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Original

Sleep-Related Eating Disorder among Japanese Psychiatric Outpatients Receiving Ultra-Short-Acting Benzodiazepine Receptor Agonists: A Cross-Sectional Pilot Study

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Background: Sleep-related eating disorder (SRED) is a parasomnia characterized by involuntary nocturnal eating with amnesia, which can result in serious injuries, weight gain, and metabolic complications. Reports implicate the use of ultra-short-acting benzodiazepine receptor agonists (USBZRAs)—especially zolpidem—in SRED, but the magnitude of this risk in psychiatric patients remains unclear.

Methods: We performed a cross-sectional observational survey at two Japanese hospitals of 157 psychiatric outpatients receiving one of four USBZRAs (triazolam, zopiclone, zolpidem, or eszopiclone) for ≥7 days. High-dose USBZRA therapy was defined as the maximum recommended dose per package insert. SRED was assessed using an International Classification of Sleep Disorders-based checklist. A Firth bias-reduced logistic regression model with four prespecified clinically relevant variables was employed due to the limited number of events.

Results: Fourteen patients met SRED criteria (8.9%; 95% CI, 4.9–14.4). Zolpidem use (adjusted odds ratio [aOR] 5.98; 95% CI, 1.57–33.58) and high-dose USBZRA therapy (aOR 4.87; 95% CI, 1.56–17.51) were independently associated with SRED. Age (aOR 0.98; 95% CI, 0.94–1.02) and female sex (aOR 0.75; 95% CI, 0.22–2.87) were not significant.

Conclusions: The observed prevalence aligns with earlier reports, confirming that nearly one in eleven psychiatric outpatients receiving USBZRAs experiences SRED. Our study extends prior work by showing that SRED risk is highest at the maximum recommended dose, and especially with zolpidem. The wide confidence intervals reflect the small number of events and should be interpreted as hypothesis generating rather than definitive. These findings support limiting USBZRA dosage, favoring lower-risk hypnotics, and actively screening for nocturnal eating. This pilot study warrants validation in larger cohorts.

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Introduction

Sleep-related eating disorder (SRED) is classified in the International Classification of Sleep Disorders, Third Edition (ICSD-3) as a non-rapid eye movement parasomnia characterized by recurrent, involuntary episodes of eating or drinking during partial arousals from sleep, with sub-

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sequent partial or complete amnesia for the event[1]. While clinically distinct from night eating syndrome—in which patients consciously eat during the night—SRED confers comparable metabolic and safety risks, including weight gain, injury, and metabolic complications, while often remaining under-recognized by clinicians[2]. SRED episodes are a variant of somnambulism, involving complex behaviors (preparing and consuming food) without conscious awareness[2]. Epidemiological studies and case series implicate a multifactorial etiology in which coexisting arousal disorders (especially sleepwalking), mood or anxiety disorders, female sex, younger adult age, circadian disruption (e.g., shift work), and certain neurologic conditions (such as narcolepsy) may synergistically promote SRED[2–4]. For example, idiopathic SRED (occurring without medication triggers) disproportionately affects women in early adulthood[3], and approximately one-third of patients with narcolepsy type 1 report nocturnal eating events over their lifetime[5], while about 8% are currently affected[6]. Among putative precipitants of SRED, sedative-hypnotic medications—particularly ultra-short-acting benzodiazepine receptor agonists (USBZRAs)—are notable. In Japan, the benzodiazepine triazolam was approved in 1982, followed by the non-benzodiazepine "Z-drugs" zopiclone (1989), zolpidem (2000), and eszopiclone (2012). These agents are favored for their rapid sleep-onset efficacy and minimal residual sedation. However, numerous reports have linked USBZRA use to nighttime eating episodes. Triazolam was first implicated in 1992, through a case of recurrent nocturnal binge-eating with anterograde amnesia[4]. Subsequently, most published cases of hypnotic-induced SRED have involved zolpidem, with isolated reports attributed to zopiclone or zaleplon[7]. A literature review compiled 40 cases of zolpidem-associated SRED, 95% of which occurred at doses ≥10 mg nightly[8]. Pharmacovigilance analyses have confirmed that newer Z-drugs such as eszopiclone can precipitate similar nocturnal behaviors[8]. Meanwhile, other psychotropic medications have been implicated in triggering SRED, including certain antidepressants (particularly serotonin–norepinephrine reuptake inhibitors) and atypical antipsychotics[9,10]. Notably, a recent global disproportionality analysis identified 35 drugs with potential associations with SRED, prominently zolpidem (accounting for up to 36% of medication-associated cases) but also agents such as immediate-release sodium oxibate (up to 27%) and the antipsychotic quetiapine (up to 14%)[9]. Despite growing recognition of medication-triggered SRED, robust epidemiological data are limited. In Japanese psychiatric outpatients, Takaesu et al.[11] reported an SRED prevalence of 8.4%, with high cumulative hypnotic dosage and antipsychotic polypharmacy as significant risk factors. In contrast, an internet-based survey of young adults[12] found a prevalence of only 2.2%, identifying "use of any hypnotic" as the sole significant risk factor, without distinguishing drug classes. Whether the strong USBZRA–SRED association documented in case reports and pharmacovigilance databases extends to routine clinical practice remains unclear, particularly in populations with psychiatric comorbidities. In this context, we conducted a cross-sectional pilot study to (i) determine the prevalence of SRED among adult psychiatric outpatients prescribed USBZRAs in Japan and (ii) identify medication-related factors associated with SRED, focusing on potential differences among the four most prescribed USBZRAs.

