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Insomnia and daytime sleepiness identify distinct behavioural phenotypes of binge eating and mood in clinical obesity: the MOCC cohort (n = 2,016)

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Abstract

Background/Objectives.

Insomnia and excessive daytime sleepiness are common in adults with obesity but typically examined in isolation. We tested whether, jointly, they identify behavioural phenotypes with distinct eating and mood profiles.

Subjects/Methods.

Cross-sectional analysis of 2,016 adults from the Montpellier Obesity Care Cohort (MOCC) (76% women; mean BMI 37.1 kg/m²). Participants completed the Insomnia Severity Index (ISI), Epworth Sleepiness Scale (ESS), Binge Eating Scale (BES), Hospital Anxiety and Depression Scale (HADS), Perceived Stress Scale, and Intuitive Eating Scale-2 (IES-2). Four phenotypes were defined a priori using ISI ≥ 15 and ESS ≥ 11: good sleeper, isolated insomnia, isolated sleepiness, combined. Multivariable regressions were adjusted for age, sex and BMI.

Results.

Phenotype distribution was 53.6% good sleeper, 18.2% isolated insomnia, 13.8% isolated sleepiness, 14.4% combined. ISI and ESS were only moderately correlated (ρ = 0.30). Isolated insomnia was most strongly associated with anxiety (HAD-A ≥ 11: adjusted OR [aOR] 4.48, 95% CI 3.41–5.89) and depression (aOR 3.09, 2.20–4.33); isolated sleepiness was most strongly associated with severe binge eating (BES ≥ 27: aOR 2.55, 1.69–3.83) and lower intuitive eating (IES-2 β = − 0.24, − 0.33 to − 0.15). The combined phenotype carried the highest risk overall. BMI did not differ across phenotypes. Findings were robust to OSA exclusion and to alternative insomnia thresholds.

Conclusions.

Insomnia and daytime sleepiness identify distinct behavioural phenotypes in clinical obesity, independently of BMI. Joint screening at intake may help personalise behavioural obesity management.

1. INTRODUCTION

Obesity is a chronic, multifactorial disease affecting more than one billion adults worldwide and carrying a heavy burden of cardiometabolic and psychological morbidity.[1,2] Among the most consistent non-metabolic comorbidities in adults with obesity are sleep disturbances and psycho-behavioural disturbances of eating and mood: up to 50–80% of patients seeking specialist obesity care report clinically significant insomnia symptoms, excessive daytime sleepiness, or both,[3–5] and a similar proportion report disordered eating, anxiety or depression. These two symptom domains are strongly intercorrelated, and their accurate characterisation is increasingly considered a core component of personalised obesity care.

The two most commonly assessed self-reported dimensions of sleep symptoms in clinical practice — insomnia, indexed by the Insomnia Severity Index (ISI),[6] and excessive daytime sleepiness, indexed by the Epworth Sleepiness Scale (ESS)[7] — are typically reported in isolation in the obesity literature, or summarised as a single "poor sleep" index. The two instruments however capture partially distinct phenomena: ISI scores the subjective difficulty in initiating and maintaining sleep and the resulting daytime impairment, whereas ESS scores the propensity to doze in passive situations, a construct more tightly linked to objective sleep-disordered breathing and to shortened total sleep time.[8] In the general population, ISI and ESS typically correlate at r = 0.2–0.4,[9] reflecting their partial independence. Whether their joint assessment in adults with obesity adds clinically meaningful information beyond either scale alone — and whether the two symptoms mark functionally distinct behavioural phenotypes — remains unresolved.

Insomnia and daytime sleepiness have been independently associated with adverse eating and mood outcomes. Chronic insomnia is strongly and prospectively linked to anxiety and depression,[10][11] while shortened and fragmented sleep, partly reflected by daytime sleepiness, has been consistently associated with disordered eating and, in particular, with binge eating and emotional eating.[12][13] Yet these two sleep dimensions are usually examined separately, and when a single sleep measure is reported, it is often taken as a proxy for global "sleep health" without regard to the specific symptom pattern. Few studies have simultaneously characterized insomnia and sleepiness in large clinical-obesity cohorts and asked whether their combinations identify clinically meaningful phenotypes.

Such joint phenotyping is clinically relevant because insomnia and sleepiness may point to different therapeutic targets: insomnia to cognitive-behavioral therapy, relaxation, and screening for affective disorders; sleepiness to sleep duration, circadian stability, screening for obstructive sleep apnea (OSA), and potentially to behavioral eating interventions. If the two symptoms mapped onto a single unidimensional "sleep burden," their joint assessment would add little; if instead they marked biologically and clinically distinct phenotypes, joint screening would be justified in routine obesity care.

