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**Review**

# The Interplay Between Obstructive Sleep Apnea and Respiratory Infections: A Review Article

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## ABSTRACT

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by recurrent upper airway collapse, intermittent hypoxia, and sleep fragmentation. Increasing evidence suggests a bidirectional association between OSA and lower respiratory tract infections (LRTIs), including pneumonia, bronchitis, and exacerbations of chronic lung disease. Multiple mechanisms may underlie this relationship. Intermittent hypoxia and sleep disruption promote systemic inflammation and immune dysregulation. Impaired mucociliary clearance, microaspiration, alterations in airway microbiota, together with obesity and related comorbidities, further contribute to increased susceptibility to and severity of infections. Observational studies demonstrate that individuals with untreated OSA have higher rates of pneumonia, more severe infections, and delayed recovery from infections compared with non-OSA populations. These risks are particularly evident among older adults and patients with cardiopulmonary comorbidities. Continuous positive airway pressure (CPAP) therapy may mitigate infection risk by maintaining airway patency, reducing hypoxemia, and improving mucociliary clearance. However, concerns remain regarding CPAP device-associated microbial colonization, highlighting the importance of strict hygiene practices and equipment maintenance. OSA appears to be an underrecognized risk factor for LRTIs; this association is driven by overlapping pathophysiological mechanisms and is supported by emerging epidemiological data. Recognizing this interplay may guide infection prevention strategies and improve clinical outcomes in high-risk populations.

**KEYWORDS:** Obstructive sleep apnea, lower respiratory tract infections, immune dysregulation, CPAP therapy, airway microbiota, COVID-19, community-acquired pneumonia

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## INTRODUCTION

Obstructive sleep apnea (OSA) is the most common sleep-breathing disorder, characterized by collapse of the soft tissues of the upper airway during sleep, causing recurrent intermittent hypoxia and hypercapnia, and resulting in frequent nocturnal awakenings and sleep fragmentation.[1] Those alterations induce sympathetic activation, oxidative stress, and metabolic dysregulation,[2] which lead to various cardiovascular diseases and pulmonary, neurological, and cognitive dysfunctions.[3,4] The exact prevalence of OSA is underestimated; available data indicate that about 20% of middle-aged Americans have OSA, and a higher prevalence has been observed among Asian populations.[5,6] The incidence is higher in males aged 50–70 years (17%) than in females diagnosed with moderate-to-severe OSA (9%).[7]

Lower respiratory tract infections (LRTIs) are a major public health problem associated with increased morbidity and mortality. LRTIs include bronchitis, bacterial and viral pneumonia.[8] The host immune response and the virulence of the susceptible organism determine the outcomes of respiratory infections.[9] The proinflammatory state induced by OSA[10] has highlighted the coexistence of OSA and respiratory infections through enhancement of proinflammatory stimuli.[11] The coronavirus disease 2019 (COVID-19) pandemic has heightened awareness of viral LRTIs.[12] In 2019, LRTIs were recognized as one of the leading causes of mortality worldwide. An American observational study in Chicago, using healthcare

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system records, reported that OSA patients had an increased risk of COVID-19 infection, hospitalization, and worse sequelae compared with patients of the same age who received similar treatment.[13] It was assumed that elevated inflammatory markers in OSA patients contributed to unfavorable COVID-19 outcomes. Moreover, COVID-19 and OSA share several risk factors, such as diabetes, hypertension, cardiovascular disease, and obesity.[13,14]

This review explores the potential link between OSA and LRTIs and the burden of both conditions in adults.

### Objective

To evaluate, through a literature review, the impact of OSA on susceptibility, pathogenesis, and clinical outcomes of respiratory infections, and to discuss potential management strategies.

## METHODS LITERATURE SEARCH

A systematic search of PubMed, Web of Science, and CENTRAL, using specified keywords, was conducted to identify publications up to January 31, 2025. Our search strategy followed a stepwise approach to ensure comprehensive capture of relevant literature. We began with search terms for obstructive sleep apnea (Search #1), combining MeSH terms such as "Obstructive Sleep Apnea" and "Sleep Apnea, Obstructive" with title and abstract keywords, including "Sleep-related breathing disorders" and "OSA". We also included broader MeSH terms like "Sleep-Disordered Breathing" and "Sleep Apnea Syndromes" to ensure comprehensive coverage for respiratory infections, we constructed three separate search sets: one for bacterial pneumonia (Search #2) using terms such as "Pneumonia"[MeSH], "bacterial pneumonia," community-acquired pneumonia, and "Lower Respiratory Tract Infections"[MeSH]; another for influenza (Search #3) using "Influenza, Human"[MeSH] and related terms; and a third for COVID-19 (Search #4) using appropriate MeSH terms and keywords. These respiratory infection searches were combined using the Boolean operator "OR" (Search #5) and were then intersected with the OSA search using "AND" (Search #6). Finally, we applied filters for English-language publications, human studies, an adult population (19+ years), and publication year 2025 (Search #7). The summary of the research strategy is listed in Table 1.

**Main Points**

- Emerging evidence suggests that obstructive sleep apnea (OSA) may increase lower respiratory tract infections, particularly in individuals with severe disease and higher hypoxic burden.
- Biological mechanisms such as intermittent hypoxia, systemic inflammation, immune dysregulation, impaired mucociliary clearance, and microaspiration may contribute to the observed association between OSA and infection severity.
- Consideration of preventive strategies-including vaccination, comorbidity optimization, and careful continuous positive airway pressure hygiene- may help reduce infectious morbidity in patients with moderate-to-severe OSA.

## SEARCH STRATEGY LITERATURE

Both authors (A.M.E., S.K.) searched for, and conducted a full-text review of, the retrieved literature. The two authors discussed the exclusion and inclusion criteria of the retrieved studies and reached a consensus.

