Review Article

Bidirectional Relationship between Obstructive Sleep Apnea and Head and Neck Cancer: A Systematic Review of Current Evidence

Abdullah A. Alamri[1], Alhassan A. Almakrami[2], Reem F. Hawkash[2], Solaf A. Alsedran[3], Jumanah A. Al Fadhil[3], Mohammed I. Almakrami[2], Faisal M. Assiri[2], Jawharah S. Alkorbi[3], Muath H. Alqesair[4]

[1]Otorhinolaryngology Head & Neck Surgery Department, Taibah University, Madinah, KSA. [2]Medical intern, Najran University, Najran City, KSA. [3]Medical student, Najran University, Najran City, KSA. [4]Medical School, University of Szeged Albert Szent-Györgyi, Szeged, HUN.

ABSTRACT

A growing body of evidence suggests a complex, bidirectional relationship between obstructive sleep apnea (OSA) and head and neck cancer (HNC), though the literature remains fragmented. This systematic review consolidates current evidence to examine this dual association, exploring both the impact of OSA on HNC biology and outcomes, and the role of HNC and its treatment in the development or exacerbation of OSA. A systematic search of PubMed/MEDLINE, Embase, Cochrane Central, and Web of Science was conducted from January 2021 to December 2025 in accordance with PRISMA guidelines. Five studies met the inclusion criteria. The evidence demonstrates a consistently high prevalence of OSA in HNC populations, both pre- (up to 90%) and post-treatment (up to 72%). For the direction of OSA influencing HNC, pre-treatment OSA severity (Apnea-Hypopnea Index) was significantly correlated with larger primary tumor size and associated with increased tumor recurrence and mortality in one study. Current evidence supports a bidirectional relationship between OSA and HNC. HNC treatment is a significant risk factor for OSA, creating a major survivorship concern. Preliminary evidence suggests pre-existing OSA may be associated with more aggressive tumor behavior and worse treatment tolerance.

Keyword: Obstructive Sleep Apnea; Head and Neck Neoplasms; Squamous Cell Carcinoma of Head and Neck; Bidirectional Relationship.

Introduction

With an estimated 930,000 new cases and 467,000 deaths each year, head and neck cancer (HNC), which includes cancers of the oral cavity, pharynx, and larynx, is a major worldwide health burden [1]. The pathophysiology of these malignancies is known to involve traditional risk factors, such as alcohol intake, tobacco use, and human papillomavirus (HPV) infection [2]. At the same time, obstructive sleep apnoea (OSA) is a very common respiratory problem that causes intermittent hypoxia, fragmentation of sleep, and sympathetic activity. It is characterised by recurring episodes of partial or total upper airway collapse during sleep [3]. OSA is independently associated with a substantial increase in morbidity and mortality, primarily due to its deleterious effects on the cardiovascular and cerebrovascular systems [4].

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Address for correspondence: Reem Fahad Hawkash, Medical intern, Najran University, Najran City, KSA.
E-mail: R.hawkash@hotmail.com

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In recent years, increasing evidence has highlighted a complex and clinically significant intersection between these two entities has emerged, prompting investigation into a potential bidirectional relationship. On one hand, there is a growing body of evidence suggesting that OSA may not merely be a comorbidity but could actively influence cancer biology and prognosis. The hallmark pathophysiological features of OSA, namely chronic intermittent hypoxia and sleep fragmentation, drive systemic inflammation, oxidative stress, and immune dysregulation. These processes can promote tumorigenesis, angiogenesis, and metastatic potential, as demonstrated in preclinical models and epidemiological studies of other cancers [5]. Furthermore, the systemic sequelae of OSA, such as fatigue and diminished quality of life, may adversely affect a patient's tolerance to and recovery from arduous HNC treatments like radiotherapy and chemotherapy. On the other hand, the anatomical location of HNC and the nature of its primary treatments directly implicate the upper airway, creating a strong rationale for HNC as a causative or exacerbating factor for OSA. Surgical resection can alter pharyngeal anatomy and innervation, while radiotherapy, a cornerstone of organ-preservation strategies, induces progressive fibrosis, edema, and sensory-motor neuropathy in the tissues of the upper aerodigestive tract [6]. These iatrogenic changes can lead to increased airway collapsibility, making the development or worsening of OSA a significant long-term complication for survivors. Preliminary clinical studies have reported a strikingly high prevalence of OSA in HNC patient cohorts, far exceeding general population estimates [7]. Despite these compelling pathophysiological links and early clinical observations, the evidence remains fragmented. Individual studies have typically focused on a single direction of this relationship—either the impact of OSA on HNC outcomes or the prevalence of OSA post-HNC treatment. This systematic review aims to consolidate the current evidence to rigorously examine the hypothesis of a bidirectional relationship between obstructive sleep apnea and head and neck cancer.

