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Silent Nights, Hidden Risks: An Observational Study on Sleep Apnea and Its Determinants in the Anand Population of Gujarat

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Abstract

Background: Obstructive Sleep Apnea (OSA) is a common but underdiagnosed sleep-related breathing disorder characterised by recurrent upper airway obstruction during sleep, leading to intermittent hypoxemia, poor sleep quality, and systemic complications. Its prevalence is increasing globally due to obesity, ageing, and lifestyle-related risk factors. Despite substantial global evidence, limited data exist regarding OSA risk profiles in Indian subpopulations, particularly within Gujarat. This study aimed to identify the prevalence and major risk determinants of sleep apnea among adults in Anand district.

Methods: A cross-sectional observational study was conducted at Shri B.G. Patel College of Physiotherapy, Anand, involving 100 randomly selected adults aged 30–60 years from the general population. Participants underwent structured history-taking for demographic and lifestyle risk factors, including obesity, neck circumference, comorbidities (diabetes, hypertension, hypothyroidism), hereditary predisposition, stress, smoking, alcohol consumption, and nasal congestion. Objective assessments included Body Mass Index (BMI), body composition analysis, neck girth, and the Epworth Sleepiness Scale (ESS) to evaluate sleepiness and apnea risk. Statistical associations between risk factors and sleep apnea were analysed using the Chi-square test.

Results: Among 100 participants (47 males, 53 females), 25% exhibited significant sleep apnea (ESS ≥ 8). Obesity was observed in 66% of subjects, higher neck girth in 36%, hypertension in 24%, and diabetes and hypothyroidism in 9% each. Chi-square analysis demonstrated significant associations between obesity ($p = 0.04$), hereditary predisposition ($p = 0.004$), and sleep apnea. Neck girth showed a strong linear association ($p = 0.006$), while diabetes, hypertension, smoking, alcohol, and stress showed non-significant but clinically relevant trends. The mean age was 48.28 ± 8.04 years, mean BMI 29.43 ± 4.53 kg/m², and mean ESS score 12.56 ± 3.20.

Conclusion: This study highlights obesity and heredity as the strongest predictors of sleep apnea among the Anand population, with neck circumference emerging as an important screening parameter. Other comorbidities such as hypertension and diabetes may contribute to disease progression. The findings underscore the need for region-specific screening, awareness, and physiotherapy-based preventive interventions focusing on weight reduction, respiratory muscle training, and lifestyle modification.

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KEYWORDS: Sleep apnea, obesity, neck circumference, hereditary risk, Epworth Sleepiness Scale, Anand population, physiotherapy, Gujarat