### Results of Literature Search

A systematic search of the literature was conducted in PubMed, CENTRAL, and Web of Science to evaluate whether OSA in adults is associated with an increased risk of respiratory infections.

The search strategies included free-text terms and controlled vocabulary for OSA ("Sleep Apnea, Obstructive," "OSA," "Sleep-related breathing disorders") and for respiratory infections ("respiratory tract infections," "pneumonia," "bronchitis," "influenza," "COVID-19," and related terms).

The search retrieved a total of 3,975 records (PubMed: 1,440; CENTRAL: 1,294; Web of Science: 1,241). After removal of 1,070 duplicate records, 2,905 unique records remained for title and abstract screening. Full-text articles were subsequently assessed for eligibility, and the reasons for exclusion were recorded. The final numbers of included studies are presented in the PRISMA flow diagram Figure 1.

## PATHOPHYSIOLOGICAL MECHANISMS

### Hypoxia and Inflammation

Healthy sleep plays a crucial role in regulating the immune system.[15,16] OSA patients experience chronic sleep deprivation and intermittent hypoxia. Immune perturbations secondary to disrupted sleep led to increased oxidative stress and systemic inflammation, which may render those patients susceptible

**Table 1.** Summary of the research strategy

| Items | Specification |
|---|---|
| Search date | January 31, 2025 |
| Databases searched | PubMed; Web of Science; CENTRAL |
| Search terms | "Obstructive Sleep Apnea"; "Sleep-related breathing disorders"; OSA; Pneumonia; bacterial pneumonia; community-acquired pneumonia; CAP; respiratory infection; bronchopneumonia |
| Timeframe | Up to January 31, 2025 |
| Inclusion criteria | English language; non-randomized trials; randomized controlled trials (RCTs); proof-of-concept studies; observational studies (cross-sectional, cohort, case-control); case reports; research protocols |
| Exclusion criteria | Unavailable full text (abstracts and letters to the editor) |
| Selection process | Article screening: S.K. and A.M.E.; Literature classification: S.K. and A.M.E.; Review content preparation: S.K. and A.M.E. |

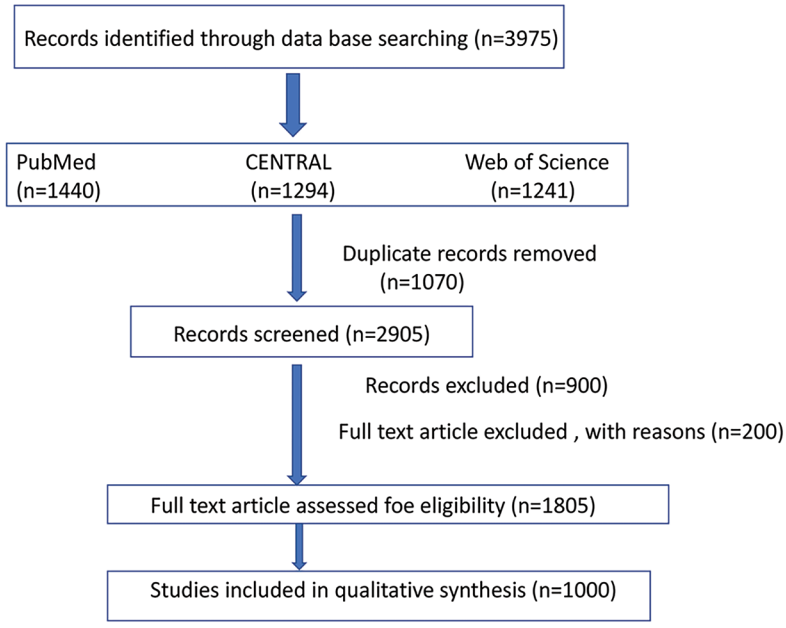


**Figure 1.** PRISMA flow diagram

to pathogen invasion and LRTIs.[15-18] Impaired lung neutrophil function resulting from hypercapnia that may be associated with OSA could render those patients susceptible to LRTIs.[19]

### Impaired Mucociliary Clearance

OSA is associated with alterations in intrathoracic pressure and breathing patterns. Those alterations are more frequently observed during rapid eye movement sleep. They are attributed to inhibition of cortical input to the brainstem cough center. Moreover, at night there is an increase in the peripheral nerves threshold that cause a weak or even an absent cough reflex that leads to an inability to clear pathogens from the airways.[20,21]

### Microaspiration Risk

During the night, aspiration of small amounts is normal in healthy individuals.[22] During sleep, patients with OSA have a higher risk of pulmonary aspiration than individuals without OSA. Following episodes of apnea-hypopnea, patients are stimulated to breathe against a closed airway, leading to a greater increase in negative intrathoracic pressure. As a result, the pressure gradient increases, creating a vacuum effect in the upper airway that leads to excessive microaspiration. Excessive microaspirations lead to increases in bacterial pathogens and upper airway and laryngeal inflammation.[23-28] Treatment of gastroesophageal reflux (GER) may lead to a significant decrease in the apnea-hypopnea index (AHI).[25,29]

### The Role of Comorbidities and Obesity

There is evidence that obesity and comorbid conditions such as diabetes, hypertension, cardiovascular disorders, cerebrovascular disease, GER, Parkinson's disease, epilepsy, and chronic liver and kidney diseases are associated with greater OSA risk. Those comorbidities increase the risk of infections, including LRTIs. Also, in patients with comorbid chronic obstructive pulmonary disease (COPD) and asthma, those who receive corticosteroids have an increased risk of LRTIs.[30]

Obesity increases the risk of complications in critically ill patients.[31-33] However, the "obesity paradox" confers a protective effect against mortality from LRTIs among obese patients. This effect is the result of an early presentation in those patients, increased hospital admission, and changed immune response characterized by lesser inflammation in those patients (Figure 2).[34-36]