Using baseline data from 2,016 adults seeking specialist nutrition-obesity care in France, we tested the pre-specified hypothesis that a 2 × 2 phenotyping of participants on insomnia (ISI ≥ 15) and daytime sleepiness (ESS ≥ 11) would yield four behavioural phenotypes with distinguishable profiles of disordered eating and mood, and specifically that insomnia would be preferentially associated with anxio-depressive symptoms whereas daytime sleepiness would be preferentially associated with severe binge eating and with reductions on the 18-item French Intuitive Eating Scale-2 (IES-2).[14][15] By testing these two sleep dimensions jointly in a large clinical-obesity sample, this study aims to inform the personalisation of psychological and behavioural screening at intake of specialist obesity care.

2. METHODS

2.1. Study design and participants

We conducted a cross-sectional analysis of baseline data from the Montpellier Obesity Care Cohort (MOCC), an ongoing observational cohort of adults seeking specialist nutrition-obesity care at the Centre Spécialisé de l'Obésité (CSO) of Montpellier University Hospital and at affiliated centres in the Occitanie region of France. Data are collected via the Aviitam digital health platform — a business-to-business platform launched in 2014 to support self-administered validated questionnaires and multidisciplinary obesity care.[16] The structured questionnaire battery, aligned with international recommendations for core variables in obesity research,[17] is administered at the initiation of specialist care, and anthropometric data are subsequently confirmed by health-care providers during clinical consultations. Eligible participants for the present analysis were adults (≥ 18 years) who completed the Intuitive Eating Scale-2 (IES-2), the Insomnia Severity Index (ISI), and the Epworth Sleepiness Scale (ESS) between January 2015 and December 2022, and who consented to secondary use of their pseudonymized data for research. Data were anonymized before analysis. The study was approved by the institutional review board of the University Hospital of Montpellier (IRB-MTP_2023_02_202301328) and registered on ClinicalTrials.gov (NCT06125600). A previously published analysis used baseline data from a broader subset of MOCC to derive a two-cluster solution combining intuitive eating and emotional distress and to assess its association with weight loss under semaglutide (Avignon et al., Obesity 2025; reference 30); the present analysis addresses an entirely distinct scientific question, with different central variables (ISI and ESS), pre-specified phenotype definitions, and no overlap of analyses, outcomes, or conclusions with that earlier publication.

2.2. Sleep phenotypes

The ISI (Morin)[6] is a 7-item self-report measure of insomnia severity over the preceding 2 weeks, with total scores ranging from 0 to 28. Following established clinical guidance, scores ≥ 15 indicate moderate-to-severe insomnia.[6] The ESS[7] is an 8-item measure of propensity to doze in passive situations, with scores ranging from 0 to 24; scores ≥ 11 indicate clinically significant excessive daytime sleepiness.[7] Using these pre-specified thresholds, we defined four mutually exclusive sleep phenotypes: (i) good sleeper (ISI < 15 and ESS ≤ 10); (ii) isolated insomnia (ISI ≥ 15 and ESS ≤ 10); (iii) isolated sleepiness (ISI < 15 and ESS ≥ 11); (iv) combined phenotype (ISI ≥ 15 and ESS ≥ 11).

2.3. Outcomes

Primary outcomes were severe binge eating (Binge Eating Scale [BES] ≥ 27[18]), clinically significant anxiety (Hospital Anxiety and Depression Scale – Anxiety subscale [HAD-A] ≥ 11[19]), and clinically significant depression (HAD-D ≥ 11[19]). Secondary outcomes were the French 18-item IES-2 global score and its three subscales — Eating for Physical rather than Emotional reasons (EPR), Reliance on Hunger and Satiety Cues (RHSC), and Unconditional Permission to Eat (UPE)[14][15] — the BES total score,[18] the HADS total score,[19] and the Perceived Stress Scale (PSS).[20] All instruments were administered in their validated French versions.[15][21–23]

2.4. Covariates

Age, sex, body weight, and height were measured by health-care providers at the consultation contemporaneous with questionnaire completion. BMI was computed as weight (kg) divided by height (m) squared. Documented obstructive sleep apnea (OSA) was coded from the structured medical-history field of the Aviitam record by searching for terms including “SAOS”, “sleep apnea”, “CPAP”, and “PPC”; this captures previously diagnosed OSA but not incident or undiagnosed disease.

2.5. Statistical analysis

Descriptive statistics are reported as mean ± SD or median [IQR] for continuous variables and as n (%) for categorical variables. Differences between phenotypes were assessed with one-way ANOVA for continuous variables and chi-squared tests for categorical variables, with the good-sleeper group as reference throughout. The joint distribution of ISI and ESS scores was summarized by Pearson and Spearman correlations.

Adjusted associations between phenotype and each binary outcome (BES ≥ 27, HAD-A ≥ 11, HAD-D ≥ 11) were estimated with multivariable logistic regression, adjusted for age (continuous), sex (binary), and BMI (continuous). For continuous outcomes (IES-2 global and subscale scores), we used multivariable linear regression with the same covariates. All regressions used the good-sleeper phenotype as reference.