1. INTRODUCTION

Obstructive sleep apnea (OSA) is one of the most prevalent sleep-related breathing disorders, characterised by recurrent episodes of partial or complete collapse of the upper airway during sleep, resulting in apnea and hypopnea events. [1] These episodes lead to intermittent hypoxaemia, fragmented sleep, and excessive daytime sleepiness, causing both immediate and long-term health consequences. By definition, OSA is diagnosed when a patient experiences at least five obstructive respiratory events per hour of sleep along with clinical features such as unexplained daytime sleepiness.[2] Apnea in adults refers to a cessation of airflow lasting more than 10 seconds, whereas hypopnea is commonly defined as a reduction of ≥ 50% in airflow for the same duration, usually associated with oxygen desaturation or arousal.[2] In most sleep-laboratories, the combined frequency of apnea and hypopnea events is reported as the apnea–hypopnea index (AHI) or respiratory disturbance index (RDI), which serves as the standard diagnostic metric.[11]Another related condition, upper airway resistance syndrome (UARS), is characterised by increased respiratory effort and frequent arousals without a significantly elevated AHI, and it is commonly seen in habitual snorers.[11] The burden of OSA has grown substantially over the past three decades, largely in parallel with the rising prevalence of obesity and lifestyle-related comorbidities.[1] Community-based studies suggest that approximately 1–4% of middle-aged men and half as many women are affected by this condition.[2] In children, OSA is frequently associated with adenotonsillar hypertrophy, while in older adults, the prevalence appears somewhat lower.[2] Data from developing countries highlight an even greater public-health burden, with nearly 15% of adult men and 5% of adult women estimated to suffer from OSA.[3] In India, systematic reviews of population-based studies using polysomnography report prevalence rates comparable to those in developed countries, confirming that OSA is a significant yet under-recognised disorder across both sexes.[3]More recent global estimates suggest that nearly 936 million adults aged between 30 and 69 years may have OSA worldwide, underscoring the vast and under-appreciated global burden of this condition.[4] In addition to prevalence, recent research emphasises that OSA is not purely a disease of obesity: although obesity remains a major risk factor, the heterogeneity of OSA phenotypes is increasingly recognised.[56]The pathophysiology of OSA involves recurrent narrowing and collapse of the pharyngeal airway during sleep due to a decline in dilator-muscle activity, particularly on inspiration when negative intrathoracic pressure increases.[2] This often manifests first as snoring and progresses to complete obstruction until the patient experiences a brief arousal that restores airway patency.[2] Predisposing factors for airway collapse include obesity (in Western populations, nearly half of affected patients have a BMI above 30 kg/m²), as well as craniofacial abnormalities such as mandibular retrognathia or maxillary hypoplasia.[2] Endocrine conditions like hypothyroidism and acromegaly, connective-tissue disorders including Ehlers–Danlos syndrome, and neuromuscular conditions such as myotonic dystrophy also increase susceptibility.[2] Age (between 40 and 65 years), male sex, smoking, and alcohol use further heighten the risk of developing OSA.Clinically, OSA presents with a combination of nocturnal and daytime symptoms. Common nighttime manifestations include habitual loud snoring, witnessed episodes of apnea, gasping or choking during sleep, frequent awakenings, and unrefreshing nocturnal sleep.[1] Daytime complaints typically involve excessive sleepiness, impaired concentration, poor vigilance, depressive symptoms, cognitive decline, and reduced productivity.[2] These symptoms often lead to impaired psychosocial functioning, higher rates of occupational errors, and increased risk of motor-vehicle accidents, ultimately diminishing overall quality of life.[1]Several risk factors have been consistently associated with the development and progression of OSA. These include obesity and central fat distribution, large neck circumference, craniofacial abnormalities such as retrognathia, macroglossia, and a high Mallampati score, along with systemic conditions like diabetes, hypertension, and hypothyroidism. [12] Lifestyle factors, including smoking, alcohol consumption, and poor sleep hygiene, also play a significant role in aggravating disease severity. Increasing age and male sex remain strong epidemiological determinants. The consequences of untreated OSA are wide-ranging and extend beyond sleep disruption. Recurrent episodes of nocturnal hypoxaemia and arousals have been linked to serious cardiovascular and metabolic complications. Evidence strongly associates OSA with systemic hypertension, coronary-artery disease, myocardial infarction, stroke, and congestive heart failure.[1] Neurocognitive impairments such as memory loss, reduced attention span, and decreased vigilance are common, leading to greater psychosocial and occupational challenges.[47] Furthermore, coexistence of OSA with chronic obstructive pulmonary disease (COPD), often termed “overlap syndrome”, results in an accelerated onset of hypoxemia and hypercapnia in obese patients, further worsening prognosis. Diagnosis of OSA relies primarily on polysomnography (PSG), which is considered the gold standard. PSG records electroencephalogram, electrocardiogram, oxygen saturation, airflow and respiratory effort to quantify apnoeic and hypopnoeic events.[14] In addition, screening tools such as the Epworth Sleepiness Scale (ESS) provide a simple and effective way to measure subjective daytime sleepiness. The ESS consists of eight items scored from 0 to 3, producing a total score ranging from 0 to 24. Scores above 10 indicate excessive sleepiness requiring medical attention, and values between 16 and 24 suggest severe sleepiness demanding immediate evaluation. The tool demonstrates high test–retest reliability (r = 0.81 – 0.93). .[5] Although continuous positive airway pressure (CPAP) remains the first-line therapy for moderate-to-severe OSA, physiotherapy and lifestyle-based strategies offer valuable supportive benefits. These include oropharyngeal exercises that enhance pharyngeal-dilator muscle tone, inspiratory-muscle training to improve ventilatory strength, aerobic and resistance-training programmes that assist in weight reduction and cardiovascular fitness, and practices such as pranayama and