## EVIDENCE FROM THE STUDIES

In this retrospective cohort study, adult OSA patients (aged ≥20 years) were enrolled from the Research Database of the Taiwan National Health Insurance. Compared to a cohort control group without OSA, having the same age, sex, and comorbidities. The two cohorts were followed for the occurrence of pneumonia. The study included 34,100 patients (6,816 with OSA and 27,284 controls). During a follow-up period of approximately 4.50 years, 2,757 (8.09%) participants developed pneumonia, including 638 (9.36%) OSA patients and 2,119 (7.77%) in the control cohort. They concluded that pneumonia incidence was higher among OSA patients, based on Kaplan-Meier analysis (log-rank test, $P < 0.001$), and a 1.20-fold increased risk (95% confidence interval 1.10–1.31) after multivariate adjustment. They also stated that the risk was even greater among patients who were treated by continuous positive airway pressure (CPAP).[37] CPAP is the gold-standard therapy for moderate-to-severe OSA. It may decrease sputum expectoration, thereby increasing the risk of aspiration. In addition, humidifiers provide a potential source of bacterial contamination.[38] Therefore, maintenance and regular cleaning of CPAP devices are crucial to avoid the risk of infection.[39]

Chiner et al.[40] conducted a study that included 82 community-acquired pneumonia (CAP) patients and 41 patients with non-respiratory infections. They found that severe OSA patients

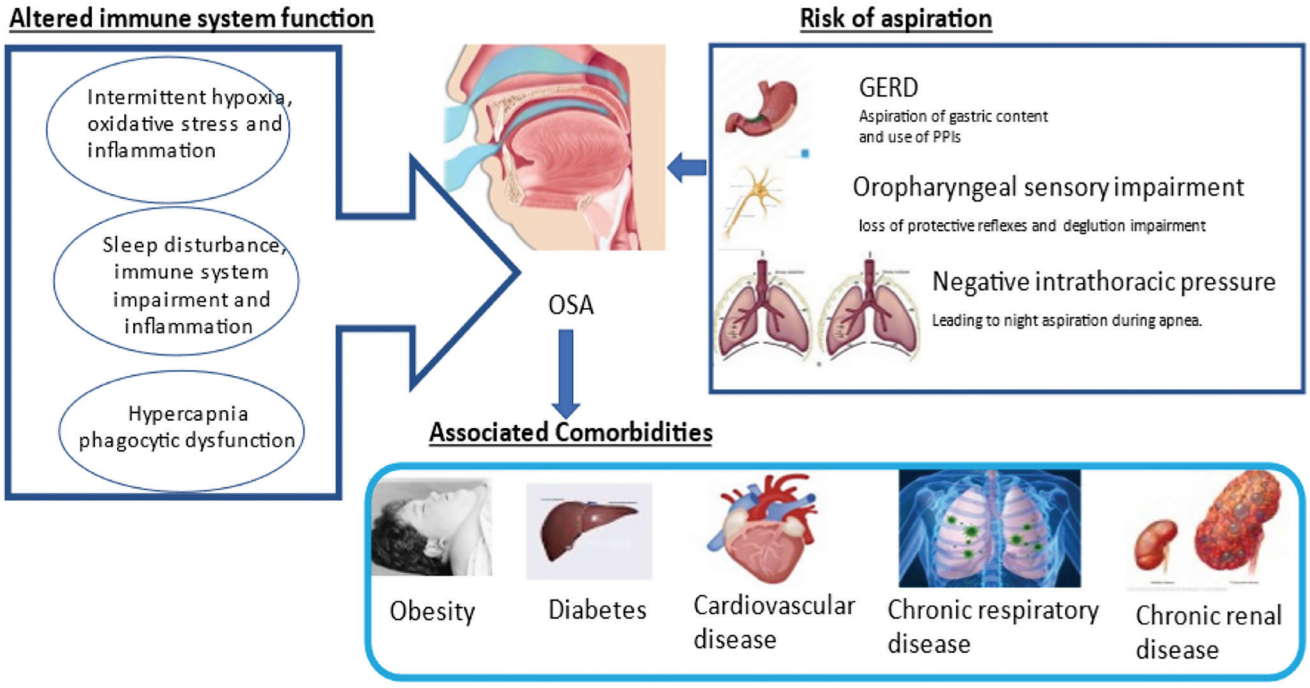


**Figure 2.** Risk factors and pathophysiological interaction of LRT infections and OSA

*LRT: lower respiratory tract infection, OSA: obstructive sleep apnea*

had threefold greater odds (odds ratio: 3.18; 1.11–11.56) of developing pneumonia during sleep than normal-breathing patients. They also concluded that, based on AHI results and various indices of oxygen saturation, higher OSA grade was associated with more severe CAP presentation, which highlights the role of OSA as a risk factor that increases susceptibility and influences the clinical outcomes of CAP.[40]

Notably, during a follow-up period of up to 20.4 years, the Atherosclerosis Risk in Communities prospective cohort study reported that severe OSA increased the risk of pneumonia by 87%. Also, they found that a hypoxic burden [which is the time of oxygen saturation below 90% during total time of sleep (T90)] greater than 5%, compared with less than 1%, was associated with a 50% increased risk of pneumonia. This association suggests that the hypoxic burden and its physiological consequences increase the risk of pneumonia, extending beyond the effects of obesity alone.[41]

Moreover, a nationwide Finnish retrospective population-based case-control study, using data from the national primary and secondary health care registers from 2015–2019, investigated the incidence of LRTs in the first year before and after diagnosis of OSA. They included controls matched for sex, age, and multimorbidity. They analyzed the effect of comorbidities and patient characteristics on the risk of LRTIs and on their recurrence. They reported that OSA patients develop LRTIs at a higher incidence than controls. Hazard ratios were 1.35 (95% confidence interval 1.16–1.57) one year before the diagnosis of OSA and 1.39 (95% confidence interval 1.22–1.58) one year after the diagnosis of OSA. They found that in OSA patients, a previous LRTI before OSA diagnosis, having more than one morbidity, asthma, COPD, and age greater than 65 years increased the incidence and recurrence of LRTIs in those patients.

