a multiple predictor model [32] – Prevalence of SP in those with good sleep vs prevalence of SP in those with bad sleep in SP, 5.8% vs 10.1% [28] – Prevalence of SP in those with good sleep vs prevalence of SP in those with bad sleep in SP, 4.1% vs 9.2%, OR (CI) = 0.47 (0.35–0.64) – good sleep as reference [39] – Significant predictor of SP in a multiple predictor model	
Excessive daytime sleepiness	[18] – Significant predictor of SP in a multiple predictor model, β (CI) = 2.49 (0.45–4.52) [24] – Prevalence of SP in those with EDS vs those without EDS, 6.8% vs 8.3%, OR (CI) = 1.23 (1.16–1.30). Significant predictor of SP in a multiple predictor model	[20] – Prevalence of EDS in those with SP vs those without, 19.2% vs 8.3%. Ns in a multiple predictor model [25] – No association
Sleep hygiene		
Bedtime	[24,28] – Before 22:00 h as reference. 22:00–24:00 h reduced SP odds OR = 0.69–0.91. 00:00 to 02:00 h and later increased SP odds OR = 1.28–1.36	[32] – No difference in SP prevalence between before 12:00 h and after 12:00 h
Wake-up time		[32] – No difference in SP prevalence between before 06:30 h and after 06:30 h
Naptime	[32] – Presence of naps associated with increased odds of SP, OR (CI) = 2.30 (0.90–6.00) [24,28] – Excessively long naps (>30 min) associated with increased odds of SP OR = 1.08–1.26	
Sleep duration	[32] – Less than 6 h associated with increased odds of SP compared to 6 h or more, OR (CI) = 3.50 (1.80–6.70) [24,28] – 7–8 h as reference. Increasing odds of SP with	

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 3 (*continued*)

Associated variable	Association	No association
	shorter (greatest odds at <5 h, OR = 1.47–1.82) and longer (greatest odds at >9 h, OR = 1.43–1.71) durations	
Initiating sleep	[24] – Difficulty initiating sleep associated with increased odds of SP. OR (CI) = 1.22 (1.14–1.30)	
Sleeping position	[53] – Sleeping position during SP – supine (58.07%), prone (7.95%), side (16.87%), variable (17.11%). No effect of position when falling asleep [54] – Majority of SP episodes occur in the supine position (57.9%–83.8%). No effect of position when falling asleep	
Time during sleep	[53] – SP more frequent at beginning of sleep compared to middle or end [55] – Hypnomesic SP episodes most frequent overall. Hypnagogic SP episodes most frequent close to bedtime. Hypnomesic SP episodes most frequent in the middle of the sleep period. Hypnopompic SP episodes most frequent near the end of sleep	
Shift work	[51] – Prevalence of SP in those who perform shiftwork vs those who do not perform shiftwork, 48% vs 36%	
Section 2 – Intensity of sleep paralysis episodes/associated hallucinations		
Lucid dreaming	[39] – SP featuring intense vestibular-motor hallucinations significant predictor of lucid dreaming frequency, β (CI) = 0.36 (0.03–0.68)	[39] – SP featuring intruder hallucinations ns predictor of lucid dreaming frequency in a multiple predictor model. SP featuring incubus hallucinations not associated with lucid dreaming (frequency, $r = -0.004$; intensity, $r = 0.03$).
Subjective sleep quality	[39] – Subjective sleep quality significant predictor of intruder and incubus hallucination frequency. All in multiple predictor models.	[39] – Ns predictor of intruder intensity, incubus intensity, vestibular-motor frequency, or vestibular-motor intensity. All in multiple predictor models.
Timing during sleep	[53] – Intruder hallucinations less common at the end of sleep, M SD = 2.58 (0.99) compared to beginning (2.93 (0.95)) and middle (2.88 (0.97). Same results for incubus, end (2.30 (0.97), beginning (2.48 (0.98) and middle (2.49 (0.99)). Greater vestibular-motor hallucinations at the beginning of sleep (2.11 (1.00) compared to the middle (1.81 (0.89) or end (1.80 (0.86) of sleep	
Sleep walking/talking	[22] – Both sleep walking and sleep talking associated with SP episode intensity	

Note. CI = 95% confidence intervals, EDS = excessive daytime sleepiness, EEG = electroencephalography, EHS = exploding head syndrome, M = mean, ns = not significant, OBE = out-of-body experiences, OR = odds ratio, SD = standard deviation, SP = sleep paralysis. Article reference numbers are shown in square brackets.

episodes featuring V-M hallucinations [39]. Hypnopompic (sleep-offset) hallucinations were a significant predictor of sleep paralysis, though it is possible that these hallucinations were not occurring during sleep paralysis [20]. Exploding head syndrome (hearing loud noises such as explosions at wake–sleep or sleep–wake transitions) is more common in individuals reporting episodes of fearful isolated sleep paralysis than in those not reporting sleep paralysis (37% vs 14%) [47]. Out-of-body experiences (OBE), often associated with REM sleep [48,49], were reported to be associated with sleep paralysis although the possibility that the OBE was a hallucination experienced during sleep paralysis cannot be ruled out [22].

Two experimental studies were able to induce episodes of sleep paralysis in a laboratory environment [42,50]. Sleep can be distinguished into different stages. Currently a broad distinction is made between rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. Within NREM, sleep is further divided into stage 1, stage 2, and stage 3. A typical sleep cycle comprises of the individual progressing from stage 1 to stage 2 to stage 3, and then back to stage 1 before entering a period of REM sleep. In a study of 16 participants, who had all self-reported having sleep paralysis at least twice, it was shown that SOREMPs triggered by sleep disruption, led to six episodes of sleep paralysis (10% of all SOREMPs), with all but one reporting multisensory hallucinations and feelings of fear and dread [50]. This study induced six episodes from 64 sleep disruptions (9%). Polysomnography (PSG) recordings showed sleep paralysis episodes to contain abundant alpha (8–13 Hz) EEG activity, indicative of relaxed wakefulness, i.e., not expected during sleep, occurring with persistence of muscle atonia during sleep paralysis [50].

A second study using 13 participants, again selected based on self-reporting at least two lifetime episodes of sleep paralysis, induced a total of eight episodes of sleep paralysis from 184 sleep interruptions (4%) using a multi-phasic sleep wake schedule [42]. Six of the eight episodes occurred following a SOREMP whilst no episodes occurred following a regular REM period. The other two sleep paralysis episodes occurred immediately after sleep onset and, even though PSG data suggested a REM period was imminent, activity could not be scored as REM in the conventional manner [42]. Participants who had episodes of sleep paralysis in the laboratory showed increased amounts of stage 1 sleep, compared to participants who did not experience sleep paralysis. Furthermore, poorer performance on a vigilance task (longer reaction times) was found before a SOREMP with sleep paralysis. More generally, poorer vigilance task performance, and increased subjective sleepiness were found in the sleep paralysis group [42]. It should be noted in both of these studies, SOREMPs were defined as REM periods occurring within 25 min of sleep onset. This differs from the American Academy of Sleep Medicine definition, which defines a SOREMP as REM sleep occurring within 15 min of sleep onset [1]. Related to sleep disruption, shiftwork was related to a higher self-reported prevalence of sleep paralysis (48% vs 36%) [51].

Subjective sleep quality, as assessed via measures such as the Pittsburgh sleep quality index [52], was found to be related to sleep paralysis in multiple studies [17,18,24,28,32,39]. Excessive daytime sleepiness was associated with sleep paralysis in two studies [18,24], with another two finding no association [20,25]. Particular aspects of 'sleep hygiene' (which refers to habits and practices that are conducive to sleeping well on a regular basis) were associated with greater odds of reporting sleep paralysis. Specifically, excessively short (<6 h) [24,28,32] or long (>9 h) [24,28] sleep duration and napping [32], especially long naps (>2 h) [24,28], were associated

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 4
Associations between sleep paralysis episodes and sleep disorders.