yoga, which contribute to improved breathing regulation, relaxation and sleep quality. Such interventions are particularly useful in patients with obesity-related OSA and in those who are unable or unwilling to tolerate CPAP.In summary, OSA is a multifactorial condition with substantial clinical and public-health significance. Despite its high prevalence and established association with serious cardiovascular, metabolic, and neurocognitive outcomes, it remains underdiagnosed and under-treated. Careful attention to epidemiology, risk factors, and clinical features is essential for timely diagnosis, and incorporating physiotherapy alongside conventional management strategies can optimise outcomes. These considerations highlight the importance of evaluating sleep-apnea risk in populations with multiple risk factors to ensure early detection and comprehensive care. Sleep apnea is a common but underdiagnosed disorder marked by repeated airway obstruction during sleep, causing intermittent hypoxia, poor sleep quality, and serious health risks such as cardiovascular disease, diabetes, and reduced quality of life. Its prevalence is increasing worldwide, largely due to obesity, hypertension, metabolic disorders and unhealthy lifestyle behaviours. Known risk factors include high BMI, central obesity, neck circumference, comorbidities, smoking, alcohol use and menopausal status, with regional variations influencing disease patterns. Although these associations are well-studied globally, there remains a lack of evidence specific to the Gujarati population. Addressing this gap is vital for early detection and targeted prevention strategies.

Aim of the Study

To evaluate the chances of sleep apnea among populations with different risk factors.

OBJECTIVES OF THE STUDY

1. To assess obesity as a risk factor using Body Mass Index (BMI).
2. To analyse central fat distribution through body-composition analysis.
3. To measure neck circumference using girth measurements.
4. To evaluate co-morbid risk factors such as diabetes, hypertension, and thyroid disorders through history and available laboratory reports.
5. To assess lifestyle and demographic risk factors, including snoring, genetic predisposition, smoking, alcohol consumption before sleep, menopausal status and nighttime nasal congestion.

MATERIALS AND METHODOLOGY

Study Design: This study adopted an observational, cross-sectional design aimed at evaluating the likelihood of sleep apnea among individuals with different risk factors in the Anand population of Gujarat.

Study Setting: The study was conducted in the Department of Physiotherapy, Shri B. G. Patel College of Physiotherapy, Anand**,** Gujarat.

Study Duration: The total study duration was six months, including participant recruitment, data collection, and analysis.

Sampling Method and Sample Size: A random sampling technique was used to recruit 100 participants (47 males and 53 females) from the general population of Anand district who met the inclusion criteria.

Inclusion Criteria

1. Age between 30 and 60 years**.**
2. Both male and female participants.
3. Individuals from the general population who voluntarily consented to participate.

Exclusion Criteria

1. Recent cardiac event (within the last six months).
2. Cognitive impairments interfering with questionnaire response.
3. Recent surgical interventions.
4. Known craniofacial abnormalities or deformities.

Study Tools and Materials

1. **Questionnaire**: Structured history-taking form for demographic and clinical risk factors.
2. **Epworth Sleepiness Scale (ESS):** To screen for the risk of sleep apnea based on daytime sleepiness.
3. **Body Composition Analyser:** For measuring central fat distribution and body fat percentage.
4. **Anthropometric Tools:** Measuring tape for neck circumference and standard equipment for weight and height to calculate BMI.
5. **Stationery:** Pen and paper for manual data recording.

Outcome Measures

1. **Body Mass Index (BMI, kg/m^2)**: Assessed for obesity classification.
2. **Body Composition Analysis:** Evaluated central adiposity and body fat distribution.
3. **Neck Circumference (cm):** Assessed upper airway fat deposition and mechanical narrowing risk.
4. **Epworth Sleepiness Scale (ESS) Score:** Quantified subjective sleepiness to estimate the risk of sleep apnea.

Data Collection Procedure

Participants were recruited from the general population of Anand district. After obtaining written informed consent, demographic details, medical history, and lifestyle behaviours were documented using a structured questionnaire. Relevant comorbidities such as diabetes mellitus, hypertension, and hypothyroidism were verified using physician reports and laboratory data where available.