Materials and Methods

Study Design and Participants

We performed a retrospective cross-sectional study of adults (≥18 years old) who attended the psychiatric outpatient clinics of Nippon Medical School Chiba Hokusoh Hospital or Nippon Medical School Hospital between April 1 and June 30, 2022, and who were treated with a USBZRA (triazolam, zopiclone, zolpidem, or eszopiclone). During routine visits, a trained investigator screened participants for SRED using a structured questionnaire (**Table 1**) based on the ICSD-3 criteria for non-rapid eye movement parasomnias.

Ethics

The study employed an opt-out consent procedure in accordance with the Ethical Guidelines for Life Science and Medical Research Involving Human Subjects. Details were posted on the websites of both hospitals. The protocol ("Study on Abnormal Behaviors as Parasomnias" [Principal Investigator Haruki Saito]) was approved by the Nippon Medical School Central Ethics Committee (approval number: M2023-105, June 8, 2023) and the Chiba Hokusoh Hospital Ethics Committee (approval number: R2023-056, June 20, 2023).

Exclusion Criteria

Participants were excluded if they used two or more USBZRAs concurrently; had a history of an eating disorder, night eating syndrome, or another abnormal eating behavior; had an organic brain disorder or intellectual disability; or were otherwise deemed unsuitable by the in-

Table 1 Questionnaire items regarding sleep-related parasomnias

No.	Question item
1	Found oneself dressed in different clothes upon awakening at night without any recollection of changing clothes.
2	Discovered unexplained dirt on one's feet upon awakening.
3	Noticed injuries or abrasions that were definitely not present before going to bed.
4	Awoke to find one's sleeping location had significantly shifted from where one had fallen asleep.
5	Found evidence of having eaten something during sleep but having no memory of having done so.

To align with ICSD-3 diagnostic criteria[1], also confirm:
- Recurrence of episodes (frequency)
- Incomplete arousals (partial consciousness)
- Amnesia for the event
- Exclusion of other medical, psychiatric, or substance-related causes
- Associated daytime dysfunction or injury risk

vestigators. Patients receiving a USBZRA together with another (short-, intermediate-, or long-acting) benzodiazepine hypnotic or a non-BZRA hypnotic (e.g., ramelteon or a dual orexin receptor antagonist) were retained in the sample. Of 246 eligible patients, 183 were interviewed. Excluding 11 patients with incomplete records and 15 who met the exclusion criteria, 157 participants were included in the final analysis. Among the excluded cases, six patients had concomitant use of two or more ultra-short-acting benzodiazepine receptor agonists (USBZRAs); none screened positive for parasomnias, including SRED.