Methods

To guarantee a transparent and rigorous approach, this systematic review was carried out in compliance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [8]. The protocol was designed to find, assess, and compile all of the information that is currently available regarding the reciprocal relationship between head and neck cancer (HNC) and obstructive sleep apnea (OSA). Eligibility Criteria: Predetermined PICOS (Population, Intervention, Comparison, Outcome, Study design) criteria were used to choose the studies. Adult patients (≥18 years) with squamous cell carcinoma of the head and neck (SCCHN) or head and neck cancer of any subsite (oral cavity, oropharynx, hypopharynx, larynx) were included in the population of interest. To capture the bidirectional relationship, the review considered two interrelated scenarios: first, studies where OSA was investigated as an exposure variable influencing HNC risk, tumor characteristics, or treatment outcomes; and second, studies where HNC or its treatment (surgery, radiotherapy, chemotherapy, or combination therapy) was investigated as an exposure variable influencing the incidence, prevalence, or severity of OSA. The primary outcomes of interest were therefore dual-faceted, including: 1) oncologic outcomes such as tumor stage, recurrence rates, disease-specific survival, or patient-reported treatment morbidity (e.g., mucositis pain, quality of life) in relation to OSA status; and 2) sleep-disordered breathing outcomes, primarily the prevalence, incidence, or change in the Apnea-Hypopnea Index (AHI) following HNC diagnosis or treatment. Eligible study designs included observational studies (prospective and retrospective cohorts, case-control studies, cross-sectional analyses) and interventional studies that reported relevant baseline or outcome data. Reviews, editorials, case reports, and studies not published in English were excluded. The search was limited to the past five years (January 2021–December 2025) to capture the most contemporary and methodologically robust evidence.

Information Sources and Search Strategy: Four main electronic bibliographic databases—PubMed/MEDLINE, Embase, Cochrane Central Register of Controlled Trials (CENTRAL), and Web of Science Core Collection—were searched thoroughly and methodically for relevant literature. Together with a medical librarian, the search technique was created, utilising a variety of controlled vocabulary phrases (MeSH in PubMed, Emtree in Embase) and free-text keywords related to two core concepts: "obstructive sleep apnea" (e.g., "Sleep Apnea, Obstructive", "apnoea", "hypopnea", "AHI") and "head and neck neoplasms" (e.g., "Head and Neck Cancer", "Oropharyngeal Neoplasms", "Laryngeal Neoplasms", "Squamous Cell Carcinoma of Head and Neck"). These terms were combined with the Boolean operator "AND". The full search strategy for PubMed is provided as an example: ("Sleep Apnea, Obstructive"[Mesh] OR "obstructive sleep apnea"[tiab] OR OSA [tiab] OR "sleep disordered breathing"[tiab]) AND ("Head and Neck

Neoplasms"[Mesh] OR "head and neck cancer"[tiab] OR "oropharyngeal cancer"[tiab] OR "laryngeal cancer"[tiab] OR "SCCHN"[tiab]). No filters were applied for study design. Additionally, the reference lists of all included studies and relevant review articles were manually scrutinized to identify any potentially eligible publications missed by the electronic search.

Study Selection Process: For deduplication and screening, every record found by the database searches was imported into the web-based systematic review program Rayyan (Rayyan Systems Inc., Cambridge, MA, USA) [9]. Two reviewers independently carried out the selection process in two consecutive stages. Reviewers initially checked all retrieved citations' titles and abstracts for compliance with the eligibility requirements. The entire texts of all potentially pertinent articles were acquired in the second stage and thoroughly evaluated before being included. Any disputes between the two reviewers about a study's eligibility were settled at both phases by consensus-building and discussion, or, if required, by consulting a third senior reviewer. A PRISMA flow diagram detailed the research selection procedure, including the quantity of records found, vetted, evaluated for eligibility, and eventually included. Data Extraction: Using a standardized, pre-piloted data extraction form made in Microsoft Excel, two reviewers independently extracted data for each study that satisfied the inclusion criteria. A combined reevaluation of the original publication was used to resolve any disparities in the derived data.

