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Obstructive Sleep Apnea and Cancer: A Comprehensive Review

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ABSTRACT

Obstructive sleep apnoea (OSA) is a very common sleep disorder that causes the upper airway to collapse repeatedly during sleep. This causes intermittent hypoxia, sleep fragmentation, and systemic inflammation. In the last ten years, more and more evidence has pointed to possible links between OSA and the development, progression, and outcomes of cancer. This review rigorously evaluates the existing knowledge concerning the association between OSA and cancer, investigating biological mechanisms, epidemiological data, and clinical ramifications. The pathophysiological mechanisms that may connect obstructive sleep apnoea (OSA) to carcinogenesis encompass intermittent hypoxia-induced modifications in the tumour microenvironment, oxidative stress, systemic inflammation, immune dysregulation, and metabolic dysfunction. Epidemiological studies have produced varied results; however, several extensive cohort studies have established correlations between the severity of obstructive sleep apnoea (OSA) and elevated cancer incidence, aggressiveness, and mortality, especially for specific cancer types. This review also talks about what these findings mean for clinical practice, including how treating OSA might affect cancer outcomes and what future research should focus on. Comprehending the intricate relationship between OSA and cancer may enhance screening methodologies, therapeutic approaches, and clinical outcomes in impacted patients.

Keywords: *Obstructive Sleep Apnea, Cancer Pathogenesis, Intermittent Hypoxia, Tumour, Microenvironment*

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1. INTRODUCTION

Obstructive sleep apnoea (OSA) is marked by repeated instances of partial or total upper airway collapse during sleep, leading to intermittent hypoxia, hypercapnia, fluctuations in intrathoracic pressure, sleep fragmentation, and increased sympathetic activity. Obstructive sleep apnoea (OSA) affects 9% to 38% of adults, making it a major public health issue, especially since it is linked to problems with the heart, metabolism, and brain. In recent years, an increasing corpus of evidence has indicated a multifaceted relationship between obstructive sleep apnoea (OSA) and cancer. Because both conditions are a big problem around the world, this relationship needs to be looked into more. Cancer is still the second leading cause of death around the world. In 2020, there were about 19.3 million new cases and 10 million cancer deaths. If confirmed, the potential role of OSA in cancer incidence, progression and treatment outcome could have important implications for public health policy and clinical management.

This association is biologically plausible because several pathophysiological features of OSA may plausibly influence cancer initiation and progression. Hypoxia, one of the most notable features of obstructive sleep apnoea (OSA), is an important driver of hypoxia-inducible factor-1 (HIF-1), which promotes tumour neovascularization, metabolic adaptation and its metastatic potential3,4. In addition, OSA has been associated with systemic inflammation, oxidative stress, and alteration of immune function that may be involved in cancer initiation and progression. Objective To perform a

comprehensive review of available evidence on the relationship between obstructive sleep apnoea (OSA) and cancer. It focuses in particular on assessing the biological pathways that may mediate associations between OSA and carcinogenesis, provides a critical review of epidemiological studies of OSA and cancer incidence, progression, and mortality. It also reviews implications for clinical practice, including potential effects of OSA Understanding the complexity of the association between OSA and cancer may advance screening methods, risk assessment, and treatment, ultimately optimizing outcomes for patients affected by each of these conditions.

2. BIOLOGICAL MECHANISMS LINKING OSA AND CANCER

2.1 Intermittent Hypoxia and HIF-1 Activation

The distinct intermittent hypoxia in OSA is caused by recurrent cycles of hypoxia and reoxygenation, which is unique and different from chronic hypoxia. They activate a pathway that activates hypoxia-inducible factor-1 (HIF-1), a master regulator of cellular responses to hypoxia. HIF-1 induces many genes critical for angiogenesis, glucose metabolism, cell growth, and survival. These processes contribute significantly to the development and metastasis of tumours. Mouse studies found that intermittent hypoxia promotes the growth and spread of tumours through HIF-1-dependent pathways. Almendros et al. Intermittent hypoxia accelerated melanoma growth and increased lung metastasis in mouse models, effects that were inhibited by HIF-1 blockade (Almendros et al. 2012). Martínez-García et al. Notes (2016) also found that intermittent hypoxia increased the malignancy of lung cancer cells by promoting proliferation, migration, and anti-apoptosis. A key point is that the indirect effects of a TME on the disease may have a greater impact than the hypoxic TME itself on tumour biology due to the potential for intermittent hypoxia to have a greater impact than chronic hypoxia. Though chronic hypoxia ultimately leads to adaptation or cell death, hypoxia, as a condition, is a permanent state in OSA where the chronic maladaptive responses may facilitate tumour growth.