Two pre-specified sensitivity analyses were performed: (i) exclusion of all participants with OSA documented in the medical-history field, to reduce confounding by previously diagnosed sleep-disordered breathing; (ii) re-derivation of the phenotypes using an alternative insomnia threshold of ISI ≥ 8 (subthreshold-or-greater insomnia[6]). A phenotype × sex interaction was tested for the primary outcome (BES ≥ 27). All tests were two-sided; $p < 0.05$ was considered statistically significant. Because primary associations were pre-specified, no correction for multiple testing was applied; 95% confidence intervals are reported throughout. Missing data for covariates and outcomes were handled by complete-case analysis; the number of observations for each model is reported in the corresponding table or figure caption.

3. RESULTS

3.1. Cohort characteristics

Of 2,303 adults completing the IES-2 questionnaire at Aviitam enrollment, 2,016 (87.5%) also completed both the ISI and the ESS and were included in the analysis (Table 1). The mean age was 45.2 ± 14.3 years, 76.2% were women, and the mean BMI was 37.1 ± 8.6 kg/m². BMI class distribution was: normal (< 25 kg/m²) 7.0%, overweight (25–29.9) 14.6%, obesity class I (30–34.9) 22.8%, class II (35–39.9) 21.9%, and class III (≥ 40) 33.7%. Previously diagnosed OSA was documented in 81 participants (4.0%).

Table 1
Cohort characteristics by sleep phenotype (n = 2,016).

Variable	Good sleeper	Isolated insomnia	Isolated sleepiness	Combined	Test
N (%)	1080 (53.6%)	367 (18.2%)	278 (13.8%)	291 (14.4%)	
Age (years), mean ± SD	45.2 ± 15.0	46.4 ± 13.9	44.8 ± 14.4	43.3 ± 13.2	F = 2.40
Sex — Women, n (%)	791 (73.2)	292 (79.6)	198 (71.2)	225 (77.3)	χ^2=7.60
Sex — Men, n (%)	274 (25.4)	71 (19.3)	74 (26.6)	64 (22.0)	χ^2=7.60
BMI (kg/m²), mean ± SD	37.8 ± 8.6	36.4 ± 8.7	37.1 ± 8.5	36.8 ± 8.8	F = 2.54
ISI (Morin) score, mean ± SD	7.1 ± 4.2	18.6 ± 3.0	8.9 ± 3.9	19.3 ± 3.4	F = 1319.34
ESS (Epworth) score, mean ± SD	5.3 ± 2.7	5.7 ± 3.0	14.4 ± 3.0	15.1 ± 3.1	F = 1433.92
HADS total, mean ± SD	13.0 ± 6.3	18.1 ± 6.8	15.7 ± 6.6	19.4 ± 6.4	F = 105.40
HAD-Anxiety, mean ± SD	7.7 ± 3.8	10.5 ± 4.1	8.9 ± 3.8	11.1 ± 4.1	F = 85.47
HAD-Depression, mean ± SD	5.3 ± 3.5	7.6 ± 3.9	6.8 ± 4.0	8.2 ± 3.9	F = 64.86
Perceived Stress Scale, mean ± SD	26.2 ± 7.9	30.5 ± 7.6	29.9 ± 7.6	32.7 ± 7.6	F = 59.80
BES total, mean ± SD	13.9 ± 8.9	15.2 ± 9.5	17.8 ± 9.4	19.4 ± 9.9	F = 31.30
IES-2 global, mean ± SD	3.0 ± 0.7	2.9 ± 0.7	2.7 ± 0.6	2.8 ± 0.7	F = 16.11
IES-2 EPR, mean ± SD	3.0 ± 1.0	2.9 ± 0.9	2.6 ± 0.9	2.7 ± 0.9	F = 17.43
IES-2 RHSC, mean ± SD	2.9 ± 1.0	2.8 ± 1.0	2.6 ± 1.0	2.7 ± 1.0	F = 5.59
IES-2 UPE, mean ± SD	2.9 ± 0.9	2.8 ± 0.9	3.0 ± 0.9	2.8 ± 0.9	F = 2.89
Documented OSA in chart, n (%)	35 (3.2)	21 (5.7)	13 (4.7)	12 (4.1)	χ^2=4.78

Values are mean ± SD or n (%). Documented OSA refers to previously diagnosed obstructive sleep apnea recorded in the structured medical-history field. F or χ^2 test reported for the four-group comparison.

3.2. Joint distribution of ISI and ESS and phenotype definition

The ISI and ESS were only moderately correlated (Pearson r = 0.31, Spearman ρ = 0.30). Applying the pre-specified clinical thresholds (ISI ≥ 15; ESS ≥ 11) partitioned the cohort into four phenotypes: good sleeper (n = 1,080, 53.6%), isolated insomnia (n = 367, 18.2%), isolated sleepiness (n = 278, 13.8%), and combined (n = 291, 14.4%). The four groups were comparable for age ($F = 2.4$, $p = 0.07$) and BMI ($F = 2.5$, $p = 0.06$); women were marginally over-represented in the insomnia (79.6%) and combined (77.3%) phenotypes compared with good sleepers (73.2%) and sleepiness (71.2%); $\chi^2 = 7.6$, $p = 0.06$. The prevalence of documented OSA was non-differentially distributed across phenotypes (3.2–5.7%, $\chi^2 = 4.8$, $p = 0.19$).

