International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	THE IMPACT OF OBESITY ON THE DEVELOPMENT AND PROGRESSION OF SELECTED RESPIRATORY DISEASES — OBSTRUCTIVE SLEEP APNEA, ASTHMA, AND COVID-19
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5863
RECEIVED	18 March 2026
ACCEPTED	15 June 2026
PUBLISHED	18 June 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

THE IMPACT OF OBESITY ON THE DEVELOPMENT AND PROGRESSION OF SELECTED RESPIRATORY DISEASES — OBSTRUCTIVE SLEEP APNEA, ASTHMA, AND COVID-19

Ewa Wojtanowska *(Corresponding Author, Email: ewa.wojtanowska@op.pl)*
Medical University of Lodz, Lodz, Poland
ORCID ID: 0009-0007-4813-9473

Ewa Jachimczak
University Hospital in Krakow, Poland
ORCID ID: 0009-0009-6899-3460

Paulina Jaruga
Regional Specialized Hospital No.4, Bytom, Poland
ORCID ID: 0009-0009-3178-3785

Karolina Grodkowska-Szukała
Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0000-6179-7242

Julia Kozicka
Regional Specialized Hospital No.4, Bytom, Poland
ORCID ID: 0009-0003-8138-6231

Julia Paczyna
Regional Specialized Hospital No.4, Bytom, Poland
ORCID ID: 0009-0006-5485-7167

Marcin Mazur
Medical University of Lodz, Lodz, Poland
ORCID ID: 0009-0008-2408-2107

Vanessa Gąsiorowska
Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0008-2420-627X

Aleksandra Suszczyńska
Medical University of Lodz, Lodz, Poland
ORCID ID: 0009-0009-0923-8529

Angelika Wiatr
Medical University of Lodz, Lodz, Poland
ORCID ID: 0009-0002-0202-4535

ABSTRACT

Obesity (BMI ≥30 kg/m²) is a growing global health problem that significantly affects respiratory function and disease. Excess adipose tissue can alter lung mechanics, reducing respiratory system compliance and lowering functional residual capacity while increasing airway resistance, particularly when lying down. Concurrently, obesity promotes a chronic, low-grade inflammatory state through hypertrophic adipocytes and macrophage infiltration, resulting in the overproduction of cytokines (e.g. IL-6 and TNF-α), adipokines (e.g. leptin) and mediators (e.g. HMGB1). These mechanical and inflammatory changes contribute to airway remodelling, endothelial dysfunction and an increased susceptibility to chronic and infectious respiratory diseases.

This review summarises the current evidence on the impact of obesity on asthma, obstructive sleep apnoea (OSA), and SARS-CoV-2 infection. In asthma, obesity defines a distinct phenotype that is often difficult to treat, characterised by more persistent disease, a reduced response to inhaled glucocorticoids and spirometric changes that improve with weight loss. In OSA, obesity — particularly central and visceral fat — narrows the upper airway, destabilises ventilatory control, and markedly increases disease prevalence and severity. Indices of abdominal adiposity correlate more strongly with apnoea–hypopnoea burden than BMI alone. In the context of SARS-CoV-2 infection, obesity is a major risk factor for severe disease, higher rates of hospitalisation, ICU admission, ARDS and death, with risk rising across obesity classes. Obese patients also more often require invasive ventilation or ECMO, and they may experience prolonged respiratory impairment and long-term symptoms of post-viral fatigue.

KEYWORDS

Obesity, Covid-19, Asthma Obesity, Obstructive Sleep Apnea

CITATION

Ewa Wojtanowska, Ewa Jachimczak, Paulina Jaruga, Karolina Grodkowska-Szukała, Julia Kozicka, Julia Paczyna, Marcin Mazur, Vanessa Gąsiorowska, Aleksandra Suszczyńska, Angelika Wiatr. (2026) The Impact of Obesity on the Development and Progression of Selected Respiratory Diseases — Obstructive Sleep Apnea, Asthma, and COVID-19. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5863

1. Introduction

Obesity (body mass index or BMI ≥ 30 kg/m^2) has become a global epidemic and a public health crisis. It is increasing in Western countries.High-calorie diets are a contributing factor(Green i in., 2020). These are characterised by an imbalance between caloric intake and energy expenditure. This promotes adipose tissue expansion. Adipose tissue expansion is necessary to buffer nutrient excess(Marcelin i in., 2019). Sedentary lifestyles have also been identified as a significant contributor to this widespread issue.