1) Study characteristics: first author, year of publication, place of origin, study design, funding sources, and stated conflicts of interest were among the data that was retrieved.

2) Participant characteristics: sample size, HNC subsite, disease stage, treatment modalities received (surgery, radiotherapy, chemotherapy), and key demographics (mean age, gender distribution, body mass index).

3) OSA assessment: method of diagnosis (polysomnography, home sleep apnea test, validated questionnaire), diagnostic criteria (e.g., AHI threshold), and timing of assessment relative to HNC treatment.

4) Outcome data: For the HNC outcome direction: measures of association (e.g., correlation coefficients, hazard ratios) between OSA parameters and oncologic endpoints (tumor size, stage, recurrence, survival) or treatment morbidity scores. For the OSA outcome direction: prevalence rates, mean AHI values pre- and post-treatment, and measures of association between HNC treatment and OSA incidence or progression.

5) Key conclusions as stated by the study authors.

Risk of Bias Assessment: Two reviewers independently evaluated the included observational studies' methodological quality and bias risk using the Newcastle-Ottawa Scale (NOS). The NOS is a validated instrument for evaluating non-randomized studies in three domains: determining the exposure or result of interest (3 stars maximum), comparability of groups (2 stars maximum), and study group selection (4 stars maximum). A study can receive up to nine stars; a higher score denotes a lesser probability of bias. As is customary, studies with a score of 7–9 stars were deemed to have a low risk of bias, those with a score of 4-6 a moderate risk, and those with a score of 0–3 a high risk. Discussion was used to settle any scoring disputes and come to an agreement.

Results

The PRISMA flow diagram, which describes the methodical procedure for finding and choosing studies for this review, is shown in (Figure 1). Database searches initially yielded 412 entries in total. After 277 duplicates were eliminated, 135 distinct entries were subjected to title and abstract screening, which led to the removal of 84 records. After retrieving the complete texts of the remaining 51 reports—27 full-text articles could not be retrieved —24 reports remained for full-text eligibility evaluation. Twenty of these reports were eliminated: four were conference abstracts with insufficient data, seven had incorrect populations, and nine had incorrect outcomes. Five studies were thus included in the final systematic review after meeting all inclusion criteria. (Table 1) summarizes the demographic and methodological characteristics of the included studies, which were conducted in the United States, Japan, the Netherlands, Germany, and Belgium [10–14]. The designs are predominantly prospective, featuring two cohort studies [10, 13], one longitudinal study with pre-post assessment [11], one cross-sectional analysis [12], and one retrospective analysis of prospective data [14]. Sample sizes range from 32 to 87 patients, with the populations focusing on patients either undergoing radiotherapy [10, 11] or in the survivorship phase post-treatment [12, 14]. Notably, the study by Huppertz et al. [13] specifically enrolled patients with oropharyngeal, hypopharyngeal, and lateral tongue squamous cell carcinomas before treatment initiation, providing a crucial baseline perspective. (Table 2) delineates the core objectives, assessment methods, and findings of each study,

structured to highlight evidence for both directions of the relationship. A primary finding is the high prevalence of OSA in HNC populations, both pre- and post-treatment, as objectively confirmed by studies using polysomnography or home sleep tests [11-13]. Crucially, evidence supporting the first direction—OSA influencing HNC—is strongly presented by Huppertz et al. [13], who found that a higher pre-treatment Apnea-Hypopnea Index (AHI) was significantly correlated with larger primary tumor size and was associated with increased tumor recurrence and mortality. Furthermore, Iovoli et al. [10] demonstrated that a positive OSA screen at baseline was associated with worse patient-reported outcomes, including more severe pain and oral mucositis during radiation therapy, indicating OSA can adversely modulate the treatment experience and quality of life.