## DIAGNOSTIC AND SCREENING CONSIDERATIONS

### OSA and LRTIs

CAP is defined as an acute disease characterized by one or more of the following symptoms: cough with or without sputum production, dyspnea, pleuritic chest pain, and fever. On auscultation, adventitious and bronchial breath sounds may be heard. An increased total leucocytic count and the presence of infiltrates on the chest X-ray were observed.[42] Investigations include a complete blood picture, serum C-reactive protein (CRP) levels, and arterial blood gas analysis. Detection of the causative organism is performed by sputum culture or by protected bronchial lavage via fiberoptic bronchoscopy.

After hospitalization, CAP patients suspected of having OSA should be screened with sleep questionnaires to assess OSA symptoms. Daytime sleepiness is screened by the Epworth sleepiness scale.[43] During history taking, smoking status, alcohol consumption, and the presence of associated comorbidities (COPD, diabetes, previous pneumonia, heart failure, asthma, steroid intake during the last 6 months, hospitalization during the previous 3 months, liver and kidney failure, and cerebrovascular disease) should be assessed. Physical examinations include height, weight, body mass index, and the Mallampati scale. The Diagnosis of OSA is confirmed by polysomnography when the AHI is equal to or greater than 5 events per hour of sleep.

Sleep deprivation in OSA patients leads to altered immunity and increases the risk of LRTIs. Hypoxemia and related oxidative stress are linked to activation of the immune system, and inflammatory marker levels increase [proinflammatory cytokines, e.g., interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-α), and CRP].[44-47] Moreover, sleep deprivation without

hypoxemia induces inflammation and elevates inflammatory markers in untreated sleep apnea patients.[48,49]

### OSA and Pneumonia Severity

Chronic inflammation, hypoxia, and sleep fragmentation associated with the pathogenesis of OSA lead to alterations in immune system activity and an increased risk of severe infection.[50-52] Gastro-esophageal reflux disease associated with OSA increases the risk of pulmonary aspiration and the subsequent development of aspiration pneumonia.[53] The airway microbiome in OSA patients is altered compared with that in healthy individuals. It increases predisposition to respiratory infection as its severity increases.[54]

## CLINICAL IMPLICATION AND MANAGEMENT

### Role of CPAP Therapy

Treatment with a positive airway pressure device, together with lifestyle interventions, remains the gold-standard treatment for moderate-to-severe OSA. CPAP use for at least four hours per night has been shown to decrease sleep fragmentation and improve oxygenation, thereby reducing inflammation and enhancing sleep quality. Special consideration of the use of a suitable interface and its proper hygiene to decrease the risk of infections.[55]

### Role of Antibiotic Therapy

Determining the causal agent of CAP is challenging. The American Thoracic Society and Infectious Diseases Society of America (ATS/IDSA) 2019 guidelines recommended empirical antibiotic therapy.[56] Regimen choice is based on the patient characteristics, the pneumonia severity index (PSI), the presence of comorbidities, risk of complications, and the clinical decision to hospitalize.[56-58]

OSA may augment PSI scores and lead to hospitalization because associated comorbid conditions necessitate broader antimicrobial coverage.

The ATS/IDSA guidelines for outpatient management recommend regimens that depend on the risk of complications.[56]

**Table 2.** The summary of the most relevant literature on the association between OSA and LRTIs

| Author and date | Design | Total n (OSA n) | Inclusion and exclusion criteria | Outcomes | Key Findings | Limitations |
|---|---|---|---|---|---|---|
| Maas et al.,[13] 2021 | Multicentric, retrospective cohort | 5544,884 (~44,877) | I: All patient encounters; January to June 2020 | COVID-19, hospitalization, respiratory failure. | OSA: ↑ COVID-19, OR: 8.6, ↑ hospitalization, OR: 1.65, ↑ respiratory failure, OR: 1.98 | No PSG data, no data on OSA treatment |
| Cade et al.,[14] 2020 | Multicentric, retrospective cohort | 4668 (443) | I: Positive COVID-19 PCR; A minimum of two clinical notes, two encounters, and three ICD diagnoses | Mortality, composite (mortality, MV, ICU), hospitalization. | OSA or CPAP not linked with mortality, MV, ICU, and hospitalization | No PSG data, no data on OSA treatment |
| Chiner et al.,[40] 2016 | Single center case-control | 123 (85) | I: Cases: Hospitalized for CAP; Controls: Hospitalized for non-respiratory/non-ENT infection. E: Previous OSA diagnosis and CPAP | Pneumonia, PSI | AHI ≥10: ↑ pneumonia OR: 2.86; AHI ≥30: ↑ pneumonia OR: 3.184; AHI positively correlated with PSI | Small sample size, no data on OSA treatment |
| Keto et al.,[49] 2023 | Case-control from Finland | 50,648 (25,324) | I: ICD code for OSA. E: OSA in the two years preceding the index date | LRTI, recurring LRTI | ↑ LRTI in the year preceding OSA RR: 1.35, and during the year after OSA RR: 1.39 | No PSG data, no data on OSA treatment, no BMI data |
| Girardin et al.,[59] 2021 | Retrospective cohort from NYC and LI | 4446 (290) | I: Positive COVID-19 PCR | Hospital mortality | OSA not linked to mortality | No PSG data, no data on OSA treatment, no BMI data |
| Lutsey et al.,[41] 2023 | Post-hoc analysis of the multicentric prospective cohort | 1586 (772) | I: Valid PSG data; Self-identify as White. E: CSA; Already had the outcome of interest at the time of visit | Hospitalization: with pneumonia; with respiratory infection; with any infection | OSA not linked to outcomes; T90 >5% ↑ hospitalized pneumonia HR: 1.59, ↑ hospitalized respiratory infection HR: 1.53, ↑ hospitalized any infection HR: 1.25 | No data on OSA treatment, mostly White population |