Genetic obesity is rare. This includes monogenic and syndromic obesity. These are complex neuro-endocrine pathologies. The genetic contribution is predominant(Fitch i in., 2024). The effects of obesity on lung function are attributed to two things. Firstly, there are mechanical factors. Secondly, there are complex metabolic effects. These contribute to a pro-inflammatory state. Obesity is a significant risk factor and disease modifier for asthma, obstructive sleep apnea, obesity hypoventilation syndrome (OHS), acute respiratory distress syndrome (ARDS) and chronic obstructive pulmonary disease (COPD)(Dixon & Peters, 2018). Respiratory infections are more likely to occur in obese people. Obesity has been shown to increase oxygen consumption, elevate levels of carbon dioxide, and impose additional mechanical strain on the respiratory system. The underlying mechanism of this phenomenon involves the stiffening effect of obesity, which has been identified as a significant contributor to the observed effects(Sood, 2009). In addition, if they are hospitalised for respiratory disease, they are more likely to be admitted to hospital than healthy-weight people(Lempesis & Georgakopoulou, 2023).

2. Review Methods

A literature review was conducted using the PubMed database to identify relevant and up-to-date scientific articles on obesity and its physiological and clinical implications.

The search strategy was based on a combination of keywords and terms from the Medical Subject Headings (MeSH) index, including "obesity," "genetic obesity," "energy balance," "respiratory function," and "inflammation", "asthma and obesity", "OSA", "COVID - 19". Inclusion criteria encompassed articles that had undergone a peer-review process, with particular emphasis on the most recent literature. Priority was given to the review of articles, systematic reviews, and meta-analyses. The inclusion of original research studies was contingent upon the provision of pertinent information regarding the mechanisms by which obesity is linked to metabolic and respiratory outcomes.

The exclusion criteria encompassed publications in languages other than English, case reports, conference abstracts, and studies not directly related to the topic of obesity and its impact on the respiratory system or that were inconsistent with the scope of this review.

Titles and abstracts were screened for relevance, followed by an evaluation of the full texts of selected articles. Priority was given to high-quality, highly cited publications indexed in PubMed.

Initially, a total of 127 articles were identified, of which 56 were included in the final analysis after applying the inclusion and exclusion criteria.

3. Pathophysiology of obesity in relation to the respiratory system.

3.1 mechanical factors

It has been established that obesity leads to significant changes in the mechanical characteristics of the lungs and chest wall. This results in a reduction in respiratory system compliance and an increase in airway resistance(Cesanelli i in., 2024) A decrease in functional residual capacity (FRC), producing a predominantly restrictive ventilatory pattern. In severe obesity, total lung capacity (TLC) and vital capacity (VC) may also decrease. Moreover, the act of respiration with a persistently diminished lung capacity engenders elevated airway resistance. This phenomenon is attributable to the diminution in the diameter of the airways, which engenders increased airflow turbulence and the promotion of airway collapse. It is noteworthy that this process is exacerbated during supine positioning and sleep, periods characterised by a reduction in lung volume.(Lin & Lin, 2012). This phenomenon can be attributed to a shift in the balance between the pressure filling the lungs and the pressure emptying them, caused by a significant accumulation of adipose tissue around the thorax and abdomen(Pelosi i in., 1998). The accumulation of adipose tissue within the thoracic and abdominal cavities exerts pressure on the intra-abdominal cavity and the pleura, consequently impeding the descent of the diaphragm and the protraction of the thorax(Cesanelli i in., 2024).