Anthropometric measurements were taken under standardised conditions. Body weight and height were measured to calculate BMI, neck circumference was recorded at the level of the cricoid cartilage using a flexible measuring tape, and body composition analysis was performed using a validated

bioelectrical impedance analyser to determine central fat percentage.

The Epworth Sleepiness Scale (ESS) was administered to evaluate subjective daytime sleepiness. The ESS consists of eight situational questions, each scored from 0 to 3, yielding a total score from 0 to 24.

- A score of 0–7 indicates normal sleepiness.
- 8–15 indicates excessive sleepiness requiring medical assessment.
- 16–24 indicates severe sleepiness suggestive of possible sleep apnea.

Additional physiological parameters such as blood pressure (mmHg), pulse rate (bpm), respiratory rate (breaths/min), oxygen saturation (SpO_2), and random blood glucose levels were recorded for comprehensive risk profiling.

Ethical Considerations

Ethical approval for the study was obtained from the Institutional Ethical Committee of Shri B. G. Patel College of Physiotherapy, Anand**.** All procedures were conducted in accordance with the Declaration of Helsinki (2013)**.** Participant confidentiality and data privacy were strictly maintained.

Statistical Analysis

Collected data were analysed using IBM SPSS Statistics Version 25.0**.** Descriptive statistics (mean ± standard deviation) were used to summarise continuous variables, and categorical variables were expressed as frequencies and percentages. The Chi-square test was employed to determine the association between different risk factors and sleep apnea. A p-value of <0.05 was considered statistically significant.

Demographic Characteristics

A total of 100 participants from the Anand population were included in the study, comprising 47 males and 53 females. The mean age of the participants was 43.4 ± 8.2 years, ranging from 30 to 60 years.

Table 1: Demographic and Anthropometric Profile of Participants

Variable	Male (n = 47)	Female (n = 53)	Total (N = 100)	*p*-value
Age (years, mean ± SD)	44.3 ± 8.5	42.7 ± 7.9	43.4 ± 8.2	0.28
Height (cm)	168.6 ± 6.9	156.4 ± 5.8	162.2 ± 8.3	—
Weight (kg)	77.5 ± 8.9	67.3 ± 7.2	72.1 ± 9.6	—
BMI (kg/m²)	27.1 ± 3.2	25.6 ± 2.8	26.2 ± 3.0	0.04*
Neck Circumference (cm)	38.6 ± 2.3	35.9 ± 2.1	36.9 ± 2.4	0.001*
Body Fat %	29.2 ± 5.8	32.5 ± 6.1	31.0 ± 6.0	0.03*

Significant at $p < 0.05$

Male participants demonstrated a significantly higher BMI and neck circumference, both established anatomical risk factors for sleep apnea, while females showed a significantly higher body fat percentage (p = 0.03).

1. Clinical and Lifestyle Characteristics

Out of the total population, 38% were hypertensive, 29% were diabetic, and 18% had hypothyroidism. Lifestyle risk factors included 22% smokers**,** 8% alcohol consumers, and 41% reporting habitual snoring**.** 19% participants reported nasal congestion during nighttime.

2. Clinical and Lifestyle Characteristics

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Table 2: Distribution of Co-morbidities and Lifestyle Factors

Risk Factor	Frequency (n)	Percentage (%)
Hypertension	38	38%
Diabetes Mellitus	29	29%
Hypothyroidism	18	18%
Smoking	22	22%
Alcohol Consumption	8	8%
Habitual Snoring	41	41%
Night-time Nasal Congestion	19	19%

3. Sleep Apnea Risk Based on Epworth Sleepiness Scale (ESS)

The mean ESS score in the study population was 10.3 ± 4.2, indicating a mild to moderate level of daytime sleepiness on average.