Statistical Analysis

Categorical variables were compared using Pearson's chi-squared test or, when an expected cell count was <5, Fisher's exact test. Four group nominal variables were examined with an overall Fisher's exact test, followed by Holm-corrected pairwise Fisher's exact tests when significant. Continuous variables were compared with independent-samples t-tests. Given the limited number of SRED events (n=14), we employed Firth bias-reduced logistic regression with four prespecified clinically relevant variables: zolpidem use, high-dose USBZRA therapy, age, and sex. Model discrimination was assessed using the C-statistic (area under the receiver operating characteristic curve). Statistical analyses were performed using JMP v18 (SAS Institute Inc., Cary, NC, USA) and EZR v2.9 (Saitama Medical University Medical Center, Saitama, Japan)[13].

Results

Participant Characteristics

The characteristics of the 157 participants (105 women, 52 men; mean age 60.80 ± 14.97 years) are summarized in **Table 2**, along with primary ICD-10 diagnoses[14]. Primary ICD-10 diagnoses[14] were mood (F3) disorders in 101 patients (64.33%), anxiety/stress-related (F4) disorders in 29 (18.50%), schizophrenia spectrum (F2) disorders in 16 (10.19%), non-organic insomnia (F51.0) in 10 (6.37%), and pervasive developmental disorders (F84) in 1 (0.64%). Zolpidem was prescribed to 75 patients (47.77%), eszopiclone to 58 (36.94%), triazolam to 20 (12.74%), and zopiclone to 4 (2.55%). Forty-eight participants (30.57%) received the maximum recommended dose, and 134 (85.35%) had been treated for ≥1 year. Concomitant sleeping medications were used by 95 patients (60.51%); these were a non-triazolam benzodiazepine hypnotic in 54 (34.39%) patients and a non-BZRA hypnotic in 41 (26.11%; lemborexant in 24, suvorexant in 10, ramelteon in 6, and ramelteon + suvorexant in 1).

Prevalence of Parasomnias

Sixteen participants (10.19%) screened positive for a parasomnia: 14 (8.92%) for SRED (13 with SRED alone, 1 with SRED plus sleepwalking), and 2 (1.27%) for sleepwalking alone. Among the SRED-positive patients, episode frequency was high (≥10 per month) in 5 (35.71%), medium (4–9 per month) in 7 (50.0%), and low (1–3 per month) in 2 (14.3%).

Group Comparison: SRED Positive vs Negative

Table 3 compares patients with SRED (n = 14) and without (n = 143). No significant differences emerged between the groups in terms of age (57.57 ± 15.29 vs 61.11± 11.01 years; t = −0.84, P = 0.40), sex (male: 28.57% vs 33.57%; Fisher's exact P = 1.00), ICD-10 diagnostic category[14] (P = 0.59), concomitant non-triazolam benzodiazepine use (14.29% vs 35.66%; P = 0.12), or concomitant non-BZRA hypnotic use (35.71% vs 25.17%; P = 0.52).

Table 2 Patient characteristics and treatment patterns (n = 157)

Characteristic	Value
Sex	
Female	105 (66.88%)
Male	52 (33.12%)
Age, mean ± SD (years)	60.80 ± 14.97
Primary diagnosis (ICD-10)	
F3: Mood [affective] disorders	101 (64.33%)
F4: Anxiety and stress-related disorders	29 (18.50%)
F2: Schizophrenia spectrum	16 (10.19%)
F51.0: Non-organic insomnia	10 (6.37%)
F84: Pervasive developmental disorders	1 (0.64%)
Hypnotic agent	
zolpidem	75 (47.77%)
eszopiclone	58 (36.94%)
triazolam	20 (12.74%)
zopiclone	4 (2.55%)
Maximum recommended dose used	48 (30.57%)
Duration of treatment ≥ 1 year	134 (85.35%)
Concomitant sleeping medications	
Non-triazolam benzodiazepine hypnotics	54 (34.39%)
Single agent	52
Two agents	2
Hypnotics not acting on benzodiazepine receptors	41 (26.11%)
lemborexant	24
suvorexant	10
ramelteon	6
ramelteon + suvorexant	1
Parasomnia screening positive	16 (10.19%)
SRED only	13 (8.28%)
Sleepwalking only	2 (1.27%)
Both SRED & sleepwalking	1 (0.63%)
SRED episode frequency (n = 14)	
High (≥ 10/months)	5 (35.71%)
Medium (4–9/months)	7 (50.00%)
Low (1–3/months)	2 (14.29%)

SRED: sleep-related eating disorder.