3.2 Inflammatory and metabolic mechanisms associated with obesity

The recruitment of polarized macrophages by hypertrophic adipocytes, especially in the visceral WAT, instigates low-grade systemic inflammation through the production of excessive inflammatory cytokines. The most significant of these are IL-6, TNF-α, IL-1, IL-10, and MCP-1, leptin, and plasminogen activator inhibitor-1 (PAI-1)(De Leeuw i in., 2021). The consequences of these actions are a state of chronic inflammation and increased levels of cytokines, leading to impaired innate immunity and creating an environment conducive to the development of a heightened inflammatory reaction(Luzi & Radaelli, 2020). Obesity-related airway remodeling has been linked to high mobility group box protein 1 (HMGB1), a nuclear protein acting as an alarmin that activates innate immune responses and contributes to chronic inflammation.This capacity enables it to trigger the activation of the innate immune system and orchestrate various physiological and pathological responses, with a particular emphasis on chronic inflammation(J. Zhang i in., 2017). It has been demonstrated that individuals with obesity frequently display elevated levels of expression of the HMGB1 protein in adipose tissue, as well as heightened levels of the protein in plasma(Guzmán-Ruiz i in., 2021), furthermore It has been demonstrated that HMGB1 is associated with mucus production, and it regulates airway inflammation by directly influencing naive T cells. In addition, it has been shown that HMGB1 induces Th2 and Th17 cell polarization via the toll-like receptors-2 (TLR2) and TLR4, and the receptor for advanced glycation end-products (RAGE)-NF-κB signalling pathways. These observations are particularly pertinent in the context of the asthma mice model(R. Li i in., 2018). Furthermore, research has identified various effects of leptin on epithelial cell lines, including the induction of intercellular adhesion molecule-1 (ICAM-1) expression, increased production of inflammatory mediators (e.g. CCL11, granulocyte colony-stimulating factor [G-CSF], vascular endothelial growth factor [VEGF] and IL-6), promotion of cell migration, inhibition of apoptosis and

stimulation of epithelial cell proliferation(Suzukawa i in., 2015). The precise mechanism by which leptin induces EMT (epithelial-mesenchymal transition) remains unclear. However, it has been hypothesised that this process may involve the activation of the ERK signalling pathway or the PI3K/Akt-dependent pathway(Listyoko i in., 2024). It is important to note that chronic inflammation, associated with obesity, also contributes to remodelling of the airways and vasculature. Pro-inflammatory mediators such as TGF-β, VEGF, IL-6, and leptin have been shown to promote angiogenesis, subepithelial fibrosis, smooth muscle hypertrophy, and endothelial dysfunction. These processes may lead to irreversible structural alterations within the respiratory system and contribute to the progression of chronic pulmonary diseases and severe respiratory infections(Peters i in., 2018).

4. Obesity and Asthma

4.1 Characteristic of asthma

Bronchial asthma, meanwhile, is a heterogeneous disease characterised by persistent respiratory system inflammation, airway hyperreactivity, and airflow obstruction. Airway remodelling, defined as alterations in airway wall structure (for example, significant epithelial damage, airway smooth muscle hypertrophy, collagen deposition and subepithelial fibrosis), is an important aspect of asthma(Savin i in., 2023). Asthma is a respiratory condition that causes symptoms such as shortness of breath, coughing, difficulty breathing and wheezing. It is characterised by recurring episodes of acute attacks and periodic symptoms(Porsbjerg i in., 2023).

4.2 Risk of asthma

The following risk factors for asthma have been identified: factors related to the mother and the prenatal period, maternal asthma, maternal smoking and exposure to secondhand smoke, maternal obesity, male sex, family history of asthma, African American ancestry, preterm birth, lower socioeconomic status, exposure to secondhand smoke, low physical activity, obesity, indoor dampness and mould, air pollution, the composition of the gut or respiratory tract microbiome in early life, viral lower respiratory tract infections.

The primary focus of this review is on factors related to obesity(Melén i in., 2024).

4.3 Obesity as a Distinct Asthma Phenotype and inflammatory mechanisms

Obese asthma is characterised as a distinct asthma phenotype that is complex and difficult to treat. It has been established that the condition is associated with an exacerbation of asthma and a reduction in the efficacy of controller medications. This has the potential to increase the risk of hospital-based care utilisation(Miethe i in., 2020).Two distinct phenotypes have been identified. 1. Allergic asthma in children with obesity, which can exacerbate pre-existing asthma, is a significant concern. This is a form of asthma that is often not allergic and that develops late in life as a consequence of obesity. In obesity, macrophages M1 infiltrate the adipose tissue, along with an increased expression of multiple mediators that amplify and propagate inflammation. This is considered the underlying cause of obesity-related inflammation. Adipose tissue is a significant source of adipokines, including pro-inflammatory leptin, which is produced in excess in obesity, and adiponectin, which has anti-inflammatory effects and is synthesised at a reduced rate(Bantulà i in., 2021). Obesity creates a pro-inflammatory state (“meta-inflammation”) with adipokines and cytokines (e.g., IL-6, TNF-α, leptin) that can promote asthma and airway hyperreactivity and can also impact the efficacy of asthma treatment, particularly by reducing responsiveness to inhaled glucocorticoids(Fang i in., 2025).