AHI: apnea-hypopnea index, BMI: body mass index, CAP: community-acquired pneumonia, CA: Canada, CPAP: continuous positive airway pressure, CSA: central sleep apnea E: exclusion criteria, HR: hazard ratio, I: inclusion criteria, ICD: International Classification of Diseases, ICU: intensive care unit, LRTI: lower respiratory tract infection, n: number of subjects included, NYC: New York City, OSA: sleep apnea, PCR: polymerase chain reaction, PSG: polysomnography, ↑: increased risk, (OSA N): number of study participants with obstructive sleep apnea, ~: approximation based on presented data

Patients with chronic comorbidities should be treated with dual therapy combining a beta-lactam anti-pneumococcal agent (cefuroxime or amoxicillin-clavulanate) and either doxycycline or a macrolide (e.g., azithromycin or clarithromycin). Fluoroquinolones (e.g., levofloxacin or moxifloxacin) can be used as monotherapy.

Patients with OSA are at increased risk of pneumococcal and streptococcal pneumonia.[60] Therefore, those patients require a more aggressive approach. The recommendations of the Specific Risks Guiding Empiric Antibiotic Therapy Guideline for regimens of antibiotics for CAP recommend to cover the most common and typical (*Streptococcus pneumoniae*, *Staphylococcus aureus*, *Moraxella catarrhalis*, *Haemophilus influenzae*) also the atypical pathogens such as (*Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella* species). Furthermore, the CDC recommends specific antiviral therapies for OSA patients with influenza or COVID-19 infection.[56]

*Pseudomonas aeruginosa* is identified in more than 10% of patients diagnosed with OSA who present with bacterial pneumonia.[40] OSA is associated with several comorbidities[59] that increase the risk of *Pseudomonas* infection. Thus, in severe LRTI cases with one or more associated comorbidities or with OSA, it is advisable to use a fluoroquinolone or an antipseudomonal beta-lactam as early coverage for *Pseudomonas*.[56]

Case reports have documented Legionnaires' disease in OSA patients using CPAP.[61,62] Legionnaires' disease is characterized by neurological disturbances, bradycardia, diarrhea, and hyponatremia.[63] Diagnosis is confirmed by detecting Legionella antigen in urine. Treatment includes respiratory fluoroquinolones.[64]

## PREVENTIVE STRATEGIES

Current CDC guidelines recommend pneumococcal vaccination for individuals aged 65 years and older and for patients under 65 years with asthma, COPD, or emphysema.[65] They also recommend annual influenza vaccinations for individuals aged six months or older.[66] COVID-19 vaccinations are advisable in light of new variants.[67,68] We recommend prioritizing patients with OSA for vaccination against these infections.

## DISCUSSION

This review highlights the emerging and clinically significant association between OSA and LRTIs. Traditionally, the clinical focus of OSA research has been directed toward cardiovascular, metabolic, and neurocognitive sequelae. However, the accumulating evidence reviewed here suggests that infectious complications —particularly bacterial and viral pneumonias— represent an underrecognized dimension of OSA-related morbidity.

**Pathophysiological Insights:** The pathophysiological mechanisms outlined in this review support a biologically plausible interplay between OSA and increased LRTIs. Intermittent hypoxia, systemic inflammation, immune dysregulation, impaired mucociliary clearance, microaspiration, and comorbid conditions such as obesity and diabetes converge to create a microenvironment favorable for respiratory pathogens. Importantly, these mechanisms extend beyond simple epidemiological associations, strengthening the argument for causality rather than coincidence. Moreover, the interplay between OSA and gastroesophageal reflux disease (GERD) adds another layer of complexity, as chronic aspiration may act as a critical driver of recurrent infections.

**Clinical Evidence:** Several cohort and case–control studies reinforce this biological plausibility by demonstrating higher rates of pneumonia and hospitalization among patients with OSA, with evidence of dose–response relationships linked to OSA severity and hypoxic burden. However, heterogeneity across studies should be acknowledged. Some cohort studies suggest an increased risk only in the year before or the year after OSA diagnosis, raising questions about surveillance bias and the role of comorbidities.

Although CPAP therapy improves sleep-related outcomes, observational studies caution that it may increase the risk of aspiration or lead to microbial colonization of equipment, underscoring that therapy can be both preventive and risk-enhancing when hygiene protocols are not strictly followed.

### Strengths and Limitations of Current Evidence

The strengths of the available evidence include large-scale population studies, long-term prospective cohorts, and consistent findings across different geographical regions. Nonetheless, several limitations must be considered. First, most studies rely on administrative databases, which may underestimate undiagnosed OSA and introduce misclassification bias. Second, confounding by obesity, smoking, or comorbid cardiopulmonary disease is difficult to eliminate, even with multivariable adjustments. Third, mechanistic studies directly linking OSA-related immune dysfunction to clinical infections remain scarce. Finally, heterogeneity in the definitions of both OSA (AHI thresholds and diagnostic modalities) and LRTIs (clinical versus microbiological confirmation) complicates direct comparisons. A summary of the most relevant studies on the association between OSA and LRTIs is listed in Table 2.

**Clinical and Public Health Implications:** From a clinical standpoint, recognition of OSA as a risk factor for LRTIs warrants heightened vigilance. Early identification of OSA in patients presenting with recurrent pneumonia, aspiration events, or unexplained severe CAP could guide tailored management strategies. Preventive interventions such as vaccinations (pneumococcal, influenza, and COVID-19), optimization of comorbid conditions, and patient education regarding CPAP hygiene may have additional benefits in reducing infectious morbidity. In the intensive care setting, awareness of OSA may aid in risk stratification for ventilatory support needs, infection risk, and prognosis.