2.2 Oxidative Stress and DNA Damage

The quick reoxygenation that happens after hypoxic episodes in OSA makes reactive oxygen species (ROS), which causes oxidative stress. High levels of ROS can cause DNA damage, genomic instability, and mutations, which are all known ways that cancer can start. Oxidative stress can also turn on redox-sensitive transcription factors, such as NF-κB, which control genes that are important for cell survival, growth, and inflammation. Clinical studies have consistently shown elevated oxidative stress markers in individuals with OSA. Lavie et al. (2004) reported elevated levels of lipid peroxidation products and reduced antioxidant capacity in OSA patients compared to controls. These alterations were positively correlated with OSA severity and partially reversed with continuous positive airway pressure (CPAP) treatment. The persistent oxidative burden in untreated OSA may have cumulative effects on DNA integrity, potentially increasing cancer risk over time. Indeed, several studies have documented increased DNA damage in peripheral blood lymphocytes of OSA patients, with the extent of damage correlating with disease severity.

2.3 Systemic Inflammation and Immune Dysregulation

OSA induces systemic inflammation through multiple mechanisms, including intermittent hypoxia, sleep fragmentation, and sympathetic activation. Elevated levels of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and C-reactive protein (CRP), have been consistently reported in OSA patients. Chronic inflammation is a well-established contributor to carcinogenesis, influencing tumour initiation, promotion, and progression. Inflammatory cytokines can activate signal transducer and activator of transcription 3 (STAT3) and NF-κB signaling pathways, promoting cell proliferation, survival, and angiogenesis while inhibiting apoptosis. Furthermore, OSA is associated with alterations in immune function, including reduced natural killer (NK) cell activity and T-cell dysregulation. Impaired immune surveillance may allow malignant cells to escape detection and elimination, facilitating cancer development and progression. Crucially, these immunological alterations appear to be related to OSA severity and may improve with effective treatment.

Table 1: Inflammatory Markers Associated with OSA and Their Relevance to Cancer

Inflammatory Marker	Elevation in OSA	Cancer-Related Effects	Reference
Interleukin-6 (IL-6)	Significant increase correlated with AHI	Promotes tumor growth, angiogenesis, and metastasis	Arnardottir et al., 2009
Tumor Necrosis Factor-α (TNF-α)	2–3 fold increase in severe OSA	Activates NF-κB pathway; promotes cell survival	Lavie et al., 2004
C-Reactive Protein (CRP)	Linear relationship with OSA severity	Associated with increased cancer risk and poor prognosis	Gozal et al., 2016

Nuclear Factor-κB (NF-κB)	Activated by intermittent hypoxia	Master regulator of inflammatory response and cell survival	Grivennikov et al., 2010
Hypoxia-Inducible Factor-1α (HIF-1α)	Stabilized by intermittent hypoxia	Drives angiogenesis and metabolic adaptation in tumors	Semenza, 2012

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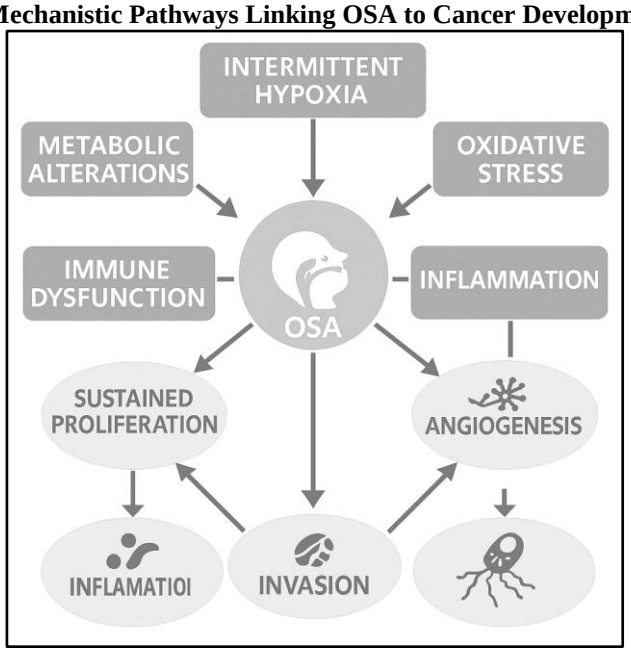
2.4 Metabolic Dysfunction