3.3. Mood, stress and eating profiles across phenotypes

Z-standardized means of the psycho-behavioral measures across the four phenotypes are shown in Fig. 1 (raw means ± SD in Table 1). On the mood and stress axis (HADS total, HAD-A, HAD-D, PSS), z-standardized means were lowest in good sleepers, highest and of similar magnitude in isolated insomnia and combined phenotypes, and intermediate in isolated sleepiness. On the eating axis (BES, IES-2 global and EPR subscale), z-standardized means were lowest in good sleepers, highest and of similar magnitude in isolated sleepiness and combined phenotypes (with BES higher and IES-2 scores lower), and intermediate in isolated insomnia. The UPE subscale did not differ significantly across phenotypes after adjustment ($F = 2.9$, $p = 0.03$ unadjusted; see section 3.5).

3.4. Adjusted associations with severe binge eating and mood symptoms

After adjustment for age, sex, and BMI (Fig. 2), adjusted odds ratios (aOR, 95% CI) for severe binge eating (BES ≥ 27) versus good sleepers were 1.69 (1.13–2.54, $p = 0.01$) for isolated insomnia, 2.55 (1.69–3.83, $p < 0.001$) for isolated sleepiness, and 3.38 (2.31–4.93, $p < 0.001$) for the combined phenotype. For clinically significant anxiety (HAD-A ≥ 11), aORs were 4.48 (3.41–5.89, $p < 0.001$) for isolated insomnia, 1.81 (1.33–2.46, $p < 0.001$) for isolated sleepiness, and 4.49 (3.34–6.04, $p < 0.001$) for the combined phenotype. For clinically significant depression (HAD-D ≥ 11), aORs were 3.09 (2.20–4.33, $p < 0.001$) for isolated insomnia, 1.99 (1.33–2.98, $p < 0.001$) for isolated sleepiness, and 3.22 (2.25–4.62, $p < 0.001$) for the combined phenotype. Crude prevalences of each threshold by phenotype are shown in Fig. 3: 27.3% of participants with the combined phenotype met criteria for severe binge eating versus 9.0% of good sleepers, and 55.1% met criteria for clinically significant anxiety versus 22.5% of good sleepers.

3.5. Adjusted associations with intuitive eating

In multivariable linear regression (adjusted for age, sex, and BMI), all three non-reference phenotypes showed lower IES-2 global scores compared with good sleepers, with the largest effect in isolated

sleepiness (β = − 0.24, 95% CI − 0.33 to − 0.15, p < 0.001), followed by the combined phenotype (β = − 0.23, − 0.32 to − 0.14, p < 0.001) and isolated insomnia (β = − 0.12, − 0.20 to − 0.04, p = 0.004). A similar but quantitatively stronger pattern was observed for the EPR subscale (isolated sleepiness β = − 0.37, combined β = − 0.32, isolated insomnia β = − 0.16; all p ≤ 0.007). No phenotype was significantly associated with the UPE subscale after adjustment.

3.6. Sensitivity analyses

After excluding the 81 participants with documented OSA (n = 1,935), aORs for BES ≥ 27 were 1.78 (1.18–2.68) for isolated insomnia, 2.69 (1.77–4.08) for isolated sleepiness, and 3.65 (2.49–5.37) for the combined phenotype; aORs for HAD-A ≥ 11 were 4.61 (3.48–6.11) for isolated insomnia and 1.78 (1.29–2.45) for isolated sleepiness. Point estimates and rank ordering across phenotypes were within 5–10% of those in the full cohort.

With the alternative insomnia threshold of ISI ≥ 8 (capturing subthreshold insomnia symptoms), phenotype prevalences shifted substantially (good sleeper 28.8%, isolated insomnia 43.0%, isolated sleepiness 4.7%, combined 23.5%). At this threshold, aORs were as follows: HAD-A ≥ 11 — isolated insomnia 4.04 (3.00–5.44), combined 5.61 (4.06–7.75); BES ≥ 27 — isolated sleepiness 2.31 (1.12–4.77), combined 4.15 (2.70–6.39). Confidence intervals for the isolated-sleepiness cell were wider, reflecting the smaller cell size at this threshold.

3.7. Effect modification by sex

Phenotype × sex interaction terms for severe binge eating were non-significant: insomnia × female OR 0.85 (95% CI 0.23–3.16, p = 0.81), sleepiness × female OR 0.74 (0.23–2.34, p = 0.61), and combined × female OR 1.47 (0.43–5.05, p = 0.54). Direction and magnitude of the main-effect aORs were similar in sex-stratified models, with wider confidence intervals in men (n = 483) than in women (n = 1,506).