At the public health level, the intersection of two highly prevalent conditions —OSA and LRTIs— magnifies the global burden. In low- and middle-income countries, where OSA remains underdiagnosed and access to diagnostic sleep studies is limited, the potential additive impact of OSA on pneumonia outcomes warrants urgent attention. Screening strategies integrated into primary care and chronic disease management may provide cost-effective opportunities for risk reduction.[69,70]

**Future Research Directions:** Several important research gaps remain. Future studies should (1) explore biomarkers of OSA-related immune dysfunction (e.g., CRP, IL-6, TNF-α) as predictors of infection risk. (2) Clarify the temporal relationship among OSA onset, diagnosis, and LRTI incidence to disentangle causal pathways from detection bias. (3) Assess the risks and benefits of CPAP therapy in relation to infection outcomes, including prospective studies with microbiome analyses. (4) Investigate whether OSA indices beyond the AHI (e.g., hypoxic burden, sleep fragmentation, nocturnal desaturation) better predict infection susceptibility. (5) Evaluate preventive strategies, including targeted vaccination campaigns and GERD management, among individuals with OSA.

## CONCLUSION

The authors conclude that OSA may increase susceptibility to acute LRTIs and the risk of more severe forms of these infections, although there is no clear evidence that it increases pneumonia-related mortality. They emphasize that preventive strategies, such as vaccination and infection control measures, should be considered, particularly for patients with severe OSA.

This review article examines the association between OSA and both acute and chronic LRTIs, including CAP, viral pneumonia (influenza), and COVID-19. It provides a comprehensive synthesis of the existing literature on pathophysiological mechanisms (hypoxia, inflammation, impaired mucociliary clearance, microaspiration, obesity, and comorbidities), epidemiological data, diagnostic approaches, management strategies, and preventive measures.

To our knowledge, there is no indication that a work of this scope and structure has been published previously.

### Footnotes

### Authorship Contributions

Concept: A.M.E., S.K., Design: A.M.E., S.K., Data Collection or Processing: A.M.E., S.K., Analysis or Interpretation: A.M.E., S.K., Literature Search: S.K., Writing: S.K.

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## REFERENCES


1. Veasey SC, Rosen IM. Obstructive sleep apnea in adults. *N Engl J Med*. 2019;380(15):1442-1449. [Crossref]
2. Sánchez-de-la-Torre M, Campos-Rodriguez F, Barbé F. Obstructive sleep apnoea and cardiovascular disease. *Lancet Respir Med*. 2013;1(1):61-72. [Crossref]
3. Bucks RS, Olaithe M, Eastwood P. Neurocognitive function in obstructive sleep apnoea: a meta-review. *Respirology*. 2013;18(1):61-70. [Crossref]
4. Lattimore JD, Celermajer DS, Wilcox I. Obstructive sleep apnea and cardiovascular disease. *J Am Coll Cardiol*. 2003;41(9):1429-1437. [Crossref]
5. He QY, Feng J, Zhang XL, et al. Relationship of daytime blood pressure and severity of obstructive sleep apnea among Chinese: a multi-center investigation in China. *Chin Med J (Engl)*. 2010;123(1):18-22. [Crossref]
6. Chen R, Xiong KP, Lian YX, et al. Daytime sleepiness and its determining factors in Chinese obstructive sleep apnea patients. *Sleep Breath*. 2011;15(1):129-135. [Crossref]
7. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol*. 2013;177(9):1006-1014. [Crossref]
8. Feldman C, Shaddock E. Epidemiology of lower respiratory tract infections in adults. *Expert Rev Respir Med*. 2019;13(1):63-77. [Crossref]
9. Mizgerd JP. Acute lower respiratory tract infection. *N Engl J Med*. 2008;358(7):716-727. [Crossref]
10. Ludwig K, Huppertz T, Radsak M, Gouveris H. Cellular immune dysfunction in obstructive sleep apnea. *Front Surg*. 2022;9:890377. [Crossref]
11. Faverio P, Zanini U, Monzani A, et al. Sleep-disordered breathing and chronic respiratory infections: a narrative review in adult and pediatric population. *Int J Mol Sci*. 2023;24(6):5504. [Crossref]
12. Docherty AB, Harrison EM, Green CA, et al. Features of 20133 UK patients in hospital with covid-19 using the ISARIC WHO Clinical Characterisation Protocol: prospective observational cohort study. *BMJ*. 2020;369:m1985. [Crossref]
13. Maas MB, Kim M, Malkani RG, Abbott SM, Zee PC. Obstructive sleep apnea and risk of COVID-19 Infection, hospitalization and respiratory failure. *Sleep Breath*. 2021;25(2):1155-1157. [Crossref]
14. Cade BE, Dashti HS, Hassan SM, Redline S, Karlson EW. Sleep apnea and COVID-19 mortality and hospitalization. *Am J Respir Crit Care Med*. 2020;202(10):1462-1464. [Crossref]
15. Irwin MR. Sleep and inflammation: partners in sickness and in health. *Nat Rev Immunol*. 2019;19(11):702-715. [Crossref]
16. Besedovsky L, Lange T, Born J. Sleep and immune function. *Pflugers Arch*. 2012;463(1):121-137. [Crossref]
17. Bouloukaki I, Mermigkis C, Kallergis EM, Moniaki V, Mauroudi E, Schiza SE. Obstructive sleep apnea syndrome and cardiovascular disease: the influence of C-reactive protein. *World J Exp Med*. 2015;5(2):77-83. [Crossref]
18. Patel SR, Malhotra A, Gao X, Hu FB, Neuman MI, Fawzi WW. A prospective study of sleep duration and pneumonia risk in women. *Sleep*. 2012;35(1):97-101. [Crossref]
19. Gates KL, Howell HA, Nair A, et al. Hypercapnia impairs lung neutrophil function and increases mortality in murine pseudomonas pneumonia. *Am J Respir Cell Mol Biol*. 2013;49(5):821-828. [Crossref]
20. Sullivan CE, Kozar LF, Murphy E, Phillipson EA. Arousal, ventilatory, and airway responses to bronchopulmonary stimulation in sleeping dogs. *J Appl Physiol Respir Environ Exerc Physiol*. 1979;47(1):17-25. [Crossref]
21. Wang HD, Nakagawa T, Sekizawa K, Kamanaka M, Sasaki H. Cough reflex in the night. *Chest*. 1998;114(5):1496-1497. [Crossref]
22. Ramsey D, Smithard D, Kalra L. Silent aspiration: what do we know? *Dysphagia*. 2005;20(3):218-225. [Crossref]
23. Schindler A, Mozzanica F, Sonzini G, et al. Oropharyngeal dysphagia in patients with obstructive sleep apnea syndrome. *Dysphagia*. 2014;29(1):44-51. [Crossref]
24. Ravelli AM, Panarotto MB, Verdoni L, Consolati V, Bolognini S. Pulmonary aspiration shown by scintigraphy in gastroesophageal