OSA is strongly associated with metabolic disorders, including obesity, insulin resistance, and type 2 diabetes—conditions that independently increase cancer risk. The metabolic consequences of OSA, including adipose tissue dysfunction and altered adipokine production, may create a microenvironment favourable for cancer development. Additionally, intermittent hypoxia induces metabolic reprogramming in cells, favouring glycolysis over oxidative phosphorylation even under normoxic conditions—a phenomenon known as the Warburg effect, which is characteristic of many cancer cells. This metabolic shift supports rapid cell proliferation and may confer a survival advantage to developing tumour cells.

2.5 Circadian Disruption and Sleep Fragmentation

Sleep fragmentation, a hallmark of OSA, disrupts circadian rhythms and alters the expression of clock genes that regulate cell cycle, DNA repair, and metabolism. Circadian disruption has been associated with increased cancer risk in epidemiological studies, particularly in shift workers. Experimental studies have demonstrated that sleep fragmentation, independent of intermittent hypoxia, can accelerate tumour growth in animal models. Hakim et al. (2014) showed that sleep fragmentation enhanced tumour progression and invasiveness in mice with breast cancer, effects that were mediated through macrophage recruitment and alterations in the tumour microenvironment.

Figure 1: Potential Mechanistic Pathways Linking OSA to Cancer Development and Progression



3. EPIDEMIOLOGICAL EVIDENCE

3.1 OSA and Over all Cancer Incidence

Several large-scale epidemiological studies have investigated the association between OSA and cancer incidence, yielding somewhat heterogeneous results. In a landmark study, Campos-Rodriguez et al. (2013) analysed data from 4,910 patients with suspected OSA and found that severe OSA (apnea-hypopnea index [AHI] ≥30) was independently associated with increased cancer incidence (adjusted hazard ratio [HR] 1.65, 95% confidence interval [CI] 1.06-2.57) compared to patients without OSA. Notably, this association was stronger in younger patients and those with adequate CPAP adherence. Similarly, a population-based cohort study by Marshall et al. (2014) followed 400 participants for 20 years and found that moderate-to-severe OSA was associated with a 2.5-fold increase in cancer mortality after adjusting for potential confounders. Importantly, this association was independent of obesity, smoking, and other established cancer risk factors Some other studies, on the other hand, have found weaker or no connections. The Wisconsin Sleep Cohort Study followed 1,522 people for 22 years and found a positive but not significant link between the severity of OSA and the risk of cancer (HR 1.48, 95% CI 0.93-2.37) for people with severe OSA compared to people with no OSA. A recent meta-analysis of 19

studies revealed a pooled relative risk of 1.40 (95% CI 1.01-1.95) for cancer incidence in patients with OSA, exhibiting considerable heterogeneity among the studies. The inconsistency of results may be due to differences in OSA measurement, cancer outcomes, follow-up time, and adjustment for potential confounding factors. The association between OSA and cancer may not be homogenous by cancer type, thus site-specific analyses are warranted.

Table 2: Major Epidemiological Studies Investigating the Association Between OSA and Cancer

Study	Design	Sample Size	Follow-up	Main Findings	Limitations
Campos-Rodriguez et al., 2013	Multicenter cohort	4,910	4.5 years	Severe OSA linked to increased cancer incidence (HR 1.65, 95% CI 1.06–2.57)	Diagnostic bias; limited follow-up
Marshall et al., 2014	Population-based cohort	400	20 years	Moderate–severe OSA associated with increased cancer mortality (HR 2.5)	Small sample size; limited OSA assessment
Nieto et al., 2012	Prospective cohort	1,522	22 years	No significant association with cancer mortality (HR 1.48, 95% CI 0.93–2.37)	Limited power for cancer subtypes
Chen et al., 2014	Case-control	1,236 cases, 4,944 controls	N/A	OSA linked to increased colorectal cancer risk (OR 1.8, 95% CI 1.1–2.9)	Potential recall bias; limited OSA validation
Zhang et al., 2020	Meta-analysis	19 studies	Variable	Pooled RR for cancer in OSA patients: 1.40 (95% CI 1.01–1.95)	Significant heterogeneity across studies