Disorder	Association	No association
Insomnia disorder		[20] – Prevalence of insomnia disorder in individuals with SP vs those without SP, 4.3% vs 1.7%, ns in a multiple predictor model
Symptoms of insomnia:		
Early morning awakenings		[20] – Prevalence of early morning awakenings in individuals with SP vs those without SP, 21.3% vs 11.3%, ns in a multiple predictor model
Disrupted sleep	[56] – Significant predictor of SP in a multiple predictor model, OR (CI) = 2.54 (1.36–4.76)	[20] – Prevalence of disrupted sleep in individuals with SP vs those without SP, 38.6% vs 19.6%, ns in a multiple predictor model
Difficulty falling asleep		[20] – Prevalence of difficulty falling asleep in individuals with SP vs those without SP, 25.7% vs 12.6%, ns in a multiple predictor model
Non-restorative sleep	[20] – Prevalence of non-restorative sleep in individuals with SP vs those without SP, 32.8% vs 12.5%, OR (CI) = 1.85 (1.18–2.90)	
Obstructive sleep apnoea	[57] – Prevalence of SP in patients with obstructive sleep apnoea vs prevalence of SP in controls, 20% vs 5%	[20] – Prevalence of obstructive sleep apnoea in individuals with SP vs those without SP, 5.6% vs 1.3%, ns in a multiple predictor model
Circadian rhythm disorder		[20] – Prevalence of circadian rhythm disorder in individuals with SP vs those without SP, 3.4% vs 1.3%, ns in a multiple predictor model
Narcolepsy	[46] – Prevalence of SP in patients with narcolepsy vs prevalence of SP in controls, 58.5% vs 15.1% [58] – Prevalence of SP in patients with narcolepsy showing low levels of CSF hypocretin-1 vs prevalence of SP patients showing normal levels of CSF hypocretin-1, 52.5% vs 33%. No significant difference in SP severity, M (SD) = 3.85 (1.65) vs 2.70 (0.70)	[20] – Prevalence of narcolepsy in individuals with SP vs those without SP, 1.1% vs 0.1%, ns in a multiple predictor model
Idiopathic hypersomnia		[60] – Prevalence of SP in hypersomnia vs narcolepsy and narcolepsy with cataplexy, 40% vs 32% and 53%, ns [61] – Prevalence of SP in hypersomnia with vs without long sleep time, 28.6% vs 26.7%, ns
Confusional arousals		[20] – Prevalence of confusional arousals in individuals with SP vs those without SP, 4.2% vs 0.6%, ns in a multiple predictor model
Sleep talking		[20] – Prevalence of sleep talking in individuals with SP vs those without SP, 16.5% vs 9.7%, ns in a multiple predictor model [22] – No association
Sleep walking		[22] – No association
Periodic limb movement disorder		[20] – Prevalence of periodic limb movement disorder in individuals with SP vs those without SP, 2.2% vs 0.4%, ns in a multiple predictor model
Sleep-related leg cramps	[20] – Prevalence of sleep-related leg cramps in individuals with SP vs those without SP, 4.9% vs 0.7%, OR (CI) = 4.02 (1.65–9.78)	
Sleep starts		[20] – Prevalence of sleep starts in individuals with SP vs those without SP, 7.1% vs 1.5%, ns in a multiple predictor model

CI = 95% confidence intervals, M = mean, ns = not significant, OR = odds ratio, SD = standard deviation, SP = sleep paralysis. Article reference numbers are shown in square brackets.

with increased odds of sleep paralysis. Long self-reported sleep latency (>30 min) and difficulty initiating sleep were related to an increased likelihood of reporting sleep paralysis [24,32]. Bedtimes also showed some relationship to sleep paralysis. With a bedtime of 22:00 h as the reference group, the odds of having ever experienced sleep paralysis (compared with never having experienced sleep paralysis) were significantly reduced in participants reporting a bedtime between 22:00 h and midnight, and significantly increased in participants reporting a bedtime later than midnight [24,28].

Sleep paralysis episodes are most likely to occur when the individual is lying in the supine position (on the back) [53,54]. Despite this, it was shown that people who had reported experiencing at least one episode of sleep paralysis, as compared to those who had not, were no more likely to fall asleep lying in the supine position [53].

Distribution of sleep paralysis events show a weakly bi-modal distribution with 28% of episodes occurring within an hour after bedtime and 26% occurring five or more hours following bedtime [55]. Perhaps unsurprisingly, hypnagogic (when falling asleep) episodes of sleep paralysis were most prominent in the first hour after bedtime, hypnomesic (sometime during sleep) episodes most common between 1 and 3 h after bedtime, and hypnopompic episodes were most common between 6 and 12 h post bedtime [55]. Overall, hypnomesic episodes were most common [55].

Sleep disorders

Associations between sleep paralysis and other sleep disorders are shown in Table 4. In a large population-level study, insomnia disorder was not associated with increased prevalence of sleep paralysis, however non-restorative sleep, a common symptom of insomnia [1], was associated with increased sleep paralysis rates (33% vs 13%) [20]. In a further study, disrupted sleep (defined as waking up repeatedly during the night) was a significant predictor of sleep paralysis, with those reporting disrupted sleep more than

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 5
Associations between sleep paralysis and symptoms of psychiatric illness.

Associated variable	Association	No association
Section 1 – Frequency of sleep paralysis episodes		
General mental health	[18] – Increased SP episode frequency associated with worse mental health, β (CI) = −4.80 (−9.12–0.49) [22] – Mean psychopathology factor score in those with SP vs those without SP, M (SD) = 2.92 (12.18) vs −2.70 (10.34) [25] – 68.3% of SP episodes attributed to poor psychological health [24] – Prevalence of SP in those with poor mental health vs those with good mental health, 11.9% vs 5.3%, OR (CI) = 1.54 (1.45–1.63) [28] – Prevalence of SP in those with poor mental health vs those with good mental health, 9.36% vs 4.64%, OR (CI) = 1.23 (1.16–1.29) [42] – More general neurotic complaints in those with SP vs those without SP, M (SD) = 1.92 (1.34) vs 0.53 (0.57)	[26] – General mental health complaints in those who experience SP vs those who do not, M (SD) = 1.80 (1.90) vs 1.26 (1.83) ns [27] – General mental health complaints in those who experience SP vs those who do not, M (SD) = 3.05 (3.04) vs 2.13 (2.35) ns
Anxiety symptoms	[17] – Significant predictor of SP in a multiple predictor model, OR (CI) = 1.39 (1.13–1.71) [38] – in precare participants, correlation with SP, $r = 0.33$ [39] – Significant predictor of SP in a multiple predictor model	[56] – Ns in a multiple predictor model OR (CI) = 0.75 (0.50–1.72)
Anxiety sensitivity	[19] – Higher anxiety sensitivity scores in those with SP vs those without SP, M (SD) = 21.1 (10.4) vs 16.7 (7.4) [29] – Significant correlations between anxiety sensitivity and both lifetime ($r = 0.35$) and recurrent ($r = 0.23$) fearful SP	
Social anxiety	[64] – SP distress associated with dysfunctional social imagery	
Depressed mood	[37] – Higher depression symptom score in those with SP vs those without SP, M (SD) = 20.6 (12.1) vs 10.1 (10.0) [36] – Higher depression symptom score in those with SP vs those without SP, M (SD) = 18.0 (10.5) vs 11.5 (9.30) [56] – Significant predictor of depressed mood in a multiple predictor model, OR (CI) = 4.48 (1.55–12.98)	[17] – Ns in a multiple predictor model, OR (CI) = 1.23 (0.99–1.53) [39] – Ns in a multiple predictor model
Hypomania	[38] – Significant correlation with SP, $r = 0.32$	
Section 2 – Intensity of sleep paralysis episodes/associated hallucinations		
Anxiety symptoms		[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.
Social anxiety	[63] – SP with sensed presence vs SP without sensed presence in socially anxious individuals, M (SD) = 45.6 (17.88) vs 24.6 (14.71)	
Depressed mood	[35] – Depressed mood correlated with intruder ($r = 0.37$), incubus ($r = 0.32$), and vestibular-motor ($r = 0.31$) hallucinations [63] – Depressed mood in those with SP with sensed presence vs those without SP, M (SD) = 3.02 (0.96) vs 1.36 (1.18)	[39] – Ns predictor of intruder, incubus, and vestibular-motor hallucinations. All in multiple predictor models.