4. DISCUSSION

4.1. Principal findings

In this large cross-sectional analysis of 2,016 adults seeking specialist care for clinical obesity, joint phenotyping on validated thresholds of insomnia severity and excessive daytime sleepiness identified four groups with distinct, partly non-overlapping profiles of eating and mood disturbance. Confirming our pre-specified hypothesis, isolated insomnia was preferentially associated with clinically significant anxiety and depression, while isolated daytime sleepiness was preferentially associated with severe binge eating and lower intuitive eating, particularly on the EPR subscale. The combined phenotype carried the highest risk across all outcomes. All associations were robust to adjustment for age, sex, and BMI, to exclusion of participants with previously diagnosed OSA, and to an alternative, lower insomnia threshold. The stability of the double-dissociation pattern after excluding participants with documented OSA argues against residual confounding by previously diagnosed sleep-disordered breathing as the main driver of our findings. Importantly, the four phenotypes differed negligibly in BMI, arguing further

that the observed psycho-behavioral heterogeneity is not a simple reflection of obesity severity. The absence of significant phenotype × sex interactions and the similar direction and magnitude of aORs in sex-stratified models suggest that the double-dissociation pattern generalizes to both women and men, though precision was necessarily lower in the smaller male subgroup.

4.2. Comparison with previous literature

The moderate ISI–ESS correlation (ρ = 0.30) observed here is consistent with general-population estimates[9] and supports the psychometric premise that the two instruments index partially independent dimensions of sleep health. Prior studies in clinical obesity have typically reported each dimension in isolation: insomnia prevalence in clinical-obesity samples ranges from 20% to 40% using ISI ≥ 15,[3,25] and ESS-defined excessive daytime sleepiness affects 20–50% depending on setting and OSA screening.[4,26] Our prevalences of moderate-to-severe insomnia (32.6% across both insomnia and combined phenotypes) and of excessive daytime sleepiness (28.2%) are within those ranges.

The differential associations we report extend a scattered literature. Insomnia is a well-established, prospectively bidirectional risk factor for anxiety and depression in the general population,[10,11,27] with meta-analytic odds ratios for depressive disorders around 2–3. Our adjusted odds ratios for HAD-defined anxiety (4.5) and depression (3.1) in the isolated-insomnia phenotype are of similar magnitude but in a clinical-obesity setting, and importantly they are largely preserved even when using a less stringent ISI threshold.

Associations between short or fragmented sleep and disordered eating, particularly binge eating, have also been reported in community and clinical samples.[12,28,29] Our finding that isolated daytime sleepiness, but not isolated insomnia, carries the strongest binge-eating signature in clinical obesity suggests that the eating-sleep pathway may be more closely linked to effective sleep quantity or to disorders such as undiagnosed OSA or circadian misalignment — both of which manifest with sleepiness rather than insomnia — than to subjective sleep-maintenance difficulty alone. Our previous work on this cohort identified a two-cluster solution combining emotional distress and intuitive eating that was associated with weight loss under semaglutide;[30] the present paper extends that framework by showing that sleep symptoms, once analyzed on their own two dimensions, help resolve the emotional and eating components into separable phenotypes. The absence of a gradient across phenotypes on the UPE subscale, contrasting with the strong gradients observed on the EPR and RHSC subscales, is consistent with the growing body of evidence that UPE operates as a structurally distinct component of intuitive eating, supporting its reconsideration in the ongoing revision of the IES.[15]

4.3. Mechanistic interpretation

Three complementary mechanisms may account for the observed double dissociation. First, the insomnia–mood pathway is likely to reflect shared hyperarousal and cortical-cognitive-emotional dysregulation, with HPA-axis activation, reduced REM sleep and alterations of limbic connectivity contributing to affective symptoms.[27] Second, the sleepiness–eating pathway may reflect sleep-duration pathways: short or fragmented sleep increases ghrelin, decreases leptin, and enhances hedonic food

reward, facilitating loss-of-control eating and binge episodes.[31][32] Third, the observation that the combined phenotype does not simply add the two single-phenotype effects but rather approximates the more severe of the two, for each outcome, is compatible with partial mechanistic redundancy at the level of the final clinical expression.

4.4. Implications for clinical obesity care

Specialist obesity care is increasingly seen as a personalised, multi-component intervention combining nutritional, pharmacological, psychological and behavioural support, and the success of newer pharmacotherapies highlights the need for behavioural sub-typing to identify which patients benefit most from which adjunct intervention. Joint screening of insomnia and daytime sleepiness — using the ISI and the ESS at intake of specialist obesity consultations — is inexpensive, feasible in under five minutes, and, on the basis of our data, identifies phenotypes that may benefit from different initial therapeutic strategies. Patients with isolated insomnia and an anxio-depressive load may benefit most from cognitive-behavioural therapy for insomnia, with concomitant screening and treatment of mood disorders, and from psychological support targeting the affective barriers to weight management. Patients with isolated daytime sleepiness may warrant polysomnography to exclude OSA, assessment of sleep duration and circadian regularity, and early behavioural support for eating regulation, including referral for binge-eating-targeted psychotherapy when indicated. Patients with the combined phenotype, who carry the highest risk across all outcomes, likely need integrated psychological and sleep interventions concurrently and warrant particular attention in obesity-care planning.