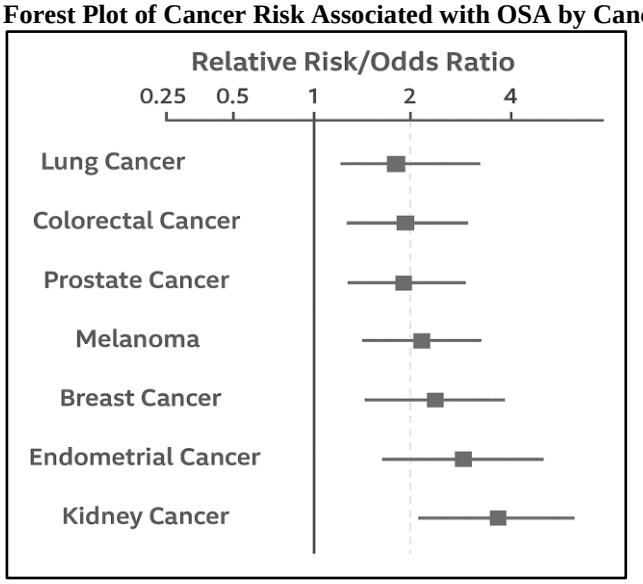
3.2 OSA and Specific Cancer Types

Evidence suggests that the OSA–cancer link may be even stronger for certain cancer types, particularly those that are hypoxia-sensitive or driven by inflammation. Several studies have found associations between obstructive sleep apnoea (OSA) and colorectal cancer (CRC). Chen et al in a case-control study for example, after adjustment for known risk factors, obstructive Sleep apnoea (OSA) was associated with a 1.8-fold increased risk of CRC (14). Biologically, this finding is plausible as both hypoxia and inflammation play an integral role in colorectal carcinogenesis. Also, OSA may influence the evolution and outcome of CRC, too. Martínez-García et al. Previous studies showed for instance that the severity of obstructive sleep apnoea (OSA) was associated with reduced progression-free and overall survival in patients with colorectal cancer (CRC), and those with cutaneous melanoma respectively.

Many studies have explored the relationship between OSA and breast cancer, but results have been inconsistent. Chang et al. Their study showed that women with OSA had a higher risk of developing BC (adjusted HR 2.09, 95%CI 1.06–4.12) but this risk was more prominent in younger women. On the other hand, Vogtmann et al. (2013) did not find a significant link in a group of postmenopausal women. Recent evidence indicates that OSA may affect the aggressiveness of breast cancer and the response to treatment. Martínez-García et al. (2016) showed that women with breast cancer who had OSA had higher levels of tumour proliferation markers and a lower response to neoadjuvant chemotherapy.

Because OSA directly exposes the lungs to intermittent hypoxia, it is biologically plausible that there is a link between OSA and lung cancer. A population-based study conducted by Brenner et al. (2015) identified a modest elevation in lung cancer risk associated with obstructive sleep apnoea (adjusted HR 1.47, 95% CI 1.05-2.06), even after accounting for smoking history. Experimental studies further support this notion whereby intermittent hypoxia in rodents was shown to promote lung tumour growth and metastasis, suggesting a direct influence of lung cancer biology by IH. There have been fewer studies showing a relationship between OSA and other cancers such as pancreatic, renal and melanoma cancers, but there is a need to study these associations more closely (12). Such tumour types are characterized by hypoxic tumour microenvironments and may be more sensitive to disruption of the homeostatic effects of intermittent hypoxia in obstructive sleep apnoea (OSA).