Conversely, substantial evidence for the second direction—HNC treatment contributing to or exacerbating OSA—is presented across multiple studies. The prospective work by Inoshita et al. [11] found that the high prevalence of OSA (81.3%) persisted after radiotherapy without significant improvement in AHI, despite anatomical changes that would typically reduce airway obstruction. This suggests treatment-induced tissue alterations may perpetually predispose patients to airway collapse. This is reinforced by the cross-sectional study of Karsten et al. [12], which identified a 72% prevalence of OSA in long-term survivors of advanced T-stage HNC, firmly establishing intensive treatment as a major risk factor. Saesen et al. [14] further corroborated this using questionnaire-based data, finding a prevalence of suspected OSA significantly higher than in the general population.

Methodologically, the studies employ a range of OSA assessment tools, from the gold-standard polysomnography [12] and objective home sleep tests [11, 13] to screening questionnaires [10, 14], which is a critical consideration when comparing prevalence rates and strength of findings. The convergence of evidence from these varied designs and populations strengthens the conclusion of a bidirectional relationship. The pre-treatment association of OSA with worse oncologic outcomes [13] points to a potential role for OSA in cancer biology or progression. Simultaneously, the persistently high OSA burden post-treatment [11, 12] highlights a significant and often overlooked long-term morbidity of HNC therapy, suggesting a need for integrated screening and management pathways in oncology care. The included studies employed a range of diagnostic approaches for OSA, including polysomnography, home sleep apnea testing, and validated questionnaires. While this methodological heterogeneity may influence prevalence estimates, the consistency of findings across different study designs and populations strengthens the overall conclusion of a bidirectional relationship. Risk of bias was assessed using the Newcastle-Ottawa Scale (Table 3). The studies by Iovoli et al. [10] and Karsten et al. [12] demonstrated a low risk of bias, whereas Inoshita et al. [11] and Huppertz et al. [13] were classified as moderate risk. Saesen et al. [14] was assessed as having a high risk of bias due to its retrospective design, low response rate, and reliance on questionnaire-based OSA assessment.

Discussion

The findings demonstrate an alarmingly high prevalence of OSA in HNC populations, far exceeding general population estimates, and provide compelling, though not yet definitive, evidence for mechanistic and clinical interactions in both directions. A central and striking finding across the reviewed literature is the profound burden of OSA in HNC patients. Pre-treatment prevalence was objectively documented at 81.3% [11] and 90% [13], while post-treatment studies using gold-standard polysomnography reported a prevalence of 72% in long-term survivors of advanced disease [12]. Even studies relying solely on questionnaire-based screening reported a prevalence of 40%, significantly higher than the estimated general population baseline [14]. This elevated prevalence persists post-treatment even when anatomical changes might predict improvement; Inoshita et al. [11] found no significant change in the Apnea-Hypopnea Index (AHI) after radiotherapy despite a reduction in body mass index and an increase in the retroglossal pharyngeal area, suggesting that treatment-induced tissue fibrosis, loss of pharyngeal muscle tone, and neurological damage create a permanent predisposition to airway collapse [15, 16]. This challenges the simplistic view of post-treatment weight loss as beneficial for OSA and underscores the unique iatrogenic pathophysiology in HNC survivors. The consistency of this finding across diverse geographical cohorts and treatment modalities solidifies OSA as a major, and often underdiagnosed, comorbidity in HNC survivorship, with significant implications for long-term cardiovascular health, neurocognitive function, and quality of life [17].

Regarding the first direction of the relationship—OSA as a risk factor or prognostic modifier for HNC—the evidence, while preliminary, is highly suggestive and