Note. CI = 95% confidence intervals, M = mean, ns = not significant, OR = odds ratio, SD = standard deviation, SP = sleep paralysis. Article reference numbers are shown in square brackets.

five nights per month being three times more likely to experience sleep paralysis compared to those reporting less than five nights of disrupted sleep per month [56]. One study reported increased sleep paralysis in obstructive sleep apnoea (OSA) patients compared to controls (20% vs 5%) [57], however no independent association was found in a separate study [20]. In studies of narcoleptic populations, patients had a higher sleep paralysis prevalence than controls (59% vs 15%) [46]. Another study showed differing sleep paralysis prevalence rates in narcolepsy patients with varying concentrations of cerebral spinal fluid (CSF) hypocretin 1 [58], which is a biomarker for narcolepsy [59]. Those with below 200 pg/ml concentration exhibited more frequent episodes of sleep paralysis (52%) than those with a normal concentration of CSF hypocretin-1 (33%). Despite the increased *frequency* of episodes, there were no significant differences in episode *severity* between groups [58]. In a different study also with narcolepsy patients, those who experienced narcolepsy with idiopathic hypersomnia showed no significant differences in sleep paralysis frequency compared to those with narcolespy without hypersomnia [60]. Prevalence of sleep paralysis did not differ between two groups of idiopathic hypersomnia patients, when comparing patients who either did or did not have a long sleep time [61].

Using minimal International classification of sleep disorders 90 (ICSD-90) criteria [62], and a large population-level sample [20], those with sleep paralysis, as compared to those without, showed higher rates of a number of dyssomnias (disorders of getting to sleep and staying asleep) and parasomnias (disorders of abnormal/unusual behaviours during sleep). Ohayon and colleagues found that no disorder other than nocturnal leg cramps predicted sleep paralysis frequency in a multiple predictor model [20,22].

Symptoms of psychiatric illness

Relationships between symptoms of psychiatric illness and sleep paralysis are shown in Table 5. In non-clinical samples, multiple studies have examined the links between sleep paralysis and general mental health problems. The latter have been indexed by scales such as the mental health short-form, which gives an index of a person's quality of mental health. These studies have shown that poorer general mental health is significantly associated

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

Table 6
Associations between sleep paralysis episodes and psychiatric illness and medication use.

Disorder	Association	No association
Psychiatric illness		
Post-traumatic stress disorder	[19] – Prevalence of SP in patients with PTSD vs prevalence of SP in controls, 27.8% vs 5.6%. [29] – Individuals with PTSD significantly more likely to have recurrent/fearful SP than those without [65] – Prevalence of SP in patients with PTSD vs prevalence of SP in controls, 67% vs 22% [66] – Prevalence of SP in patients with PTSD vs prevalence of SP in controls, 76% vs 25.9% [67] – Prevalence of SP in patients with PTSD vs prevalence of SP in controls: site 1: 66.7% vs 19.4%; site 2: 100% vs 21.8% [35] – PTSD correlated with increased intruder ($r = 0.38$), incubus ($r = 0.29$), and vestibular-motor ($r = 0.29$) hallucinations	[19] – PTSD not associated with cognitive symptoms (such as fear and distress) of SP [37] – Current (25% vs 20%) or lifetime (56% vs 48%) PTSD rates in those with SP vs those without both ns
Panic disorder	[19] – Prevalence of panic disorder in individuals with SP vs those without SP, 30.6% vs 0% [30] – Individuals with panic disorder report more SP than individuals with other anxiety disorders, and healthy controls [67] – Prevalence of SP in patients with panic disorder vs prevalence of SP in controls, 55.6% vs 18.9%	[37] –Difference in current (0% vs 3%) or lifetime (6% vs 6%) panic disorder rates in those with SP vs those without both ns
Panic attacks	[31] – Prevalence of SP in patients with panic attacks vs prevalence of SP in controls, 32% vs 15% [40] – Those who had SP more likely to experience panic attacks [37] – Prevalence of panic attacks in individuals with SP vs those without SP, 49% vs 10%	
Diagnosis of an anxiety disorder	[31] – SP more frequent in those with an anxiety disorder vs no anxiety disorder	[29] – Correlation between SP and interview measures of anxiety ns [20] – Ns in a multiple predictor model
Co-morbid anxiety disorder	[23] – Prevalence of SP in patients with a co-morbid anxiety disorder vs prevalence of SP in patients with a single anxiety disorder, 35% vs 11%	
Depression	[29] – Significant correlation with recurrent fearful SP, $r = 0.27$	[20] – Ns in a multiple predictor model [37] – Current (25% vs 15%) or lifetime (31% vs 28%) depression in those with SP and without. Both Ns
Bi-polar disorder	[20] – Significant predictor of SP in a multiple predictor model, OR (CI) = 2.11 (1.42–3.13)	
Substance/alcohol abuse disorder	[37] – Lifetime substance abuse disorder more common in individuals with SP vs those without SP, 69% vs 45%.	[37] – No association with current substance abuse disorder in individuals with SP vs those without SP, 25% vs 15%
Number of diagnoses	[29] – Significant correlations with lifetime (r = 0.23), fearful (r = 0.35), and recurrent fearful (r = 0.31) SP	
Psychiatric medication use		
Anxiolytic medication	[20] – Higher usage in individuals with SP vs without SP, 8.8% vs 3.0%, OR (CI) = 4.91 (1.50–16.11)	[23] No association
Anti-depressant medications		[20,23,29] – No association [56] – Ns in a multiple predictor model, OR (CI) = 1.38 (0.64–2.96)
Hypnotic medications		[20] – No association

CI = 95% confidence intervals, ns = not significant, OR = odds ratio, SP = sleep paralysis. Article reference numbers are shown in square brackets.

with frequency of sleep paralysis [18,22,24,25,28,42], although two studies of Nigerian health workers did not find a relationship [26,27].

Mirroring the clinical findings, anxiety-related symptoms appear to be associated with sleep paralysis [17,38,39], though this was not found in all studies [56]. Furthermore, anxiety sensitivity (fear of the physical symptoms of anxiety) showed a small but significant relationship with the presence of sleep paralysis in one study [19]. A second study showed anxiety sensitivity to be significantly correlated with lifetime and recurrent fearful sleep paralysis [29]. Social anxiety symptoms also appear to be related to sleep paralysis, with a study finding participants who experienced sleep paralysis with a 'felt presence' hallucination showing higher levels of social anxiety than participants who experienced sleep paralysis without hallucinations [63]. Felt presence hallucinations during sleep paralysis have also been related to the amount of distress associated with an episode of sleep paralysis. The level of distress related to sleep paralysis episodes was associated with dysfunctional social imagery [64].