Figure 2: Forest Plot of Cancer Risk Associated with OSA by Cancer Type



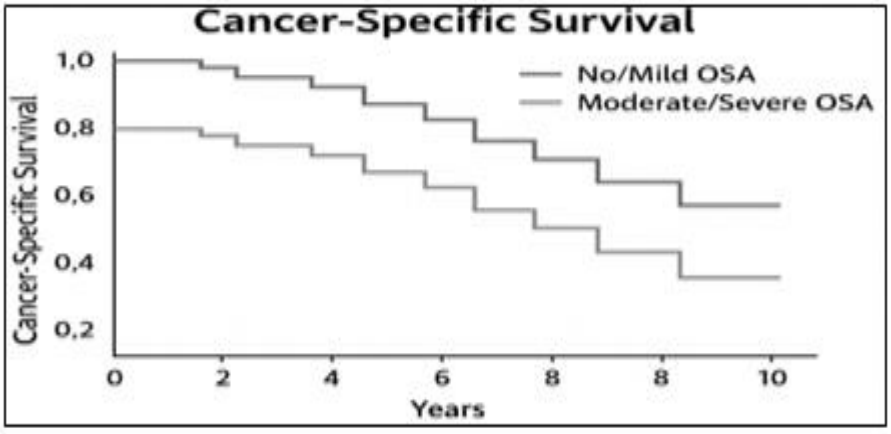
3.3 OSA and Cancer Progression and Mortality

Beyond impacting cancer incidence, OSA may influence cancer progression, treatment response, and mortality. Martínez-García et al. Their cohort study (2014) as displayed in table 4 examines large multicenter clinical data showing a significant increase cancer mortality in those with more severe OSA adjusted HR 4.8, 95% CI 1.4-16.3 (comparing severe to mild-moderate OSA). A retrospective study of 112 patients with cutaneous melanoma by Martínez-García et al. (2014) indicated that OSA correlated with accelerated tumour growth, elevated Breslow index, and diminished disease-free survival, implying an influence on melanoma aggressiveness. Importantly, these associations appear to be independent of established prognostic factors and may have implications for cancer treatment decisions and outcomes prediction.

****Table 3: Impact of OSA on Cancer Progression Markers and Clinical Outcomes**

Cancer Type	Progression Markers	Clinical Outcomes	Study Reference
Melanoma	Increased Breslow thickness, mitotic index	Reduced disease-free survival	Martínez-García et al., 2016
Breast Cancer	Elevated Ki-67, tumor size	Decreased response to neoadjuvant therapy	Martínez-García et al., 2016
Colorectal Cancer	Advanced stage at diagnosis	Reduced progression-free survival	Martínez-García et al., 2014
Lung Cancer	Enhanced metastatic potential	Increased mortality	Almendros et al., 2013
Multiple Cancer Types	N/A	4.8-fold increased cancer mortality with severe OSA	Martínez-García et al., 2014

Figure 3: Kaplan-Meier Curves for Cancer Survival Based on OSA Severity



4. CLINICAL IMPLICATIONS

4.1 Screening and Risk Stratification

The potential association between OSA and cancer raises important questions about screening strategies. While current evidence does not support universal screening, certain high-risk groups may benefit from targeted approaches. For instance, patients with cancer types strongly associated with OSA (e.g., colorectal, lung) and concurrent OSA risk factors (obesity, male gender, advanced age) might warrant OSA screening. Similarly, patients with severe OSA, particularly those with significant nocturnal hypoxemia, might benefit from age-appropriate cancer screening. Risk stratification tools incorporating OSA parameters (e.g., AHI, oxygen desaturation index, time spent below 90% oxygen saturation) alongside established cancer risk factors could help identify individuals who might benefit most from enhanced surveillance or preventive interventions.

Table 4: Proposed Risk Stratification for Cancer Screening in OSA Patients

Risk Category	OSA Severity	Hypoxemia Burden	Other Risk Factors	Recommended Screening
Low	Mild (AHI 5–15)	Minimal (ODI <5, T90 <1%)	No additional risk factors	Age-appropriate standard screening
Moderate	Moderate (AHI 15–30)	Moderate (ODI 5–30, T90 1–10%)	One additional risk factor	Consider earlier initiation of standard screening
High	Severe (AHI >30)	Severe (ODI >30, T90 >10%)	Multiple risk factors	Enhanced surveillance; consider additional modalities
Very High	Severe with treatment resistance	Persistent severe hypoxemia despite treatment	Cancer history or strong family history	Comprehensive surveillance program with shorter intervals