reflux-related respiratory disease. *Chest.* 2006;130(5):1520-1526. [Crossref]

25. Friedman M, Gurpinar B, Lin HC, Schalch P, Joseph NJ. Impact of treatment of gastroesophageal reflux on obstructive sleep apnea-hypopnea syndrome. *Ann Otol Rhinol Laryngol.* 2007;116(11):805-811. [Crossref]
26. Lindam A, Kendall BJ, Thrift AP, et al. Symptoms of Obstructive sleep apnea, gastroesophageal reflux and the risk of Barrett's esophagus in a population-based case-control study. *PLoS One.* 2015;10(6):e0129836. [Crossref]
27. Konermann M, Radü HJ, Teschler H, Rawert B, Heimbucher J, Sanner BM. Interaction of sleep disturbances and gastroesophageal reflux in chronic laryngitis. *Am J Otolaryngol.* 2002;23(1):20-26. [Crossref]
28. Payne RJ, Kost KM, Frenkiel S, et al. Laryngeal inflammation assessed using the reflux finding score in obstructive sleep apnea. *Otolaryngol Head Neck Surg.* 2006;134(5):836-842. [Crossref]
29. Suurna MV, Welge J, Surdulescu V, Kushner J, Steward DL. Randomized placebo-controlled trial of pantoprazole for daytime sleepiness in GERD and obstructive sleep disordered breathing. *Otolaryngol Head Neck Surg.* 2008;139(2):286-290. [Crossref]
30. Almirall J, Bolíbar I, Serra-Prat M, et al. New evidence of risk factors for community-acquired pneumonia: a population-based study. *Eur Respir J.* 2008;31(6):1274-1284. [Crossref]
31. Bailly S, Galerneau LM, Ruckly S, et al. Impact of obstructive sleep apnea on the obesity paradox in critically ill patients. *J Crit Care.* 2020;56:120-124. [Crossref]
32. Jehan S, Zizi F, Pandi-Perumal SR, et al. Obstructive sleep apnea and obesity: implications for public health. *Sleep Med Disord.* 2017;1(4):00019. [Crossref]
33. Anderson MR, Shashaty MGS. Impact of obesity in critical illness. *Chest.* 2021;160(6):2135-2145. [Crossref]
34. Zhao Y, Li Z, Yang T, Wang M, Xi X. Is body mass index associated with outcomes of mechanically ventilated adult patients in intensive critical units? A systematic review and meta-analysis. *PLoS One.* 2018;13(6):e0198669. [Crossref]
35. Kahlon S, Eurich DT, Padwal RS, et al. Obesity and outcomes in patients hospitalized with pneumonia. *Clin Microbiol Infect.* 2013;19(8):709-716. [Crossref]
36. Nemet M, Vukoja M. Obstructive sleep apnea and acute lower respiratory tract infections: a narrative literature review. *Antibiotics (Basel).* 2024;13(6):532. [Crossref]
37. Su VY, Liu CJ, Wang HK, et al. Sleep apnea and risk of pneumonia: a nationwide population-based study. *CMAJ.* 2014;186(6):415-421. [Crossref]
38. Gay PC. Complications of noninvasive ventilation in acute care. *Respir Care.* 2009;54(2):246-257. [Crossref]
39. Patel SR. Providing cleaning recommendations for positive airway pressure devices. *Ann Am Thorac Soc.* 2024;21(1):27-29. [Crossref]
40. Chiner E, Llombart M, Valls J, et al. Association between obstructive sleep apnea and community-acquired pneumonia. *PLoS One.* 2016;11(4):e0152749. [Crossref]
41. Lutsey PL, Zineldin I, Misialek JR, et al. OSA and subsequent risk of hospitalization with pneumonia, respiratory infection, and total infection: the atherosclerosis risk in communities study. *Chest.* 2023;163(4):942-952. [Crossref]
42. Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis.* 2007;44 Suppl 2(Suppl 2):S27-S72. [Crossref]
43. Chiner E, Arriero JM, Signes-Costa J, Marco J, Fuentes I. Validación de la versión española del test de somnolencia Epworth en pacientes con síndrome de apnea de sueño [Validation of the Spanish version of the Epworth Sleepiness Scale in patients with a sleep apnea syndrome]. *Arch Bronconeumol.* 1999;35(9):422-427. [Crossref]
44. Li K, Wei P, Qin Y, Wei Y. Is C-reactive protein a marker of obstructive sleep apnea?: A meta-analysis. *Medicine (Baltimore).* 2017;96(19):e6850. [Crossref]
45. Yokoe T, Minoguchi K, Matsuo H, et al. Elevated levels of C-reactive protein and interleukin-6 in patients with obstructive sleep apnea syndrome are decreased by nasal continuous positive airway pressure. *Circulation.* 2003;107(8):1129-1134. [Crossref]
46. Yamauchi M, Nakano H, Maekawa J, et al. Oxidative stress in obstructive sleep apnea. *Chest.* 2005;127(5):1674-1679. [Crossref]
47. Maniaci A, Iannella G, Cocuzza S, et al. Oxidative stress and inflammation biomarker expression in obstructive sleep apnea patients. *J Clin Med.* 2021;10(2):277. [Crossref]
48. Garbarino S, Lanteri P, Bragazzi NL, Magnavita N, Scoditti E. Role of sleep deprivation in immune-related disease risk and outcomes. *Commun Biol.* 2021;4(1):1304. [Crossref]
49. Keto J, Feuth T, Linna M, Saaresranta T. Lower respiratory tract infections among newly diagnosed sleep apnea patients. *BMC Pulm Med.* 2023;23(1):332. [Crossref]
50. Fine MJ, Auble TE, Yealy DM, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. *N Engl J Med.* 1997;336(4):243-250. [Crossref]
51. Frasca D, Blomberg BB, Paganelli R. Aging, obesity, and inflammatory age-related diseases. *Front Immunol.* 2017;8:1745. [Crossref]
52. Freund A, Orjalo AV, Desprez PY, Campisi J. Inflammatory networks during cellular senescence: causes and consequences. *Trends Mol Med.* 2010;16(5):238-246. [Crossref]
53. Wu ZH, Yang XP, Niu X, Xiao XY, Chen X. The relationship between obstructive sleep apnea hypopnea syndrome and gastroesophageal reflux disease: a meta-analysis. *Sleep Breath.* 2019;23(2):389-397. [Crossref]
54. Lu D, Yao X, Abulimiti A, et al. Profiling of lung microbiota in the patients with obstructive sleep apnea. *Medicine (Baltimore).* 2018;97(26):e11175. [Crossref]
55. Laitinen LA, Anttalainen U, Pietinalho A, Hämäläinen P, Koskela K; Expert Advisory Group Listed in Foreword. Sleep apnoea: Finnish National guidelines for prevention and treatment 2002-2012. *Respir Med.* 2003;97(4):337-365. [Crossref]
56. Metlay JP, Waterer GW, Long AC, et al. Diagnosis and treatment of adults with community-acquired pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America. *Am J Respir Crit Care Med.* 2019;200(7):e45-e67. [Crossref]
57. Kim MA, Park JS, Lee CW, Choi WI. Pneumonia severity index in viral community acquired pneumonia in adults. *PLoS One.* 2019;14(3):e0210102. [Crossref]
58. Grant LR, Meche A, McGrath L, et al. Risk of pneumococcal disease in US adults by age and risk profile. *Open Forum Infect Dis.* 2023;10(5):ofad192. [Crossref]
59. Girardin JL, Seixas A, Ramos Cejudo J, et al. Contribution of pulmonary diseases to COVID-19 mortality in a diverse urban community of New York. *Chron Respir Dis.* 2021;18:1479973120986806. Erratum in: *Chron Respir Dis.* 2021;18:14799731211013120. [Crossref]