Higher scores on self-report measures of depressed mood have been associated with sleep paralysis frequency [29,36,37]. It has also been shown that this relationship remains significant after anxiety symptoms have been controlled for [56]. However, other multivariate studies assessing multiple predictors (such as depressed mood, anxiety symptoms, alcohol intake, threatening events, sleep quality, dissociative experiences, daydreaming, and sensory imagery) did not find depressed mood to independently predict sleep paralysis [17,39].

Psychiatric disorders and medication use

Results focussing on sleep paralysis in relation to psychiatric illness and medication are displayed in Table 6. Patients with a diagnosis of post-traumatic stress disorder (PTSD) in American, Cambodian, and Chinese samples all showed higher prevalence of sleep paralysis (between 65 and 100%) compared to healthy controls (20–25%) [65–67]. In a study focussing on fearful isolated sleep paralysis in patients with panic attacks, individuals who also

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

were diagnosed with PTSD were significantly more likely to experience recurrent fearful isolated sleep paralysis compared with patients with panic attacks but without PTSD [29]. No differences were found with regards to lifetime isolated sleep paralysis in those with and without PTSD. Significant positive correlations between PTSD symptoms and intruder, incubus, and vestibular-motor (V-M) hallucination frequency and intensity during sleep paralysis episodes were found in one study [35]. This suggests that PTSD is associated with both the frequency and intensity of sleep paralysis episodes, though a separate study did not find a link between PTSD and fear/distress experienced during sleep paralysis [19]. In a sample of 142 African Americans, whilst both current (25% vs 20%) and lifetime (56% vs 48%) PTSD diagnosis rates were numerically higher in participants with sleep paralysis, this study did not find statistically significant differences [37].

Significantly higher prevalence rates of sleep paralysis in patients with panic disorder were also found. Lifetime prevalence rates of 56–59% were reported for panic disorder patients which was significantly higher than those without panic disorder [30,67], whose prevalence was approximately 19% [67], though not all studies found this difference [37]. Another study found 31% of participants who *had* experienced sleep paralysis met diagnostic criteria for clinical or subclinical panic disorder, whilst none of those who did *not* experience sleep paralysis met diagnostic criteria [19]. In a study comprising African American and Caucasian participants, patients with panic disorder were significantly more likely to experience recurrent sleep paralysis (defined as four+ episodes per year) (36%) as compared to patients with other anxiety disorders, such as obsessive-compulsive disorder, social phobia, and generalised anxiety disorder (11%) [30]. In a further study, individuals with frequent sleep paralysis were more likely to have experienced panic attacks than those with less frequent/no sleep paralysis, though exact prevalence rates were not reported [40]. In other studies focussing on panic attacks, sleep paralysis was more common in individuals with panic attacks (32–49%) compared to those without (10–15%) [31,37].

Studies that have considered anxiety disorders more generally have shown that participants with self-reported anxiety disorders were significantly more likely to experience sleep paralysis than those without [31]. However, other studies have failed to find anxiety disorders to be a significant predictor of sleep paralysis [20,29]. One study found slightly higher prevalence rates for patients with panic disorder (21%) and social phobia (22%) compared with generalised anxiety disorder (16%), though differences were not significant (this may be due to the low sample size, N = 62). This study did however find a significant association between the presence of sleep paralysis and a co-morbid anxiety disorder (35%), compared to being diagnosed with a single anxiety disorder (11%) [23]. Similarly, another study found that the number of fearful sleep paralysis episodes was positively correlated with the total number of anxiety and mood disorder diagnoses [29].

There is less evidence for an association between sleep paralysis and depression. Specifically, sleep paralysis prevalence was not significantly higher in a group meeting diagnostic criteria for depression (31%) compared with a group who did not meet the criteria (28%) [37]. In a study of outpatients with anxiety disorders, a comorbid depression diagnosis was not associated with an increased prevalence in sleep paralysis compared to patients without comorbid depression [23]. In a population level survey of over 4000 individuals, depression (as assessed by DSM-IV criteria) was not significantly related to sleep paralysis in a multiple predictor model (including age, physical health, sleep problems, bipolar disorder, and psychiatric medication) [20]. One study however did find a significant correlation between depression and recurrent fearful sleep paralysis, with both lifetime and lifetime fearful sleep paralysis showing no relationship to depression [29]. There has been just one study looking at bipolar disorder and sleep paralysis. Here it was found that bipolar disorder occurred more frequently in a severe sleep paralysis group, defined here as at least one episode per week (19%), compared with a no sleep paralysis group (2%) [20]. Sleep paralysis has been associated with a lifetime, but not current, diagnosis of alcohol/substance abuse disorder [37].

There is little evidence regarding the association between psychiatric medication use and sleep paralysis (see Table 6). Whilst one study found higher use of anxiolytic medication in individuals with weekly sleep paralysis as compared to those without [20], the reasons for this medication use was unclear. Other studies have failed to link anxiolytic medications with sleep paralysis [23]. The use of anti-depressant [20,23,29,56] and hypnotic medications [20] was also found not to be related to sleep paralysis frequency.

Discussion

This review set out to investigate variables associated with episodes of sleep paralysis. Whilst strengths in the current literature include a wide range of variables investigated and a diverse array of samples, plus a number of relatively large-scale studies, limitations currently exist that should be addressed in future research.

As described in the introduction, a great deal of variation in terms of measures to assess sleep paralysis was noted (as shown in Table 1). Furthermore, very few studies reviewed provided any evidence regarding the psychometric validity/reliability of their chosen measure. The Waterloo unusual sleep experiences questionnaire (WUSEQ) has been shown to have a reliable three-factor structure [2,10] and internal reliability (as assessed by Cronbach's alpha) was shown to be good in one study that reported it [39]. Furthermore, research to date has relied on retrospective reporting. As many studies assessed lifetime prevalence of sleep paralysis, the reliability of such measures is questionable. Some studies using a prospective measure of sleep paralysis have found a similar factor structure of hallucinations to when retrospective measures are used [68,69]. However, no studies to date have assessed associated variables using a prospective design. This is an important area for future research as it will allow for a more accurate assessment of sleep paralysis episodes and their predictors.

Few studies have distinguished between sleep paralysis episodes with and without clinically significant distress (i.e., fearful sleep paralysis) [29]. This is an important distinction to make when considering the clinical impact episodes are having on an individual. Whilst a large number of variables have shown an association with sleep paralysis, future studies should work to further isolate the factors that specifically predict episodes where clinically significant levels of distress are present. This would help further refine our knowledge regarding relationships between sleep paralysis and other variables, but also prove beneficial in aiding clinical interventions for those whose lives are being adversely affected by severe episodes.

Similarly, some episodes of sleep paralysis are reported as positive experiences, especially episodes containing V-M hallucinations [10]. An interesting future investigation would be to look at whether there are any characteristics that specifically predict these 'blissful' sleep paralysis experiences and whether positive and fearful episodes can be dissociated based on the variables that predict them.

From the studies conducted to date on the variables associated with sleep paralysis, no longitudinal investigations have been conducted. Whilst the cross-sectional research carried out so far has been useful in establishing the existence of important relationships, the direction of these effects cannot be inferred. Future work should use longitudinal designs to move towards

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

understanding the nature of these associations. For example, do high levels of stress and anxiety lead to more frequent episodes of sleep paralysis or do frequent episodes of sleep paralysis result in higher levels of stress and anxiety?

Potential mediators and/or moderators of relationships are also still to be evaluated. Again, studies investigating these issues would be useful in better refining our understanding of which factors precipitate the occurrence of episodes. For example, one prediction could be that anxiety is related to sleep paralysis via an indirect effect of poorer sleep quality [70], which in this instance would be acting as a mediator.