Conversely, the distribution of USBZRAs differed significantly between groups (overall Fisher's exact P = 0.04). Zolpidem use was markedly higher in the SRED group (85.71% vs 44.06%; P < 0.01; odds ratio [OR], 7.61; 95% CI, 1.64−35.57), as was high-dose USBZRA therapy (71.43% vs 26.57%; P < 0.01; OR, 6.81; 95% CI, 1.83−31.54).

Multivariable Analysis

The Firth bias-reduced logistic regression model showed good discrimination (AUC, 0.82; 95% CI, 0.72−0.94). After adjustment, zolpidem use (aOR, 5.98; 95% CI, 1.57−33.58; P = 0.007) and high-dose USBZRA therapy (aOR, 4.87; 95% CI, 1.56−17.51; P = 0.006) were associated with SRED. Age (aOR, 0.98; 95% CI, 0.94−1.02; P = 0.34) and female sex (aOR, 0.75; 95% CI, 0.22−2.87; P = 0.67) were not significantly associated (**Table 4**).

Discussion

In the context of ongoing regulatory attention to Z-drugs and accumulating evidence linking zolpidem to SRED, our findings add real-world data from Japanese psychiatric outpatients and directly compare SRED risk across USBZRAs—information that is scarce in the current literature and clinically actionable for risk-benefit discussions.

The present multicenter survey provides an up-to-date cross-sectional portrait of SRED among Japanese outpatients prescribed USBZRAs. By limiting the study to triazolam, zopiclone, zolpidem, and eszopiclone, the four

Table 3 Clinical characteristics of patients with and without SRED

Variable	SRED (n = 14)	Non-SRED (n = 143)	Test (p)	OR or mean diff (95% CI)
Age, years	57.57 ± 15.29	61.11 ± 11.01	t = –0.84, P = 0.40	–3.5 (–11.5–4.8)
Sex, n (%)			Fisher (1)	1.26 (0.34–5.80)
Female	10 (71.43%)	95 (66.43%)		
Male	4 (28.57%)	48 (40.56%)		
ICD-10 diagnosis, n			Fisher (0.59)	—
F2	1	15		
F3	12	89		
F4	1	28		
F51.0	0	10		
F84	0	1		
Hypnotic agent, n			Fisher (0.039)	—
TRZ	0	20		
ZPC	0	4		
ZPD	12	63		
ESZ	2	56		
Zolpidem use, n (%)	12 (85.71%)	63 (44.06%)	Fisher (0.0038)	7.61 (1.64–35.57)
High-dose grade (H), n (%)	10 (71.43%)	38 (26.57%)	Fisher (0.0033)	6.81 (1.83–31.54)
Duration of treatment ≥ 1 year, n (%)	12 (85.71%)	122 (85.31%)	Fisher (1)	1.03 (0.20–10.16)
Concomitant use of non-triazolam BZD hypnotics, n (%)	3 (21.43%)	51 (35.66%)	Fisher (0.383)	0.49 (0.09–1.97)
Concomitant use of non-BZD-receptor agonists (e.g. lemborexant), n (%)	5 (35.71%)	36 (25.17%)	Fisher (0.52)	1.65 (0.41–15.90)

For the multi-category comparison of ICD-10 diagnoses, Fisher's exact test showed no significant differences, and the presence of zero counts in some cells precluded valid post hoc analyses. High-dose grade (H): maximum recommended dose. Hypnotic agent: prescribed ultra-short-acting benzodiazepine receptor agonists (TRZ: triazolam, ZPC: zopiclone, ZPD: zolpidem, ESZ: eszopiclone). Fisher's exact test showed a statistically significant overall difference ($p<0.05$), but post hoc pairwise comparisons were not performed because some cells contained zero counts. Zolpidem use: analyzed separately because zolpidem (ZPD) demonstrated a markedly higher prevalence in the SRED group.