4.5. Strengths and limitations

Strengths include a large and well-characterized clinical-obesity cohort with simultaneous administration of validated instruments for insomnia, sleepiness, intuitive eating, binge eating, anxiety, depression, and stress; pre-specified phenotype definitions based on internationally used clinical thresholds; adjustment for core confounders; and multiple sensitivity analyses that preserved the main findings.

Several limitations warrant emphasis. First, the design is cross-sectional and cannot establish causality or temporal precedence; longitudinal confirmation and ideally randomized trials targeting each phenotype are needed. Second, we did not perform systematic polysomnography; documented OSA was captured from the medical-history field and substantially underestimates true prevalence, since the majority of OSA cases in adults remain undiagnosed in routine clinical settings.[24] However, the ISI–ESS phenotype associations persisted after excluding participants with documented OSA, and BMI was comparable across phenotypes, both of which argue against confounding by sleep-disordered breathing as the sole explanation of our findings. Third, the cohort is drawn from a single-country, largely regional digital platform serving specialist obesity care in France and predominantly in Occitanie; external validation in other care settings and other countries is needed. Fourth, symptom severity was ascertained by validated screening instruments rather than structured psychiatric interviews. Fifth, the BES, HADS, and ISI are subjective self-reports and may be influenced by shared method variance.

4.6. Conclusions

In adults with clinical obesity, insomnia and excessive daytime sleepiness identify distinct, partly non-overlapping behavioural phenotypes: an insomnia–anxio-depressive profile, a sleepiness–binge-eating profile, and a combined profile with the highest overall burden. These associations are independent of age, sex, and BMI, and are preserved when participants with previously diagnosed obstructive sleep apnoea are excluded. Joint screening of insomnia and sleepiness at intake of specialist obesity care is inexpensive, feasible, and may help tailor psychological and behavioural interventions, supporting the broader effort to personalise multidisciplinary obesity management.

Declarations

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AUTHOR CONTRIBUTIONS

A.A.: Conceptualization, Methodology, Data curation, Formal analysis, Visualization, Writing – original draft, Scientific supervision of the manuscript. A.A. served as both first and senior author; the order of co-authors reflects the relative magnitude of their contribution and does not imply a senior-author role for the last-listed co-author. J.-B.B., S.T., C.B., C.L.R., J.M., A.S.: Investigation / Data collection, Writing – review & editing. All authors read and approved the final version of the manuscript.

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DISCLOSURE STATEMENT

Financial disclosure: Antoine Avignon was a major shareholder in Aviitam in the past 2 years, the company that developed the online platform used to collect the data. The other authors declare no competing financial interests. Non-financial disclosure: none.

DATA AVAILABILITY

Pseudonymized data supporting the findings of this study are available from the corresponding author upon reasonable request and subject to approval by the relevant data-protection authorities.

ETHICS

The study was approved by the institutional review board of the University Hospital of Montpellier (IRB-MTP_2023_02_202301328) and registered on ClinicalTrials.gov (NCT06125600). All participants

provided informed consent through the platform's General Terms and Conditions of Use, which include a clause for research purposes.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the author used Claude (Anthropic) to assist with manuscript drafting, language editing, and structuring of the text, and to support data analysis and figure generation under the author's direct supervision. All statistical analyses, clinical interpretations, and scientific conclusions were verified and validated by the author, who reviewed and edited all AI-generated content as appropriate and takes full responsibility for the content of the publication.