Finally, do certain variables, such as a genetic predisposition, make the underlying risk of sleep paralysis more likely, but are other variables, such as experiencing a particular stressor, responsible for triggering a specific episode? Exploring this potential gene × environment interaction is an important goal for future studies. Anecdotal evidence suggests that sleep paralysis episodes often occur in 'waves', with high frequency periods followed by times of no sleep paralysis. Understanding the factors that trigger possible periods of multiple episodes should be undertaken in the future, and could be achieved by techniques such as sleep diary studies, and also experience sampling, which involves repeated sampling of sleep and waking experiences as they occur in ecologically valid settings.

One limitation of our review is the breadth of the search terms used. As we identified studies based on keywords related to sleep paralysis, it is possible that studies that included sleep paralysis not as a primary aim may have been missed. In particular, this may occur in assessment of other sleep disorders, that only asked about sleep paralysis as a secondary point. Capturing these studies would require searching for articles relating to each sleep disorder specifically and searching through texts to find sleep paralysis measures. A preliminary search for the key words 'insomnia disorder' for example, resulted in over 13,000 hits. Should we do this for every sleep disorder we would obtain many times this number of articles. It was not practical to manually search this number of articles by hand. Readers should keep this limitation in mind when considering the findings of this work.

Despite these limitations, the research reviewed here has provided important insights into variables associated with sleep paralysis. For example, rates of sleep paralysis do appear to be elevated in a number of sleep disorders, especially narcolepsy [46,58]. Studies also suggested an increased prevalence in obstructive sleep apnoea patients [57], nocturnal leg cramps [20], as well as those suffering from insomnia symptoms [20,56]. Interestingly, one study that was not included in this review because of a lack of a control group found a sleep paralysis prevalence rate of 14% in patients with Kleine–Levine syndrome [71] (a sleep disorder characterised by episodes of severe hypersomnolence in association with cognitive, psychiatric, and behavioural disturbances [1]). Without a control group it is hard to say whether this prevalence is increased relative to controls. In the only study to assess the prevalence of multiple sleep disorders in those with or without SP, few links were found [20]. However, it should be noted that diagnoses were made by a trained computer system and not by a sleep medicine clinician. In the clinical setting, sleep paralysis often occurs within the context of disturbed sleep. Preliminary experimental work into the link between SOREMPs and sleep paralysis, and self-report measures of poor sleep quality, support this idea [17,18,24,28,32,39,42,50].

Certain psychiatric groups showed higher rates of sleep paralysis compared to controls, especially patients with PTSD [29,65–67]. Whether sleep paralysis is the cause or the consequence of PTSD is unclear, however some studies focused on patients who had experienced trauma during the Cambodian civil war suggest that sleep paralysis appears as a symptom of the PTSD [65,66]. Qualitative evidence also suggests that the hallmark symptom of 'flashbacks' present in PTSD may actually manifest as hallucinations occurring during sleep paralysis [65,66]. It is also now well-documented that PTSD patients often suffer from 'disruptive nocturnal behaviours' (DNB) such as trauma-related nightmares [72]. Future research should consider DNBs alongside sleep paralysis to ascertain whether these two sleep phenomena are closely linked in this illness. Anxiety disorders generally, and panic disorder in particular, also appear to be associated with a higher prevalence of sleep paralysis [19,23,30,31,40,67], with preliminary evidence suggesting that socially anxious individuals who also experience felt presence hallucinations during wakefulness are more likely to experience sensed presence hallucinations during sleep paralysis, compared to socially anxious individuals who do not experience felt presence hallucinations during wakefulness [63]. These findings are especially important for clinicians, and suggest that experiences of sleep paralysis should be more routinely assessed within certain psychiatric groups. No studies reviewed looked at associations between sleep paralysis and schizophrenia. However one study estimated a prevalence of 15% in schizophrenia patients, though they did not have a control group [73]. This prevalence does not seem abnormally different to prevalence estimates in healthy samples [11], though differential diagnosis between these two conditions is an important consideration [74].

There is still very little work into successful interventions for reducing and/or improving outcomes for individuals suffering from severe cases of sleep paralysis. We did not find clear evidence that sleep paralysis frequency was associated with any psychiatric medications [20,23,29,56]. However, it should be noted that none of the studies on this topic had a primary aim of investigating the efficacy of any particular medication. No studies reviewed systematically investigated whether a specific medication led to a reduction in sleep paralysis episodes. Rather, these studies looked to see whether taking medications predicted sleep paralysis in a cross-sectional design, with minimal information regarding why the medication was being taken, how long the medication was being taken, and what the exact dosages were. It is currently unclear whether these substances are or are *not* effective in reducing sleep paralysis symptoms, as suitable studies to test these hypotheses are yet to be performed. This is a clear area for future research. Research into treatment options for narcolepsy have not yielded many insights into potential treatments for sleep paralysis. A 2007 report by the AASM [75] suggested sodium oxybate as an effective for treatment of cataplexy, daytime sleepiness, and disrupted sleep due to narcolepsy, but evidence for its effectiveness in treating sleep paralysis was mixed and based on lower quality evidence. One double-blind, placebo-controlled trial looking at sodium oxybate for treating narcolepsy found changes in sleep paralysis did not differ from placebo [76]. A follow-up study by the same group did however find a 6 g dosage sodium oxybate to lead to a reduction in episodes [77]. With regards to selective serotonin reuptake inhibitors (SSRIs) and other antidepressant medication, the report concluded that this recommendation is based on anecdotal clinical experience [75]. Many clinicians do prescribe antidepressant medications (such as SSRIs) to those with narcolepsy to help alleviate both symptoms of cataplexy and sleep paralysis. The hypothesised mechanism of action is to enhance postural muscle tone during REM sleep, the stage of sleep most associated with sleep paralysis [74]. Despite this reasonable theoretical link between SSRIs and sleep paralysis, more studies need to be conducted in the future on this important topic. It has been noted that sensory stimulation may help end sleep paralysis episodes, but may be even more unpleasant than the sleep paralysis itself [78].

This review suggests a couple of areas where potential interventions could be targeted. As factors such as anxiety and stress

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

appear to be linked with sleep paralysis [17,19,38,39,43], techniques aiming to reduce levels of these factors may also help in alleviating sleep paralysis episodes. Cognitive behavioural therapy (CBT) has been shown to be useful in reducing levels of anxiety [79] and coping with stress [80], and has also been shown to be effective in various sleep disorders, particularly insomnia [81,82]. A CBT for isolated sleep paralysis manual now exists as a promising therapy [3], however systematic evidence for its effectiveness is still lacking.

Poor subjective sleep quality was shown to be consistently related to sleep paralysis [17,18,24,28,32,39], suggesting that efforts to improve the quality of an individual's sleep could help in reducing sleep paralysis episodes. The importance of sleep hygiene in various parasomnias, including sleep paralysis, has been suggested before [83]. Sleep hygiene education is intended to improve sleep initiation and maintenance, through interventions such as adjusting bedroom temperature, avoiding alcohol close to bedtime, and going to bed and waking up at similar times each day [3,84]. Based on the evidence reviewed here, future work into sleep hygiene education and sleep paralysis is warranted [3].

Very little experimental work into the underlying neurophysiology of sleep paralysis has been performed. It does appear however that episodes are linked with sleep disruption and are particularly associated with SOREMPs [42,50]. Preliminary genetic evidence also suggests a link with altered circadian rhythms [17]. At a neurobiological level, the paralysis associated with REM sleep is believed to be regulated by the GABA and glycine neurotransmitter systems, which are important in the inhibition of motor neurons during REM sleep, so contributing to the muscle atonia [85]. What is currently unknown is why some people regain waking levels of consciousness during REM sleep resulting in an episode of sleep paralysis. Sleep paralysis has been suggested to be a dissociated state, featuring both REM and waking type brain activity [42,50,86–88], though see [89]. Understanding how and why this unique state of consciousness arises is an important topic for future research, not only because it will further our understanding of sleep paralysis, but also because heightened levels of awareness during REM sleep have been suggested as a potential way of studying the emergence of consciousness [90–92].