4.4 Mechanical Effects of Obesity on Lung Function and spirometry changes

Research indicates that subjects with obesity and asthma (BMI ≥ 30) are 1.5 times more likely to be diagnosed with persistent asthma than those without obesity. In the subgroup analyses, class III obesity (BMI ≥ 40) was identified as a risk factor for receiving a persistent asthma diagnosis, based on available studies without differentiation by sex. It should further be noted that excessive body fat has been shown to restrict the expansion of the chest and lungs during inhalation and to reduce the volume of air exhaled during exhalation. It can also disrupt the balance between the pressure filling and emptying the lungs, leading to a decrease in functional residual capacity(Y. Kim i in., 2025). According to international guidelines for diagnosing and treating asthma, spirometry is the preferred pulmonary study for measuring lung volumes and evaluating the bronchial muscle response to short-acting β2 adrenergic agonists. FEV1 is an indicator of the severity of bronchial obstruction.

Overweight or obese children and adults with asthma have significantly reduced maximum mid-expiratory flow (MMEF of FEF25–75) and a slight decrease in total lung capacity (TLC), residual volume (RV) and functional residual capacity (FRC). Weight loss in obese asthmatics has been shown to improve key respiratory parameters such as FEV1, FVC, mid-flows and PEF, while reducing variability. These findings support the notion that obesity plays a mechanical role in causing respiratory obstruction(Hakala i in., 2000; Reyes Noriega i in., 2023).

5. Obstructive Sleep Apnea

5.1 Characteristic

Obstructive sleep apnoea (OSA) is characterised by breathing disturbances during sleep. Severe obesity is an etiological risk factor for this condition. Repetitive pharyngeal collapse during sleep results in apnoea and hypopnoea, with consequent hypoxemia, hypercapnia, and recurrent arousals. Apnoea was defined as the complete cessation of airflow for a minimum of 10 seconds, whereas hypopnoea was characterised as either a minimum 50% reduction in airflow for a minimum of 10 seconds, or a reduction of less than 50% accompanied by either a minimum 4% decrease in oxyhaemoglobin saturation or arousal. The apnoea-hypopnoea index was determined on the basis of the number of apnoea and hypopnoea events per hour during sleep (X. Li i in., 2023).The presence of obstructive sleep apnoea is characterised by the manifestation of symptoms that are of clinical significance, including but not limited to a state of excessive daytime sleepiness. This condition has been demonstrated to function as an independent risk factor for the development of cardiovascular disease(Malhotra i in., 2024).

5.2 Diagnostic methods

The diagnostic assessment of OSA must encompass a comprehensive sleep evaluation, along with polysomnography as the gold standard test. It is recommended that polysomnography (PSG) or home sleep apnoea test (HSAT) is performed with a technically adequate device for the diagnosis of OSA in uncomplicated adults with clinical presentation suggesting the risk of moderate to severe OSA. HSAT has been determined to be less precise than PSG in the diagnosis of OSA. This is of particular concern when the test returns inconclusive or negative, as it can result in misclassification of patients. Consequently, PSG - especially full night PGS is recommended for patients with a high clinical suspicion of OSA, particularly in cases where HSAT is initially negative. However, the utilisation of HSAT has not been demonstrated to yield a clinical benefit that is inferior to that of PSG when employed in the appropriate setting. In adults suffering from comorbid conditions, the utilisation of PSG is advocated in preference to the use of home sleep apnoea test HSAT, on the basis that the evidence supporting the validity of HSAT in this particular patient population is considered to be inconclusive(Luong i in., 2024). Research has demonstrated that the Berlin Questionnaire, the Epworth Sleepiness Scale (ESS), and the Stop-Bang Test – non-polysomnographic methods employed in the diagnosis of OSA – are inadequate for diagnosis in isolation, yet they can be instrumental in the diagnosis or selection of patients referred for polysomnography. For instance, the ESS is a tool designed to evaluate daytime sleepiness. The Berlin Questionnaire is renowned for its high diagnostic sensitivity and specificity for OSA, and its capacity to assess the severity of the condition. However, its extensive length renders it impractical for clinical use. The Stop-Bang test is a valuable instrument in selecting patients for polysomnography(Güngör i in., 2025)