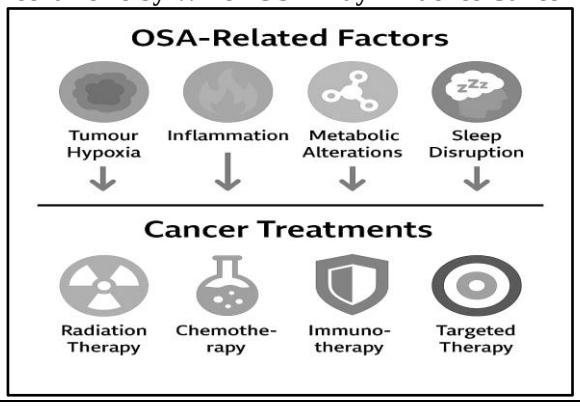
4.2 Impact of OSA Treatment on Cancer Outcomes

A crucial clinical question is whether treating OSA influences cancer outcomes. Limited evidence suggests potential benefits of CPAP therapy on cancer biology and clinical outcomes. Almendros et al. (2013) demonstrated that preventing intermittent hypoxia through supplemental oxygen reduced tumour growth and metastasis in mouse models of OSA, suggesting that normalizing oxygenation might mitigate the pro-tumourigenic effects of OSA. Additionally, several observational studies have reported associations between CPAP adherence and improved cancer outcomes, although these findings require confirmation in randomized controlled trials. Interestingly, CPAP therapy has been shown to reduce oxidative stress, systemic inflammation, and immune dysfunction in OSA patients—mechanisms implicated in carcinogenesis. Whether these improvements translate to reduced cancer risk or improved outcomes in patients with established cancer remains to be determined.

4.3 Implications for Cancer Treatment

OSA may influence cancer treatment efficacy and tolerability through multiple mechanisms. Hypoxia is a well-established factor in radiation and chemotherapy resistance, and the intermittent hypoxia characteristic of OSA might reduce treatment efficacy. Additionally, the sleep disruption and fatigue associated with OSA could impair patients' ability to tolerate aggressive treatment regimens. Preliminary evidence suggests that addressing OSA in cancer patients might improve treatment outcomes. In a small study of patients with head and neck cancer undergoing radiation therapy, CPAP treatment was associated with improved locoregional control and progression-free survival. However, larger studies are needed to confirm these findings and determine whether similar benefits extend to other cancer types and treatment modalities.

Figure 4: Proposed Mechanisms by Which OSA May Influence Cancer Treatment Response



4.4 Management Considerations in Patients with Comorbid OSA and Cancer

Managing patients with comorbid OSA and cancer presents several challenges. Cancer treatments, particularly those affecting the upper airway (e.g., surgery, radiation for head and neck cancers), may exacerbate OSA. Conversely, CPAP therapy might be more challenging in patients experiencing cancer-related symptoms or treatment side effects. Multidisciplinary care involving sleep specialists, oncologists, and supportive care providers is essential for optimizing outcomes in these complex patients. Treatment decisions should consider the potential interactions between OSA and cancer treatments, and CPAP adherence strategies may need to be tailored to accommodate the unique challenges faced by cancer patients.

Table 5: Management Considerations for Patients with Comorbid OSA and Cancer

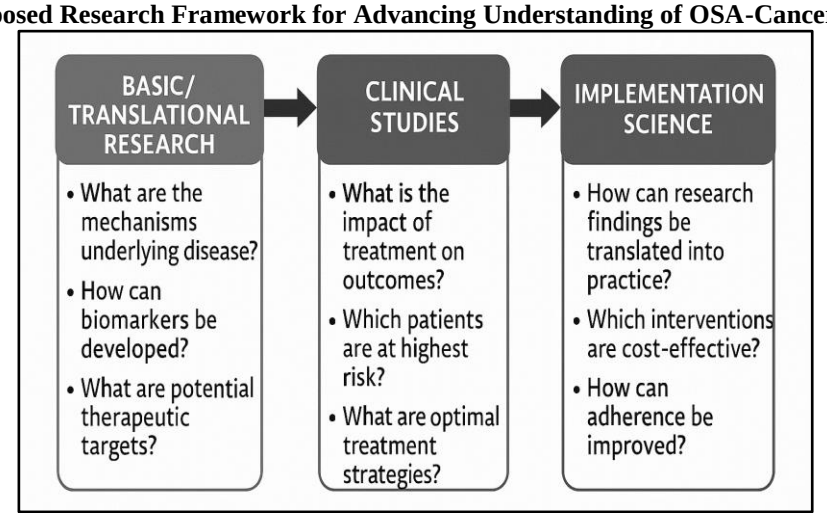
Clinical Scenario	Challenges	Management Strategies
Newly diagnosed cancer in patient with OSA	Treatment planning must consider OSA severity; potential for worsening OSA	Optimize OSA treatment before cancer therapy; consider OSA impact when selecting regimens
Cancer therapy affecting upper airway	Risk of worsening OSA; potential CPAP intolerance	Regular reassessment of OSA severity; consider alternative PAP modalities or oral devices
Fatigue during cancer treatment	Difficult to distinguish treatment-related vs. OSA-related fatigue	Objective sleep assessment; targeted interventions; energy conservation strategies
Palliative care setting	Symptom burden may limit OSA treatment adherence	Focus on comfort and symptom relief; consider nocturnal oxygen if CPAP not tolerated
Cancer survivorship	Long-term effects of cancer therapy may impact OSA	Regular follow-up for both conditions; lifestyle interventions for shared risk factors