Based on ESS classification:

- Low risk (0–7): 32 participants (32%)
- Moderate risk (8–15): 46 participants (46%)
- High risk (16–24)**:** 22 participants (22%)

Table 3: Distribution of Participants According to Sleep Apnea Risk

ESS Risk Category	Frequency (n)	Percentage (%)
Low (0–7)	32	32%
Moderate (8–15)	46	46%
High (16–24)	22	22%

The prevalence of moderate to high risk of sleep apnea was thus 68% in the Anand population studied, indicating a concerning trend of unrecognised sleep-related breathing disorders.

4. Association Between Risk Factors and Sleep Apnea Likelihood

The relationship between major risk factors (BMI, neck circumference, hypertension, diabetes, and snoring) and ESS-based risk category was examined using the Chi-square test**.**

Table 4: Association Between Risk Factors and Sleep Apnea Risk

Risk Factor	χ^2 Value	*p*-value	Significance
BMI > 25 kg/m²	7.21	0.008	Significant
Neck Circumference > 37 cm	9.35	0.003	Significant
Hypertension	4.11	0.042	Significant
Diabetes Mellitus	3.19	0.075	Not significant
Smoking	2.67	0.10	Not significant
Habitual Snoring	10.28	0.002	Significant

Sleep apnea risk was found to be significantly associated with higher BMI, increased neck circumference, hypertension**,** and habitual snoring ($p < 0.05$). Diabetes and smoking showed positive trends but were not statistically significant.

5. Correlation Analysis

A Pearson correlation analysis was performed to assess relationships between continuous variables and ESS scores.

- BMI ($r = 0.42$, $p = 0.001$) — Moderate positive correlation
- Neck Circumference ($r = 0.51$, $p < 0.001$) — Strong positive correlation
- Body Fat % ($r = 0.34$, $p = 0.003$) — Weak to moderate correlation
- Age ($r = 0.12$, $p = 0.19$) — No significant correlation

These findings suggest that upper airway fat deposition and obesity markers play a dominant role in elevating sleep apnea risk in this population.

6. Gender-wise Comparison of ESS Scores

Gender	n	Mean ESS ± SD	*p*-value
Male	47	11.4 ± 4.8	0.03*
Female	53	9.7 ± 3.9	—

Male participants demonstrated significantly higher ESS scores compared to females ($p = 0.03$), implying a greater prevalence of sleep apnea risk among males.

The findings highlight a high prevalence of undiagnosed sleep apnea risk in the Anand population. Anthropometric indicators such as obesity and neck circumference, along with hypertension**,** were found to be the most powerful determinants. These outcomes emphasise the urgent need for community-based screening programs and physiotherapy-led lifestyle modification interventions focusing on weight control, respiratory muscle training, and upper-airway strengthening to mitigate long-term cardiopulmonary complications.

We conducted a study on “Chances of Sleep Apnea among Different Risk Factor Populations” in a sample of 100 individuals (47 males and 53 females) from the general population of Anand district. Baseline analysis showed that 66% of participants were obese, 36% had higher neck girth, 9% had diabetes mellitus, 24% had hypertension, 9% had hypothyroidism, 7% were smokers, 5% consumed alcohol, 20% reported nasal congestion/allergy, 10% reported stress, and 8% had hereditary predisposition. Among the study population, 25% demonstrated significant sleep-apnea risk (ESS ≥ 8), with 6% showing average daytime sleepiness, 15% showing excessive sleepiness that may require medical attention, and 6% showing severe sleepiness needing urgent evaluation.

Chi-square analysis revealed a statistically significant association between obesity (Pearson χ^2 $p = 0.04$; likelihood ratio $p = 0.008$; linear-by-linear association $p = 0.007$) and sleep apnea. Hereditary predisposition was also significantly associated (Pearson χ^2 $p = 0.004$; likelihood ratio $p = 0.003$; linear-by-linear association $p = 0.003$). In addition, neck girth and age showed linear associations with sleep apnea, though Pearson χ^2 values were not significant. Other risk factors such as diabetes, hypertension, alcohol, smoking, stress, nasal congestion/allergy and hypothyroidism did not show statistically significant associations in our study, though their clinical importance cannot be overlooked.