5.3. Obesity as a factor of OSA

Obesity is a well-documented risk factor for obstructive sleep apnoea (OSA). However, a significant proportion of OSA patients are non-obese, and the association between obesity and OSA may vary by age and sex.Furthermore, a higher frequency was observed among younger women.(Esmaeili i in., 2024). An increase in body weight has been shown to result in the accumulation of adipose tissue within the pharynx, a process which has the potential to constrict the upper airway, thus inducing dysfunction in the pharynx dilator muscles. It has been demonstrated that weight gain can induce or exacerbate OSA(B. Kim & Choi, 2023). The body mass index (BMI) is a metric used to assess obesity and is a widely used tool for the prediction of OSA. However, it should be noted that BMI does not differentiate between differing patterns of fat distribution. OSA has been found to be predominantly linked to central fat distribution and visceral obesity, as demonstrated by research conducted on diverse populations, including Chinese and Korean immigrants residing in the United States(Lee i in., 2024).The waist-to-hip ratio (WHR), a measure of abdominal obesity, exhibits a more robust correlation with OSA in comparison to BMI. The Visceral Adiposity Index (VAI) and the Lipid Accumulation

Index (LAP) are two recently proposed indices that combine anthropometric parameters with lipid levels. The former is a sensitive indicator reflecting visceral obesity, while the latter is derived from a combination of triglyceride levels and waist circumference. As indicated by the findings of research studies, there was a moderate correlation between LAP and VAI on the one hand, and the severity of OSA on the other. The results suggest that a combination of anthropometry and markers of visceral fat may serve as a more effective diagnostic tool for OSA(Deng i in., 2023). Furthermore, a significant number of studies have indicated a positive correlation between increased visceral adipose tissue (VAT) or a higher BMI and a higher apnea-hypopnea index (AHI). In morbidly obese women, the waist-to-hip ratio exhibited a greater capacity to explain variability in AHI than BMI. Models incorporating waist and neck circumference demonstrated superior performance to those utilising BMI alone.(Gasa i in., 2019). The presence of central fat around the neck, trunk, and abdomen, in particular, has been shown to result in the mechanical narrowing of the upper airway and subsequent elevation of the diaphragm. These phenomena have been demonstrated to engender a heightened risk of airway collapse that exceeds the risks associated with BMI alone(KavithaGiri i in., 2021). Moreover, an increased propensity for systemic inflammation and metabolic alterations, which are also exacerbated by visceral fat, has been observed, thereby contributing further to compromised respiratory regulation(Liu i in., 2024).

5.4 Patophysiology of OBS

Patophysiology of obstructive sleep apnea is multifactorial in nature and can be divided into two broad categories: anatomical and non-anatomical factors (Figure 1). However, a common occurrence among patients is the narrowing or complete collapse of the airways during sleep(McNicholas & Korkalainen, 2023).

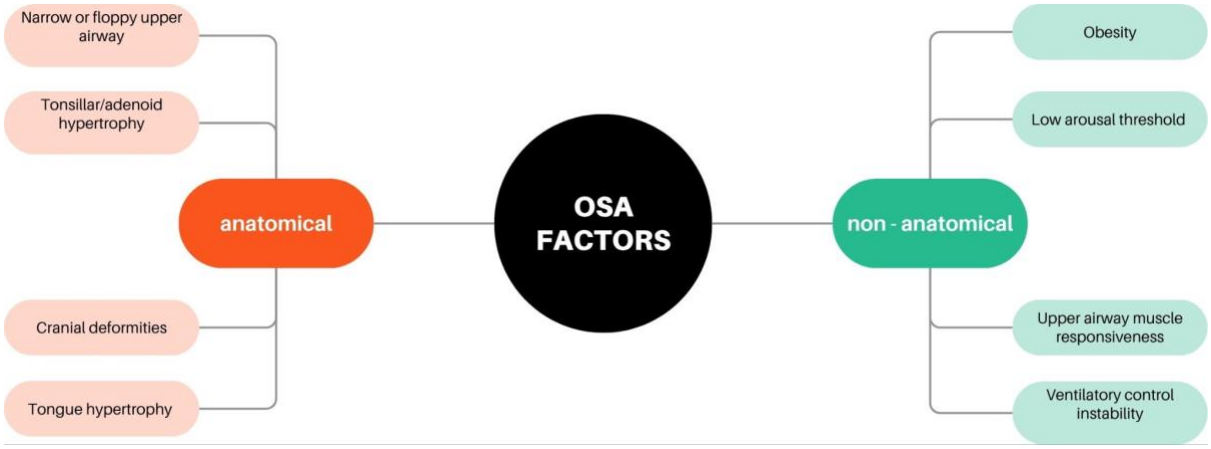

Fig. 1.