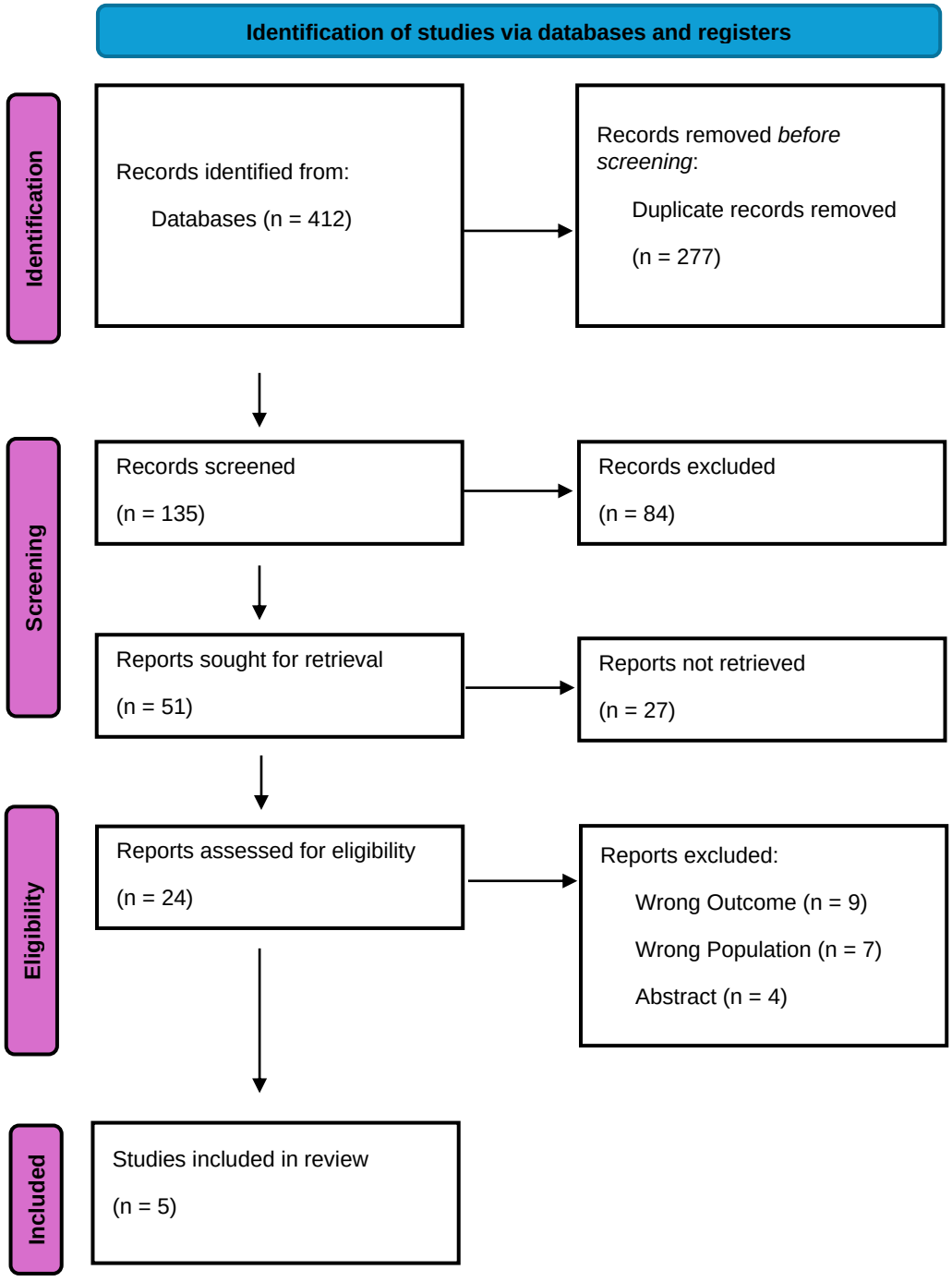

Figure 1: PRISMA Flow Diagram of Study Selection.