5. FUTURE DIRECTIONS

5.1 Research Priorities

Despite growing evidence supporting an association between OSA and cancer, several knowledge gaps remain. Future research priorities should include large-scale prospective studies with standardized OSA assessment, comprehensive cancer outcomes, and adequate adjustment for confounders to clarify the magnitude and consistency of associations across cancer types. Mechanistic studies investigating the molecular pathways linking OSA to carcinogenesis and tumour progression, particularly the differential effects of intermittent versus chronic hypoxia, are also needed. Intervention studies evaluating the impact of OSA treatment on cancer incidence, progression, and treatment response, ideally through randomized controlled trials, would provide valuable insights into the clinical significance of these associations. Additionally, biomarker development to identify OSA patients at highest risk for cancer development or progression, potentially through integration of sleep parameters, circulating markers, and genetic factors, could enhance risk stratification. Finally, health services research to optimize screening strategies, risk stratification tools, and integrated care models for patients with comorbid OSA and cancer would translate research findings into clinical practice.

Figure 5: Proposed Research Framework for Advancing Understanding of OSA-Cancer Relationships



5.2 Emerging Concepts and Technologies

Several emerging concepts and technologies may advance our understanding of the OSA-cancer relationship. Precision sleep medicine approaches that consider individual susceptibility to the carcinogenic effects of OSA based on genetic, epigenetic, and phenotypic factors could help identify high-risk individuals. Novel OSA treatment modalities beyond CPAP, including pharmacological approaches targeting specific pathways (e.g., HIF-1 inhibitors) that may have dual benefits for OSA and cancer, represent an exciting area of investigation. Advanced imaging techniques to assess tumour hypoxia and its relationship to OSA severity and treatment response could provide insights into the biological mechanisms underlying these associations. Integration of wearable technology and home sleep testing to enable longitudinal assessment of sleep parameters and their relationship to cancer outcomes in large populations would facilitate more comprehensive epidemiological studies. Application of systems biology approaches to understand the complex interactions between OSA, cancer, and common comorbidities could potentially identify novel therapeutic targets.

6. CONCLUSION

The relationship between OSA and cancer represents an emerging area of clinical and scientific interest with potentially significant implications for public health and patient care. Biological mechanisms linking OSA to carcinogenesis are biologically plausible and supported by experimental evidence, while epidemiological studies suggest associations between OSA and cancer incidence, progression, and mortality that may be particularly relevant for specific cancer types. While current evidence does not support universal screening or changes in clinical practice, awareness of these associations may inform individualized risk assessment and management decisions. Treating OSA may have benefits beyond reducing cardiovascular risk, potentially influencing cancer outcomes through improvements in tissue oxygenation, inflammation, and immune function. Future research should focus on clarifying the magnitude and specificity of OSA-cancer associations, elucidating underlying mechanisms, and evaluating the impact of OSA treatment on cancer outcomes. A multidisciplinary approach involving sleep medicine specialists, oncologists, basic scientists, and epidemiologists will be essential for advancing knowledge in this complex field. As our understanding of the OSA-cancer relationship evolves, so too will opportunities to leverage this knowledge for improved cancer prevention, early detection, and treatment strategies, ultimately enhancing outcomes for the many patients affected by these common and consequential conditions.

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