The arousal threshold (AT) is defined as the level of ventilatory drive that contributes to arousal from sleep. In contrast, a lower AT fails to allocate adequate time for the recruitment of pharyngeal muscles in response to respiratory stimuli, thus resulting in the reopening of the airway prior to arousal. This, in turn, leads to a destabilisation of breathing. The term 'loop gain' is employed to denote the sensitivity of the ventilatory control system, which is defined as the ratio of the ventilatory response to the reduction in ventilation, which relates to a change in end-tidal CO2 for a given change in ventilation, and a "controller gain", which relates to ventilatory sensitivity to CO2(Koka i in., 2021). A number of factors have been identified as contributing to airway collapse, including reduced activity of the genioglossus muscle and the hypoglossal nerve, the inability of the pharyngeal muscles to maintain airway patency or airway narrowing, hypertrophy of the tonsils and the pharyngeal tonsil, hypersensitivity of the ventilatory control system and a low respiratory drive threshold, mandibular position, cephalometric changes, such as the position of the mandible and hyoid bone and the length of the soft palate, obesity, and the shift of fat and fluids in the body from the lower to the upper body while lying down(B.H. i in., 2023).

5.5 Complication of OSA in individual obese

In obese individuals, obstructive sleep apnoea (OSA) is prevalent and has been demonstrated to be associated with high blood pressure, cardiovascular disease, daytime symptoms, and impaired quality of life. The aforementioned effects are primarily driven by recurrent drops in blood oxygen levels, surges in stress hormones, and fragmented sleep. This has been demonstrated to be concomitant with a two- to threefold elevated risk of cardiovascular and metabolic disease. In patients who are obese, obstructive sleep apnoea (OSA) has been shown to independently correlate with elevated blood pressure, increased arterial stiffness, the development of atherosclerosis, and cardiac remodelling, which is characterised by an increase in left ventricular mass and atrial enlargement. The aforementioned diseases include, albeit not limited to, hypertension, coronary artery disease, heart failure, stroke, arrhythmias and diabetes(Drager i in., 2013; Gottlieb & Punjabi, 2020). It has been demonstrated that low oxygenation, in conjunction with the resultant stress on the cardiovascular system, has the potential to exacerbate or even induce the onset of these conditions. Furthermore, OSA, with its repeated arousals from sleep, has been demonstrated to result in increased inflammation and stress, both of which have been shown to have a detrimental effect on cardiac health. Arrhythmias, particularly atrial fibrillation, have been demonstrated to be associated with OSA. The pressure fluctuations and OSA-related imbalances can cause arrhythmias by creating an arrhythmia-prone environment in the heart, which is especially risky for patients with heart problems because OSA can significantly increase the risk of these events. The long-term effects of chronic hypoxia on the respiratory system include vascular remodelling, pulmonary vasoconstriction, and a mismatch between perfusion and ventilation. This, in turn, leads to pulmonary hypertension(Drager i in., 2013). Moreover sleep apnoea is a condition characterised by recurrent episodes of cessation of breathing during sleep, leading to daytime sleepiness, fatigue, impaired alertness, memory problems, and mood disorders. The condition can also result in a reduced quality of life for the affected individual(Alterki i in., 2023).

6. COVID-19: Correlation between severe disease and obesity

6.1 Pandemic

In 2019, the world was grappling with the COVID-19 pandemic. The SARS-CoV-2 is a positive-stranded RNA virus from the Coronaviridae family. Previous outbreaks of other CoVs include severe acute respiratory syndrome (SARS) in 2002 and the Middle East respiratory syndrome (MERS) in 2012. The virus contains the S protein, the main glycoprotein, which consists of two subunits, S1 and S2. The S1 subunit, the receptor-binding subunit, is responsible for the recognition and binding of the angiotensin-converting enzyme 2 (ACE2) receptor. The latter is a type 1 membrane protein found primarily in type II alveolar cells.Research has demonstrated that the binding affinity between the SARS-CoV-2 S glycoprotein and ACE2 is 10–20 times stronger than that observed between the S glycoprotein and ACE2 of SARS-CoV. This finding may provide a scientific explanation for the high transmissibility of SARS-CoV-2.