A large body of research has now been conducted into variables associated with the frequency and intensity of sleep paralysis episodes. Despite a number of limitations, these studies have made an important contribution to our understanding of this unusual, but relatively common, condition. It has been shown that sleep paralysis is highly prevalent in PTSD and anxiety disorders and there are a number of associated variables in the general population. There is now a tremendous opportunity to expand and improve on this literature, to further our understanding of the origins of, and treatment options for, sleep paralysis experiences.

Practice points

- Sleep paralysis should be considered by clinicians when diagnosing sleep disorders, especially in narcolepsy and those exhibiting symptoms of insomnia.
- Sleep paralysis should be assessed more frequently in certain psychiatric groups, particularly those presenting with PTSD and anxiety disorders.
- Clinicians working with patients who experience severe episodes of sleep paralysis should consider the patient's mental and physical health and also the quality of their sleep as possible related factors.
- It is important to consider the level of clinical distress in patients presenting with severe cases of sleep paralysis.

Research agenda

- Reliable and valid measures of sleep paralysis, including distinctions in hallucinatory content and clinical outcomes, should be developed and routinely used, and would be useful in dissociating possible distinctions between and predictors of a) fearful sleep paralysis and b) blissful sleep paralysis.
- Longitudinal designs are required to better tease apart the direction of causation of effects between sleep paralysis and other variables.
- Further laboratory work should be conducted to improve our understanding of the neurophysiology of sleep paralysis episodes.
- Randomised controlled trials with adequate sample sizes of potential treatment options (both psychological and pharmacological) for severe episodes of sleep paralysis.

Conflicts of interest

AMG has provided guidance and educational content for a freely available educational website focused on infant sleep. This website is partially supported by Johnson and Johnson, but they do not have any influence over content and do not advertise on it. She also contributes to BBC Focus Magazine.

Acknowledgements

DD was supported by an Economic and Social Research Council Advanced Quantitative Methods PhD studentship (ES/J500215/1).

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.smrv.2017.05.005.

References

[1] American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
[2] Cheyne JA, Rueffer SD, Newby-Clark IR. Hypnagogic and hypnopompic hallucinations during sleep paralysis: neurological and cultural construction of the night-mare. Conscious Cogn 1999;8:319–37. http://dx.doi.org/10.1006/ccog.1999.0404.
[3] Sharpless BA, Doghramji K. Sleep paralysis: historical, psychological, and medical perspectives. New York, NY: Oxford University Press; 2015.
[4] Ness R. The Old Hag phenomenon as sleep paralysis: a biocultural interpretation. Cult Med Psychiatry 1978;2:15–39.
[5] Law S, Kirmayer L. Inuit interpretations of sleep paralysis. Transcult Psychiatry 2005;42:93–112. http://dx.doi.org/10.1177/1363461505050712.
[6] Davies O. The nightmare experience, sleep paralysis, and witchcraft accusations. Folklore 2003;114:181–203.
[7] French CC, Santomauro J. Something wicked this way comes: causes and interpretations of sleep paralysis. In: Della Sala S, editor. Tall tales about mind brain separating fact from fict. New York: Oxford University Press; 2007. p. 380–98.
[8] French CC, Santomauro J, Hamilton V, Fox R, Thalbourne MA. Psychological aspects of the alien contact experience. Cortex 2008;44:1387–95. http://dx.doi.org/10.1016/j.cortex.2007.11.011.
[9] McNally RJ. Sleep paralysis, sexual abuse, and space alien abduction. Transcult Psychiatry 2005;42:113–22. http://dx.doi.org/10.1177/1363461505050715.
[10] Cheyne JA. Sleep paralysis and the structure of waking-nightmare hallucinations. Dreaming 2003;13:163–79. http://dx.doi.org/10.1037/1053-0797.13.3.163.

* The most important references are denoted by an asterisk.