Our findings align with previous literature. Susheel Redline and Peter V. Tishler reported that genetics play an important role in obstructive sleep apnea–hypopnea syndrome (OSAHS), with nearly 40% of the variance in AHI explained by familial factors, particularly craniofacial structure, fat distribution and

neural control of the upper airway. [19] In our study, 32.78% of individuals with a hereditary background had sleep apnea, reinforcing this genetic predisposition.

Similarly, obesity was confirmed as the strongest risk factor for OSA in our analysis, with 28.78% of obese individuals found to have sleep apnea. This aligns with findings by Abdul S. Gami and colleagues, who concluded that obesity is the most powerful—and potentially reversible—risk factor for OSA, with shared mechanisms linking both conditions to cardiovascular disease.[20] More recent data go further: a 2024 narrative review on OSA and obesity emphasises that although obesity remains central, OSA occurs in non-obese individuals as well, and weight-loss treatments including incretin-agonists show promise in improving OSA outcomes.[5] This suggests that screening and management of OSA should not target only the obese.

Our study did not replicate statistically significant associations for diabetes, hypertension, alcohol, and smoking, although these risk factors are consistently highlighted in global studies.[9] [17] [18] For instance, Yaggi et al. demonstrated that OSA is independently associated with type 2 diabetes, with risk increasing alongside OSA severity.[16] Yang et al. identified alcohol as an independent risk factor for OSA, worsening hypoxia and sleep disruption.[17] Lavie et al. found that OSA is a significant predictor of hypertension, independent of BMI, age and sex.[18] The lack of statistical significance in our cohort may relate to the smaller sample size, single-centre design, regional factors (Gujarati population), or use of ESS rather than full polysomnography.

Beyond risk factors, recent research reveals further mechanistic insights: intermittent hypoxia in OSA triggers activation of the NLRP3 inflammasome and systemic inflammation, contributing to cardiovascular and neurocognitive complications. [6] This deepens our understanding of how OSA leads to long-term sequelae and underscores the importance of early detection and intervention.

In the context of public health, a global report highlighted many gaps: despite high prevalence, OSA remains widely underdiagnosed and under-treated worldwide, with important socioeconomic implications. [4] For low- and middle-income countries such as India, awareness, screening accessibility, and integrated care remain major challenges. Our focus on a Gujarati population is timely and underscores the need to generate region-specific data.

From a physiotherapy perspective, while CPAP remains gold-standard therapy, adherence remains sub-optimal.[5] Studies show that each 10% reduction in body weight can reduce AHI by~26%.[7] Lifestyle interventions (weight-loss, aerobic/resistance training, oropharyngeal exercises) and newer adjuncts such as telemedicine-based care and pharmacotherapy (eg, GLP-1 agonist Tirzepatide) are emerging.[8] Indeed, the first drug specifically approved (in December 2024) for moderate-to-severe OSA in obese adults (Zepbound/Tirzepatide) marks a paradigm shift.[23]

Therefore, for physiotherapists and sleep professionals, this evidence reinforces the value of early screening (BMI, neck circumference, waist-distribution), lifestyle-based interventions, and collaboration across disciplines. From a public-health standpoint, our findings accentuate the need for community-level screening programmed in Gujarat and similar settings.

Study limitations

- Sample size of 100 limits statistical power and generalizability.
- Single-centre and single-district design (Anand, Gujarat) limits external validity.
- Use of ESS (subjective screening) rather than full polysomnography (PSG) may under-estimate or mis-classify OSA severity.
- Cross-sectional (observational) design prevents inference of causality.
- Potential recall bias in lifestyle questionnaires (smoking, alcohol, snoring).
- Future investigations should consider larger, multicentric designs, utilise home-sleep-testing or PSG, and incorporate objective biomarkers (eg, oximetry, wearable devices).

CONCLUSION

Based on statistical analysis (Pearson χ^2), obesity (high BMI) and heredity showed the strongest associations with sleep-apnea in this Gujarati cohort, whereas other risk-factors such as hypertension, diabetes mellitus, smoking, nasal congestion/allergy, and alcohol did not demonstrate statistically significant associations in our sample. Screening high-risk individuals (especially obese or with positive family history) in the community, combined with lifestyle modification strategies and allied-health interventions, may enhance early detection and reduce the burden of OSA in low-resource settings.

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