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Figures

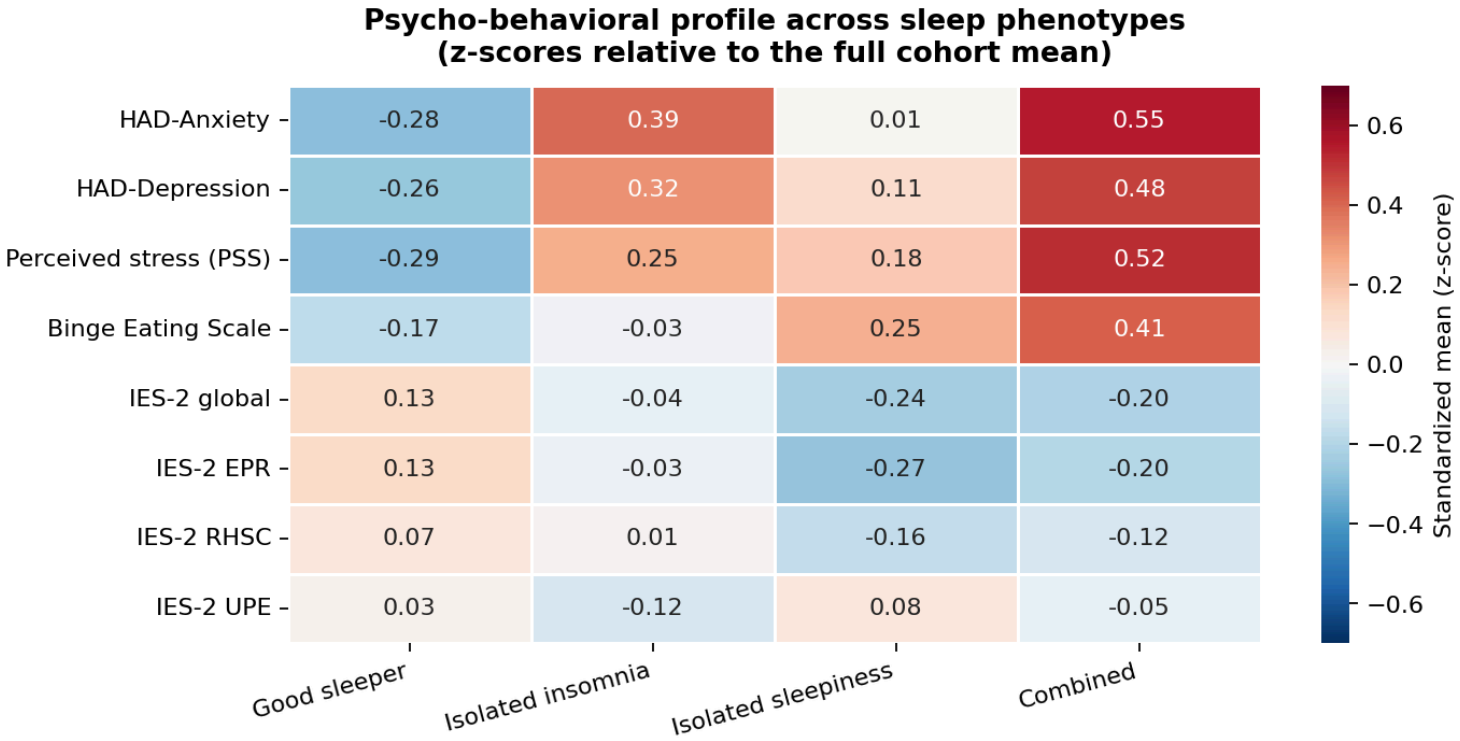


Figure 1

Psycho-behavioral profile across sleep phenotypes.

Heatmap of standardized mean (z-score relative to the full cohort mean) for validated measures of anxiety (HAD-A), depression (HAD-D), perceived stress (PSS), binge eating (BES), and intuitive eating (IES-2 global and subscales). Red indicates above-cohort mean, blue below-cohort mean. The pattern highlights a mood axis dominated by the isolated-insomnia and combined phenotypes, and an eating axis dominated by the isolated-sleepiness and combined phenotypes. n = 2,016.

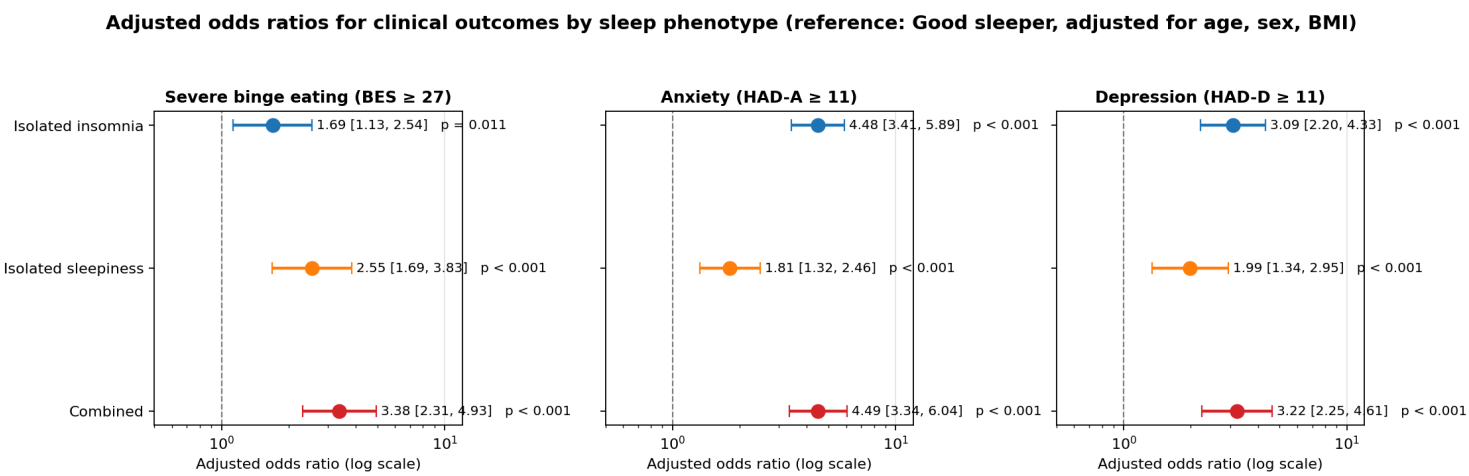


Figure 2

Adjusted odds ratios for clinical outcomes by sleep phenotype.