[11] Sharpless BA, Barber JP. Lifetime prevalence rates of sleep paralysis: a systematic review. Sleep Med Rev 2011;15:311–5. http://dx.doi.org/10.1016/j.smrv.2011.01.007.
[12] Fukuda K. One explanatory basis for the discrepancy of reported prevalences of sleep paralysis among healthy respondents. Percept Mot Ski 1993;77:803–7.
[13] Pedrazzoli M, Pontes JC, Peirano P, Tufik S. HLA-DQB1 genotyping in a family with narcolepsy-cataplexy. Brain Res 2007;1165:1–4. http://dx.doi.org/10.1016/j.brainres.2007.05.075.
[14] Mahlios J, De la Herrán-Arita AK, Mignot E. The autoimmune basis of narcolepsy. Curr Opin Neurobiol 2013;23:767–73. http://dx.doi.org/10.1016/j.conb.2013.04.013.
[15] Ohaeri J, Awadalla A, Makanjuola V, Ohaeri B. Features of isolated sleep paralysis among Nigerians. East Afr Med J 2004;81. http://dx.doi.org/10.4314/eamj.v81i10.9233.
[16] O'Hanlon J, Murphy M, Di Blasi Z. Experiences of sleep paralysis in a sample of Irish university students. Ir J Med Sci 2011;180:917–9. http://dx.doi.org/10.1007/s11845-011-0732-2.
*[17] Denis D, French CC, Rowe R, Zavos HMS, Nolan PM, Parsons MJ, et al. A twin and molecular genetics study of sleep paralysis and associated factors. J Sleep Res 2015;24:438–46. http://dx.doi.org/10.1111/jsr.12282.
[18] Hsieh S-W, Lai C-L, Liu C-K, Lan S-H, Hsu C-Y. Isolated sleep paralysis linked to impaired nocturnal sleep quality and health-related quality of life in Chinese–Taiwanese patients with obstructive sleep apnea. Qual Life Res 2010;19:1265–72. http://dx.doi.org/10.1007/s11136-010-9695-4.
[19] Ramsawh HJ, Raffa SD, White KS, Barlow DH. Risk factors for isolated sleep paralysis in an African American sample: a preliminary study. Behav Ther 2008;39:386–97. http://dx.doi.org/10.1016/j.beth.2007.11.002.
*[20] Ohayon MM, Zulley J, Guilleminault C, Smirne S. Prevalence and pathologic associations of sleep paralysis in the general population. Neurology 1999;52.1194–200.
[21] Young E, Xiong S, Finn L, Young T. Unique sleep disorders profile of a population-based sample of 747 Hmong immigrants in Wisconsin. Soc Sci Med 2013;79:57–65. http://dx.doi.org/10.1016/j.socscimed.2012.06.009.
[22] Spanos NP, McNulty SA, DuBreuil SC, Pires M, Faith Burgess M. The frequency and correlates of sleep paralysis in a university sample. J Res Pers 1995;29:285–305.
[23] Otto MW, Simon NM, Powers M, Hinton D, Zalta AK, Pollack MH. Rates of isolated sleep paralysis in outpatients with anxiety disorders. J Anxiety Disord 2006;20:687–93. http://dx.doi.org/10.1016/j.janxdis.2005.07.002.
*[24] Munezawa T, Kaneita Y, Osaki Y, Kanda H, Ohtsu T, Suzuki H, et al. Nightmare and sleep paralysis among Japanese adolescents: a nationwide representative survey. Sleep Med 2011;12:56–64. http://dx.doi.org/10.1016/j.sleep.2010.04.015.
[25] Wing YK, Lee ST, Chen CN. Sleep paralysis in Chinese: ghost oppression phenomenon in Hong Kong. Sleep 1994;17:609–13.
[26] Ohaeri JU, Odejide AO, Ikuesan BA, Adeyemi JD. The pattern of isolated sleep paralysis among Nigerian medical students. J Natl Med Assoc 1989;81:805–8.
[27] Ohaeri JU, Odejide AO, Ikuesan BA, Adeyemi JD. The pattern of isolated sleep paralysis among Nigerian nursing students. J Natl Med Assoc 1992;84:67–70.
[28] Ma S, Wu T, Pi G. Sleep paralysis in Chinese adolescents: a representative survey. Sleep Biol Rhythms 2014;12:46–52. http://dx.doi.org/10.1111/sbr.12045.
*[29] Sharpless BA, McCarthy KS, Chambless DL, Milrod BL. Isolated sleep paralysis and fearful isolated sleep paralysis in outpatients with panic attacks. J Clin Psychol 2010;66:1292–307. http://dx.doi.org/10.1002/jclp.
[30] Paradis CM, Friedman S, Hatch M. Isolated sleep paralysis in African Americans with panic disorder. Cult Divers Ment Health 1997;3:69–76. http://dx.doi.org/10.1177/1363461505050720.
[31] Paradis C, Friedman S, Hinton DE, McNally RJ, Solomon LZ, Lyons KA. The assessment of the phenomenology of sleep paralysis: the unusual sleep experiences questionnaire (USEQ). CNS Neurosci Ther 2009;15:220–6. http://dx.doi.org/10.1111/j.1755-5949.2009.00098.x.
[32] Munezawa T, Kaneita Y, Yokoyama E, Suzuki H, Ohida T. Epidemiological study of nightmare and sleep paralysis among Japanese adolescents. Sleep Biol Rhythms 2009;7:201–10. http://dx.doi.org/10.1111/j.1479-8425.2009.00404.x.
[33] Stephan FK. The "other" circadian system: food as a Zeitgeber. J Biol Rhythms 2002;17:284–92. http://dx.doi.org/10.1177/074873040201700402.
[34] Drake C, Roehrs T, Shambroom J, Roth T. Caffeine effects on sleep taken 0, 3, or 6 hours before going to bed. J Clin Sleep Med 2013;9:1195–200. http://dx.doi.org/10.5664/jcsm.3170.
[35] Abrams MP, Mulligan AD, Carleton RN, Asmundson GJG. Prevalence and correlates of sleep paralysis in adults reporting childhood sexual abuse. J Anxiety Disord 2008;22:1535–41. http://dx.doi.org/10.1016/j.janxdis.2008.03.007.
*[36] McNally RJ, Clancy SA. Sleep paralysis in adults reporting repressed, recovered, or continuous memories of childhood sexual abuse. J Anxiety Disord 2005;19:595–602. http://dx.doi.org/10.1016/j.janxdis.2004.05.003.
[37] Mellman TA, Aigbogun N, Graves RE, Lawson WB, Alim TN. Sleep paralysis and trauma, psychiatric symptoms and disorders in an adult African American population attending primary medical care. Depress Anxiety 2008;25:435–40. http://dx.doi.org/10.1002/da.20311.
[38] Bell CC, Shakoor B, Thompson B, Dew D, Hughley E, Mays R, et al. Prevalence of isolated sleep paralysis in black subjects. J Natl Med Assoc 1984;76:501–8.
[39] Denis D, Poerio GL. Terror and bliss? Commonalities and distinctions between sleep paralysis, lucid dreaming, and their associations with waking life experiences. J Sleep Res 2016. http://dx.doi.org/10.1111/jsr.12441.
[40] Bell CC, Dixie-Bell DD, Thompson B. Further studies on the prevalence of isolated sleep paralysis in black subjects. J Natl Med Assoc 1986;78:649–59.
[41] Dahlitz M, Parkes J. Sleep paralysis. Lancet 1993;341:406–7.
*[42] Takeuchi T, Fukuda K, Sasaki Y, Inugami M, Murphy TI. Factors related to the occurrence of isolated sleep paralysis elicited during a multi-phasic sleep-wake schedule. Sleep 2002;25:89–96.
[43] Mume CO, Ikem IC. Sleep paralysis and psychopathology. South Afr J Psychol 2009;15:97–100.
[44] Morandin M, Bruck D. Understanding automatic behavior in narcolepsy: new insights using a phenomenological approach. Open Sleep J 2013;6:1–7.
[45] Schredl M, Erlacher D. Lucid dreaming frequency and personality. Pers Individ Dif 2004;37:1463–73. http://dx.doi.org/10.1016/j.paid.2004.02.003.
[46] Dodet P, Chavez M, Leu-Semenescu S, Golmard J-L, Arnulf I. Lucid dreaming in narcolepsy. Sleep 2015;38:487–97. http://dx.doi.org/10.5665/sleep.4516.
[47] Sharpless BA. Exploding head syndrome is common in college students. J Sleep Res 2015. http://dx.doi.org/10.1111/jsr.12292.
[48] Nelson KR, Mattingly M, Lee SA, Schmitt FA. Does the arousal system contribute to near death experience? Neurology 2006;66:1003–9. http://dx.doi.org/10.1212/01.wnl.0000204296.15607.37.
[49] Nelson KR, Mattingly M, Schmitt FA. Out-of-body experience and arousal. Neurology 2007;68:794–5. http://dx.doi.org/10.1212/01.wnl.0000256773.87498.2f.
*[50] Takeuchi T, Miyasita A, Sasaki Y, Inugami M, Fukuda K. Isolated sleep paralysis elicited by sleep interruption. Sleep 1992;15:217–25.
[51] Kotorii T, Uchimura N, Hashizume Y, Shirakawa S, Satomura T, Tanaka J, et al. Questionnaire relating to sleep paralysis. Psychiatry Clin Neurosci 2001;55:265–6. http://dx.doi.org/10.1046/j.1440-1819.2001.00853.x.
[52] Buysse DJ, Reynolds C, Monk T, Berman S, Kupfer D. The Pittsburgh Sleep Quality Index (PSQI): a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193–213.
[53] Cheyne JA. Situational factors affecting sleep paralysis and associated hallucinations: position and timing effects. J Sleep Res 2002;11:169–77.
[54] Fukuda K, Ogilvie RD, Chilcott L, Vendittelli A, Takeuchi T. The prevalence of sleep paralysis among Canadian and Japanese college students. Dreaming 1998;8:59–66.
[55] Girard TA, Cheyne JA. Timing of spontaneous sleep-paralysis episodes. J Sleep Res 2006;15:222–9. http://dx.doi.org/10.1111/j.1365-2869.2006.00512.x.
*[56] Szklo-Coxe M, Young T, Finn L, Mignot E. Depression: relationships to sleep paralysis and other sleep disturbances in a community sample. J Sleep Res 2007;16:297–312. http://dx.doi.org/10.1111/j.1365-2869.2007.00600.x.
[57] Vernet C, Redolfi S, Attali V, Konofal E, Brion A, Frija-Orvoen E, et al. Residual sleepiness in obstructive sleep apnoea: phenotype and related symptoms. Eur Respir J 2011;38:98–105. http://dx.doi.org/10.1183/09031936.00040410.
[58] Andlauer O, Moore HE, Hong S-C, Dauvilliers Y, Kanbayashi T, Nishino S, et al. Predictors of hypocretin (Orexin) deficiency in narcolepsy without cataplexy. Sleep 2012;35:1247–55. http://dx.doi.org/10.5665/sleep.2080.
[59] Mignot E, Hayduk R, Black J, Grumet FC, Guilleminault C. HLA DQB1*0602 is associated with cataplexy in 509 narcoleptic patients. Sleep 1997;20:1012–20.
[60] Bassetti C, Aldrich MS. Idiopathic hypersomnia. A series of 42 patients. Brain 1997:120. http://dx.doi.org/10.1093/brain/120.8.1423. Pt 8:1423–35.
[61] Vernet C, Arnulf I. Idiopathic hypersomnia with and without long sleep time: a controlled series of 75 patients. Sleep 2009;32:753–9.
[62] American Sleep Disorders Association. International classification of sleep paralysis: diagnostic and coding manual (ICSD). 1990.
[63] Simard V, Nielsen TA. Sleep paralysis-associated sensed presence as a possible manifestation of social anxiety. Dreaming 2005;15:245–60. http://dx.doi.org/10.1037/1053-0797.15.4.245.
[64] Solomonova E, Nielsen T, Stenstrom P, Simard V, Frantova E, Donderi D. Sensed presence as a correlate of sleep paralysis distress, social anxiety and waking state social imagery. Conscious Cogn 2008;17:49–63. http://dx.doi.org/10.1016/j.concog.2007.04.007.
*[65] Hinton DE, Pich V, Chhean D, Pollack MH. The ghost pushes you down: sleep paralysis-type panic attacks in a Khmer refugee population. Transcult Psychiatry 2005;42:46–77. http://dx.doi.org/10.1177/1363461505050710.
*[66] Hinton DE, Pich V, Chhean D, Pollack MH, McNally RJ. Sleep paralysis among Cambodian refugees: association with PTSD diagnosis and severity. Depress Anxiety 2005;22:47–51. http://dx.doi.org/10.1002/da.20084.
[67] Yeung A, Xu Y, Chang DF. Prevalence and illness beliefs of sleep paralysis among Chinese psychiatric patients in China and the United States. Transcult Psychiatry 2005;42:135–45. http://dx.doi.org/10.1177/1363461505050725.
[68] Cheyne JA, Girard TA. Paranoid delusions and threatening hallucinations: a prospective study of sleep paralysis experiences. Conscious Cogn 2007;16:959–74. http://dx.doi.org/10.1016/j.concog.2007.01.002.
[69] Cheyne JA, Girard TA. The body unbound: vestibular-motor hallucinations and out-of-body experiences. Cortex 2009;45:201–15. http://dx.doi.org/10.1016/j.cortex.2007.05.002.
[70] Gregory AM, Buysse DJ, Willis TA, Rijsdijk FV, Maughan B, Rowe R, et al. Associations between sleep quality and anxiety and depression symptoms in a sample of young adult twins and siblings. J Psychosom Res 2011;71:250–5. http://dx.doi.org/10.1016/j.jpsychores.2011.03.011.
[71] Arnulf I, Lin L, Gadoth N, File J, Lecendreux M, Franco P, et al. Kleine-Levin syndrome: a systematic study of 108 patients. Ann Neurol 2008;63:482–93. http://dx.doi.org/10.1002/ana.21333.
[72] Germain A, Hall M, Krakow B, Katherine Shear M, Buysse DJ. A brief sleep scale for posttraumatic stress disorder: Pittsburgh sleep quality index