BZD: benzodiazepine, CI: confidence interval, ESZ: eszopiclone, Fisher: Fisher's exact test, OR: odds ratio, SRED: sleep-related eating disorder, TRZ: triazolam, ZPC: zopiclone, ZPD: zolpidem.

Table 4 Multivariate Firth's logistic regression of factors associated with SRED onset

Variable	df	Adjusted OR	95% CI	p value
Age (per year)	1	0.98	0.94–1.02	0.34
Female sex	1	0.75	0.22–2.87	0.67
High-dose administration	1	4.87	1.56–17.51	0.006
Zolpidem use	1	5.98	1.57–33.58	0.007
AUC=0.82	1		0.72–0.94	

Model: Firth bias-reduced logistic regression. C-statistic = 0.82 (95% CI, 0.72–0.94). C-statistic and 95% confidence intervals were estimated with Firth's penalized maximum-likelihood method because the number of SRED events was limited (n = 14).

df: degrees of freedom, OR: odds ratio, CI: confidence interval.

hypnotics that dominate current prescription trends quantified contemporary SRED prevalence and identified two modifiable medication-related risks: zolpidem exposure and high-dose USBZRA therapy. These findings reinforce pharmacovigilance signals first detected in the World Health Organization global safety database, where zolpidem accounted for more than one-third of drug-attributed SRED cases and showed the highest reported OR of any hypnotic[9]. Likewise, a recent systematic review highlighted that Z-drugs, especially zolpidem, are the most consistent precipitants of pharmacologic SRED[10]. Although none of these four USBZRAs are formally approved in Japan for insomnia accompanying psychiatric disorders, in routine practice they are nevertheless pre-

scribed off label to a large proportion of such patients. Consistent with this real-world use, our data showed no significant difference in SRED incidence between patients carrying a primary diagnosis of non-organic insomnia (ICD-10 F51.0) and those with major psychiatric disorders (F2–F4); this suggests that the underlying diagnosis alone does not materially modify hypnotic-related risk. After multivariable adjustment, zolpidem was associated with approximately eightfold higher odds of SRED than the other ultra-short-acting benzodiazepine receptor agonists (USBZRAs), and risk increased further with high-dose therapy (at or above the labeled maximum dose)[5,8]. This pattern mirrors a clinical series in which 90–95% of zolpidem-linked SRED episodes occurred at ≥10 mg nightly[8]. Case-based literature has also documented zolpidem-induced complex sleep behaviors, including sleepwalking, sleep-related eating disorder, and sleep-driving[15]. These observations help explain why the U.S. Food and Drug Administration added boxed warnings about complex sleep behaviors to Z-drug labeling[16]. Our epidemiologic data confirm that this risk persists in routine practice and should inform shared decision-making whenever zolpidem is considered for insomnia.

Equally notable were the variables that were not associated with SRED: age, sex, benzodiazepine polypharmacy, and concomitant non-BZRA hypnotic use showed no significant associations. A recent systematic review and a contemporary clinical review both describe heterogeneous patient profiles across cohorts and clinical contexts[8], while idiopathic SRED cohorts tend to skew toward young adult women and patients with narcolepsy[5,6]. Consistent with this, a polysomnography-focused review and a pharmacovigilance disproportionality analysis also found marked variability across age, sex, and comorbidities, with medication-triggered cases tending to be older than idiopathic cohorts; in Merino's analysis, the mean age of medication-triggered cases was close to 50 years[9,10,17,18]. Moreover, 85% of our participants had used USBZRAs for over a year, potentially homogenizing exposure patterns and diluting demographic effects. Clinicians should therefore remain alert for SRED regardless of age and sex whenever potent, short-acting hypnotics are prescribed.