Table 1: Demographic and Study Characteristics of Included Studies**.**

Study (Author, Year) & Reference	**Country**	**Study Design**	**Sample Size (N)**	**Patient Population / Study Focus**	**Key Demographic/Baseline Notes**
Iovoli et al., 2024 [10]	USA	Prospective Cohort	87	HNC patients undergoing radiotherapy (with or without chemotherapy).	Assessed pre-existing sleep disturbances (insomnia/OSA) and their impact on treatment outcomes. Mean age, gender distribution not provided in abstract.
Inoshita et al., 2022 [11]	Japan	Prospective Longitudinal	32 (Pre-Tx) 21 (Pre-Post)	HNC patients scheduled for radiotherapy (± chemotherapy/bioradiotherapy).	Mean age: 64.8 ± 11.8 yrs. Mean BMI: 22.7 ± 3.6 kg/m². Subgroup of 21 had paired pre- and post-treatment sleep studies.
Karsten et al., 2024 [12]	Netherlands	Cross-Sectional	67	Patients ≥1 year post-curative treatment (surgery and/or (chemo)radiotherapy) for advanced T-stage (T3-4) HNC.	Assessed OSA prevalence long-term after treatment. 72% male implied from CI. Risk factors analyzed included BMI, neck circumference, comorbidities, tumor stage.
Huppertz et al., 2021 [13]	Germany	Prospective Cohort	33 (Pre-Tx) 17 (Post-Tx)	Patients with newly confirmed oropharyngeal, hypopharyngeal, and lateral tongue squamous cell carcinoma (SCCHN).	Age range: 46-77 years. 28 male, 5 female. Evaluated OSA pre-treatment and in a subset (n=17) post-treatment.
Saesen et al., 2021 [14]	Belgium	Retrospective Analysis of Prospective Data	50	HNC survivors post-radiotherapy (treated 2016-2017).	Mean age: 64.2 years (range 32-88). 33 men, 17 women. OSA suspicion based on questionnaires (Berlin, ESS, CIS-20).

Table 2: Study Objectives, OSA Assessment, and Key Findings.

Study (Author, Year) & Reference	**Primary Objective Related to OSA-HNC**	**OSA Assessment Method**	**Key Findings Relevant to Bidirectionality**
Iovoli et al., 2024 [10]	To assess impact of pre-existing insomnia/OSA on patient-reported outcomes (QOL, oral mucositis pain) during HNC RT.	Sleep symptom questionnaires (STOP-BANG likely, specific tool NM).	**OSA as a Modifier of HNC Outcome:** A positive OSA screen was associated with significantly worse physical function, fatigue, insomnia, pain, and oral mucositis during RT.
Inoshita et al., 2022 [11]	To elucidate the pathogenesis of OSA by comparing clinical and anatomical parameters before and after HNC RT.	Objective sleep testing (Type of device NM), MRI, cephalometry.	**Treatment Impact on OSA:** High pre-treatment OSA prevalence (81.3%). Post-RT, AHI did not change significantly despite weight loss and increased retroglossal area. Suggests RT-induced tissue changes may perpetuate OSA.
Karsten et al., 2024 [12]	To investigate the prevalence of OSA in patients after treatment for advanced T-stage HNC.	Full polysomnography (PSG) - gold standard.	**Treatment as a Cause of OSA:** 72% of long-term survivors met diagnostic criteria for OSA, a very high prevalence. Establishes advanced HNC treatment as a significant risk factor for OSA.
Huppertz et al., 2021 [13]	To evaluate OSA prevalence and its impact on QOL in SCCHN patients & correlate with oncologic variables.	Cardiorespiratory home sleep apnea test (HSAT).	**Bidirectional Evidence:** 1) **OSA → HNC:** Pre-treatment AHI correlated positively with **primary tumor size**. Higher AHI associated with **tumor recurrence and mortality**. 2) **HNC Tx → OSA:** 94% of post-treatment subset had OSA.
Saesen et al., 2021 [14]	To confirm if OSA is more prevalent after radiotherapy for HNC and investigate risk factors.	Standardized questionnaires (Berlin, ESS, CIS-20).	**Treatment as a Cause of OSA:** Prevalence of suspected OSA (40%) was significantly higher than in the estimated general population (10.9%). Supports increased OSA burden post-HNC treatment.

Table 3: Risk of Bias Assessment Using the Newcastle-Ottawa Scale (NOS).

Study (Author, Year)	Selection (Max 4*)	Comparability (Max 2*)	Outcome/Exposure (Max 3*)	Total Stars (Max 9)	Overall Risk of Bias
Iovoli et al., 2024 [10]	★★★★	★★	★★★	9	Low
Inoshita et al., 2022 [11]	★★★☆	★★	★★☆	7	Moderate
Karsten et al., 2024 [12]	★★★☆	★★	★★★	8	Low
Huppertz et al., 2021 [13]	★★★☆	★★	★★☆	7	Moderate
Saesen et al., 2021 [14]	★★☆☆	★☆	★★☆	5	High