Forest plot of adjusted odds ratios (95% CI) for severe binge eating (BES ≥ 27), clinically significant anxiety (HAD-A ≥ 11), and clinically significant depression (HAD-D ≥ 11). Reference: good sleeper. Adjusted for age, sex, and BMI (multivariable logistic regression). Sample sizes: BES model n = 1,642 (228 events); HAD-A n = 1,747 (591 events); HAD-D n = 1,748 (276 events).

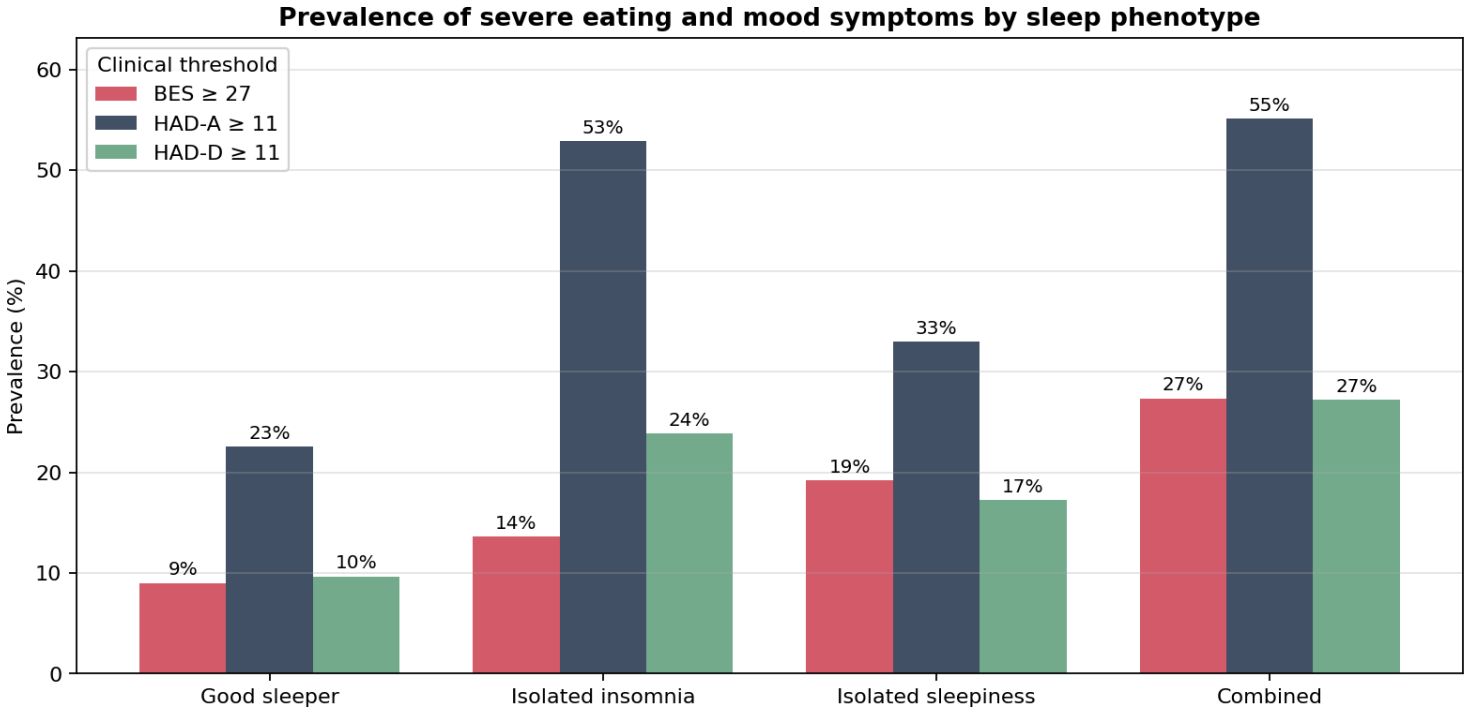


Figure 3

Prevalence of clinical thresholds by sleep phenotype.

Bar chart of observed prevalence of severe binge eating (BES ≥ 27), clinically significant anxiety (HAD-A ≥ 11), and clinically significant depression (HAD-D ≥ 11) across the four sleep phenotypes. Crude percentages; see Figure 2 for adjusted estimates.