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005

addendum for PTSD. J Anxiety Disord 2005;19:233–44. http://dx.doi.org/10.1016/j.janxdis.2004.02.001.
[73] Gangdev P, Dua V, Desjardins N. Isolated sleep paralysis and hypnic hallucinations in schizophrenia. Indian J Psychiatry 2015;57:383–5. http://dx.doi.org/10.4103/0019-5545.171834.
[74] Sharpless BA. A clinician's guide to isolated sleep paralysis. Neuropsychiatr Dis Treat 2016;12:1761–7. http://dx.doi.org/10.2147/NDT.S100307.
[75] Morgenthaler TI, Kapur VK, Brown T, Swick TJ, Alessi C, Aurora RN, et al. Practice parameters for the treatment of narcolepsy and other hypersomnias of central origin. Sleep 2007;30:1705–11.
[76] The U.S. Xyrem Multicenter Study Group. A randomized, double blind, placebo-controlled multicenter trial comparing the effects of three doses of orally administered sodium oxybate with placebo for the treatment of narcolepsy. Sleep 2002;25:42–9.
[77] Ahmed M, Bassetti C, Becker P, Michael B, Black J, Bogan R, et al. Further evidence supporting the use of sodium oxybate for the treatment of cataplexy: a double-blind, placebo-controlled study in 228 patients. Sleep Med 2005;6:415–21. http://dx.doi.org/10.1016/j.sleep.2005.03.010.
[78] Overeem S, Mignot E, van Dijk JG, Lammers GJ. Narcolepsy: clinical features, new pathophysiologic insights, and future perspectives. J Clin Neurophysiol 2001;18:78–105. http://dx.doi.org/10.1097/00004691-200103000-00002.
[79] Hofmann SG, Smits JAJ. Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. J Clin Psychiatry 2008;69:621–32. http://dx.doi.org/10.4088/JCP.v69n0415.
[80] Bryant RA, Harvey AG, Dang ST, Sackville T, Basten C. Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. J Consult Clin Psychol 1998;66:862–6. http://dx.doi.org/10.1037/0022-006X.66.5.862.
[81] Trauer JM, Qian MY, Doyle JS, Rajaratnam SMW, Cunnington D. Cognitive behavioral therapy for chronic insomnia: a systematic review and meta-analysis. Ann Intern Med 2015;163:191–204. http://dx.doi.org/10.7326/M14-2841.
[82] Espie CA, Kyle SD, Williams C, Ong JC, Douglas NJ, Hames P, et al. A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. Sleep 2012;35:769–81. http://dx.doi.org/10.5665/sleep.1872.
[83] Attarian H. Treatment options for parasomnias. Neurol Clin 2010;28:1089–106.
[84] Perlis ML, Jungquist C, Smith MT, Posner D. Cognitive behavioural treatment of insomnia: a session-by-session guide. New York, NY: Springer Science + Business Media, LLC; 2005.
[85] Brooks PL, Peever JH. Identification of the transmitter and receptor mechanisms responsible for REM sleep paralysis. J Neurosci 2012;32:9785–95. http://dx.doi.org/10.1523/JNEUROSCI.0482-12.2012.
[86] Terzaghi M, Ratti PL, Manni F, Manni R. Sleep paralysis in narcolepsy: more than just a motor dissociative phenomenon? Neurol Sci 2012;33:169–72. http://dx.doi.org/10.1007/s10072-011-0644-y.
[87] Cheyne JA. Recurrent isolated sleep paralysis. In: Thorpy M, Plazzi G, editors. Parasomnias other sleep-related Mov. Disord. Cambridge University Press; 2010. p. 142–52.
[88] Sharpless BA, Denis D. Recurrent isolated sleep paralysis. In: Stein J, editor. Ref. Modul. Neurosci. Biobehav. Psychol. Elsevier; 2017. p. 1–5. http://dx.doi.org/10.1016/B978-0-12-809324-5.01246-3.
[89] LaBerge S. Signal-verified lucid dreaming proves that REM sleep can support reflective consciousness: commentary on "The Neurobiology of Consciousness: Lucid Dreaming Wakes Up" by J. Allan Hobson. Int J Dream Res 2010;3:26–7.
[90] Mahowald MW, Schenck CH. Insights from studying human sleep disorders. Nature 2005;437:1279–85. http://dx.doi.org/10.1038/nature04287.
[91] Hobson JA. REM sleep and dreaming: towards a theory of protoconsciousness. Nat Rev Neurosci 2009;10:803–13. http://dx.doi.org/10.1038/nrn2716.
[92] Hobson JA. The neurobiology of consciousness: lucid dreaming wakes up. Int J Dream Res 2009;2:41–4. http://dx.doi.org/10.11588/ijodr.2009.2.403.

Please cite this article in press as: Denis D, et al., A systematic review of variables associated with sleep paralysis, Sleep Medicine Reviews (2017), http://dx.doi.org/10.1016/j.smrv.2017.05.005