USBZRA-induced SRED is best conceptualized as a dissociated state in which subcortical circuits governing complex motor and reward functions remain active while frontal executive networks are pharmacologically silenced[19,20]. Strong GABA_A-receptor agonism promotes rapid sleep onset but also impairs memory encoding; as plasma drug levels decline midway through the night, partial arousals may arise against a backdrop of diminished cortical control, leading to automatisms such as nocturnal eating[21]. Polysomnography typically shows a mixed-frequency electroencephalogram with features of both sleep and wakefulness, accompanied by heightened autonomic activity during SRED events[22]. Fragmented sleep due to comorbid conditions—such as restless legs syndrome, obstructive sleep apnea, or narcolepsy—further destabilizes sleep architecture and is common in SRED cohorts[5,23]. Case reports of severe injury, including burns and deep lacerations incurred during amnestic cooking episodes[23,24], underscore the public health significance of this parasomnia. Finally, neurochemical or genetic susceptibilities may determine which individuals develop SRED; recent data implicate hypocretin (orexin) signaling deficits, GABA_A-receptor polymorphisms, and limbic glucose-metabolic disturbances as potential contributors[10].

From a management standpoint, the priority in SRED is to eliminate precipitating medications. Abrupt discontinuation or gradual tapering of the offending USBZRA abolishes nocturnal eating in most reported cases[15,23,25]. Where cessation is impracticable, off-label pharmacotherapies—most notably topiramate—have yielded substantial reductions in episode frequency, presumably by dampening limbic drive and appetite[26,27]. Clinical practice guidelines from the American Academy of Sleep Medicine recommend evaluating patients for complex sleep behaviors before renewing any hypnotic prescription and favor using the lowest effective dose when such treatment is unavoidable[27]. Dual orexin receptor antagonists offer a mechanistically distinct approach to sleep promotion. While one example, suvorexant, showed a modest SRED signal in post-marketing surveillance[9], a recent narrative review suggests that the overall parasomnia risk with this class is lower than with Z-drugs, provided they are used as monotherapy and within recommended doses[28–31]. Prospective head-to-head trials will be needed to confirm these apparent safety differentials.

Study Limitations

Several limitations must be considered regarding our conclusions. First, with only 14 SRED events, our study had limited statistical power, resulting in wide confidence intervals and substantial uncertainty. The adjusted odds ratios for zolpidem (95% CI, 1.57–33.58) and high-dose therapy (95% CI, 1.56–17.51) suggest potentially large effects but with considerable imprecision; although

we used a parsimonious model based on clinical knowledge to reduce variables and applied Firth bias-reduced logistic regression to address small sample bias, these measures may still have been insufficient. Therefore, we regard this work as a pilot study requiring validation in larger cohorts. Second, the cross-sectional, retrospective design precludes causal inference and may be subject to both under-reporting (amnesia/embarrassment) and over-reporting (suggestibility/recall bias). The ICSD-3-based checklist, while having face validity, lacks formal validation against polysomnography or collateral reports. Third, non-hypnotic psychoactive drugs were not systematically recorded, precluding adjustment for potential SRED triggers such as certain antidepressants and antipsychotics[9,10]. Fourth, the small number of zopiclone users (n = 4) precluded meaningful analysis. Fifth, without a non-user control group, we cannot quantify the absolute risk attributable to USBZRAs. Finally, generalizability is limited to the psychiatric outpatient setting.

Conclusions

Zolpidem and high-dose USBZRA therapy were significantly associated with SRED in Japanese psychiatric outpatients. This pilot study suggests elevated SRED risk at maximum-recommended doses, particularly for zolpidem. The wide confidence intervals necessitate cautious interpretation and confirmation in larger, prospective cohorts. Clinicians should consider limiting USBZRA dosage, favoring alternatives when appropriate, and actively screening for nocturnal eating behaviors.

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Data Availability: Due to privacy/ethical restrictions, the raw data that support the findings of this study are not publicly available. Aggregated data may be obtained from the corresponding author upon reasonable request.

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