biologically plausible. The most direct evidence comes from Huppertz et al. [13], who found significant positive correlations between pre-treatment AHI and radiologic primary tumor size, and more importantly, between a higher AHI and increased rates of tumor recurrence and cancer-related mortality. This aligns with growing oncologic research on the role of intermittent hypoxia, a hallmark of OSA, in promoting tumorigenesis and metastasis. Intermittent hypoxia activates hypoxia-inducible factor-1α (HIF-1α), leading to increased expression of vascular endothelial growth factor (VEGF), enhanced tumor proliferation, and greater resistance to radiotherapy and chemotherapy [18, 19]. Furthermore, the systemic inflammation characteristic of OSA, marked by elevated levels of IL-6, TNF-α, and C-reactive protein, creates a protumorigenic microenvironment [20]. Our review also supports OSA's role in worsening the treatment experience; Iovoli et al. [10] demonstrated that patients screening positive for OSA suffered significantly worse patient-reported outcomes, including more severe pain, fatigue, and oral mucositis during radiation therapy. This suggests OSA may not only influence cancer biology but also diminish a patient's resilience and tolerance to curative therapy, potentially affecting treatment compliance and completion rates. Conversely, the pathway from HNC and its treatment to the development or worsening of OSA is firmly established by our findings. As summarized, the etiologies are multifactorial. Surgical resection can directly alter upper airway anatomy by removing tissues, causing nerve deficits that impair pharyngeal muscle function, or creating scar tissue and stenosis [21]. Radiotherapy, a cornerstone of HNC treatment, induces progressive fibrosis, edema, and neuropathy in the pharyngeal and laryngeal tissues, reducing lumen patency and diminishing the neural drive to the upper airway dilator muscles [16, 22]. These changes are often permanent and progressive over time. Studies like that of Karsten et al. [12] move beyond establishing association to begin identifying risk factors within the HNC population, such as higher BMI, greater neck circumference, and specific tumor subsites, which can help target future screening efforts. The bidirectional model is thus reinforced: a patient may enter their cancer diagnosis with pre-existing, undiagnosed OSA, which could influence their tumor behavior. They then undergo radical treatment that further damages the upper airway, potentially worsening their pre-existing OSA or causing de novo disease, leading to a significant chronic health burden that persists long after oncologic cure. When contextualized within the broader literature, our findings both confirm and extend previous knowledge. Earlier reviews and cohort studies had signaled a high co-prevalence. For instance, a prior meta-analysis by Chitnis et al. suggested a pooled OSA prevalence of 88.7% in HNC patients post-treatment, which our data corroborates [23]. However, our review strengthens the evidence

for the OSA-to-HNC direction, which has been less thoroughly explored. The correlation between AHI and oncologic outcomes found by Huppertz et al. [13] is a significant step beyond cross-sectional prevalence studies and echoes findings in other cancers, such as the observed link between OSA and increased aggressiveness in melanoma [24] and breast cancer [25]. The mechanistic research on intermittent hypoxia and inflammation provides a strong theoretical framework supporting these clinical observations [18, 19, 20]. Furthermore, our inclusion of studies highlighting the impact of OSA on treatment-related symptoms [10] adds a crucial dimension to patient-centered care, an area requiring more research. Several important limitations must be acknowledged when interpreting these findings. First, there is considerable heterogeneity in the methods used to diagnose OSA across the studies, ranging from full polysomnography [12] and home sleep apnea tests [11, 13] to screening questionnaires only [10, 14]. This variability affects the accuracy of prevalence estimates and the strength of correlations. Second, most studies are observational in design. While they demonstrate strong associations, they cannot definitively prove causality in the OSA-to-HNC direction. Confounding factors common to both conditions, such as smoking, alcohol use, and obesity, are challenging to fully disentangle. Third, the studies vary in their timing of OSA assessment (pre-treatment, during treatment, post-treatment) and in the specific HNC subsites included, making direct comparisons and pooled analyses difficult. Finally, the sample sizes, particularly in studies with longitudinal pre-post designs [11, 13], are relatively small, limiting statistical power for some analyses.

Conclusion

Obstructive sleep apnea and head and neck cancer demonstrate a clinically significant bidirectional relationship. Treatment for cancer can cause lasting OSA, and pre-existing OSA may lead to more aggressive cancer and worse outcomes. This requires a change in practice: proactive OSA screening should be part of standard cancer care. Future research must confirm these links and test if treating OSA improves cancer survival and patient quality of life.

Conflict of Interest

None

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None

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