60. Restrepo MI, Babu BL, Reyes LF, et al. Burden and risk factors for *Pseudomonas aeruginosa* community-acquired pneumonia: a multinational point prevalence study of hospitalised patients. *Eur Respir J.* 2018;52(2):1701190. **[Crossref]**

61. Srivali N, Chongnarungsin D, Ungprasert P, Edmonds LC. Two cases of Legionnaires' disease associated with continuous positive airway pressure therapy. *Sleep Med.* 2013;14(10):1038. **[Crossref]**

62. Schnirman R, Nur N, Bonitati A, Carino G. A case of legionella pneumonia caused by home use of continuous positive airway pressure. *SAGE Open Med Case Rep.* 2017;5:2050313X17744981. **[Crossref]**

63. Brady MF, Awosika AO, Sundareshan V. Legionnaires' disease. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024. Accessed January 31, 2025. **[Crossref]**

64. Kato H, Hagihara M, Asai N, et al. Meta-analysis of fluoroquinolones versus macrolides for treatment of legionella pneumonia. *J Infect Chemother.* 2021;27(3):424-433. **[Crossref]**

65. Kobayashi M, Pilishvili T, Farrar JL, et al. Pneumococcal vaccine for adults aged ≥19 years: recommendations of the Advisory Committee on Immunization Practices, United States, 2023. *MMWR Recomm Rep.* 2023;72(3):1-39. **[Crossref]**

66. Influenza Vaccination: A Summary for CliniciansǀCDC. Available online: https://www.cdc.gov/flu/professionals/vaccination/ vax-summary. htm#vaccinated (accessed on 10 April 2024). **[Crossref]**

67. Kaku Y, Okumura K, Padilla-Blanco M, et al. Virological characteristics of the SARS-CoV-2 JN.1 variant. *Lancet Infect Dis.* 2024;24(2):e82. **[Crossref]**

68. Jeworowski LM, Mühlemann B, Walper F, et al. Humoral immune escape by current SARS-CoV-2 variants BA.2.86 and JN.1, December 2023. *Euro Surveill.* 2024;29(2):2300740. **[Crossref]**

69. Roche J, Rae DE, Redman KN, et al. Sleep disorders in low- and middle-income countries: a call for action. *J Clin Sleep Med.* 2021;17(11):2341-2342. **[Crossref]**

70. Benjafield AV, Ayas NT, Eastwood PR, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med.* 2019;7(8):687-698. **[Crossref]**