The WHO has also classified obesity as a global pandemic, citing a threefold increase in obesity rates between 1975 and 2016. Moreover, being overweight or obese is a documented risk factor for more severe cases of the novel severe acute respiratory syndrome (SARS-CoV-2) virus. The primary means of human-to-human transmission of the virus is through respiratory droplets. The infection may present in a variety of ways, from mild and largely asymptomatic, with symptoms including a cough, fever, loss of smell and taste, and fatigue, to severe respiratory failure. The viral incubation period has been shown to range from 2–14 days, with an average of 5.2 days. The primary means of human-to-human transmission of the virus is through respiratory droplets. The infection may present in a variety of ways, from mild and largely asymptomatic, with symptoms including a cough, fever, loss of smell and taste, and fatigue, to severe respiratory failure. The preferred diagnostic method is the detection of viral genetic material in a nasopharyngeal swab or sputum sample using polymerase chain reaction (PCR), a method recently supplemented by the detection of SARS-CoV-2 antigens with antibody-based tests. A further diagnostic instrument of note is the computerised tomography (CT) scan of the chest, which has been shown to reveal ground-glass opacities, interstitial infiltration, or multiple patchy consolidations in both lungs(De Leeuw i in., 2021; Krishnan i in., 2021).

6.2 The relationship between obesity and severe infection

Excess body fat triggers inflammation by increasing the production of pro-inflammatory cytokines, such as IL-6, C-reactive protein (CRP), GM-CSF, INF-gamma and TNF- alpha. It also increases Th2 and Th1 lymphocytes, which decreases immunity and impairs the immune response.It also raises insulin and leptin levels; the role of these in inflammation was discussed earlier. Obesity can also cause chronic hypoxia. Moreover, chronic inflammation not only predisposes one to the development of chronic diseases, but also worsens infection symptoms. It is worth noting that obesity, particularly abdominal obesity, can lead to pulmonary dysfunction. Although the exact mechanism linking severe obesity to the progression of a SARS-CoV-2 infection is unclear, studies have demonstrated that obesity is a significant risk factor for respiratory infections(Popkin i in., 2020). Numerous studies and meta-analyses and cohort have been conducted on the link between obesity and an increased risk of hospitalisation, intensive care unit (ICU) admission and death. Nearly all of these studies have shown a positive correlation. Meta-analyses consistently show a higher risk of death in obesity: The overall Odds ratios (OR)is 1.61 (1.29–2.01)(Cai i in., 2021) and the overall Relative risk (RR) is 1.42 (1.24–1.63), rising from 1.27 in class I obesity to 1.92 in class III obesity(Poly i in., 2021). A large pooled analysis of 3.14 million patients found that: RR for mortality was 1.09 (1.02–1.16), with part of the observed heterogeneity being explained by age, diabetes, cardiovascular disease (CVD) and hypertension(Singh i in., 2022). A recent synthesis of 199 studies found that the odds of mortality were 34% higher for obese individuals compared to those of normal weight, with a clear dose-dependent increase in mortality risk with increasing BMI(Haber i in., 2024). A selected cohort study showed: in a U.S. hospital network (148,494 adults): obesity was a risk factor for hospitalisation and death, with the risk rising steeply beyond a BMI of ~30, particularly in patients under 65 years of age(Kompaniyets i in., 2021);

American Heart Association registry (7,606 inpatients): class I–III obesity increased the combined endpoint of death or mechanical ventilation (OR 1.28–1.80), and class III obesity increased death (HR 1.26). The effects were strongest in adults aged ≤50 years(Hendren i in., 2021). Kaiser Permanente cohort (6,916 patients): The RR for death was 2.68 at a BMI of 40–44, and 4.18 at a BMI of ≥45, compared to a BMI of 18.5–24. This was independent of comorbidities, with the greatest excess risk observed in men and in those aged ≤60 years(Kompaniyets i in., 2021).

6.3 Respiratory complication

Obesity is a major risk factor for a range of respiratory complications, including acute respiratory distress syndrome (ARDS), respiratory failure and long-term symptoms known as long COVID. Large cohort studies, meta-analyses and mechanistic reviews consistently demonstrate that obese individuals are at increased risk of severe outcomes from SARS-CoV-2 infection, including higher rates of mechanical ventilation and ARDS. Obesity impairs lung mechanics, promotes chronic inflammation and immune dysregulation, and increases susceptibility to acute and chronic respiratory complications. Multiple large-scale studies confirm that obesity, especially severe or extreme obesity, is strongly associated with an increased risk of developing ARDS during a SARS-CoV-2 infection(Heubner i in., 2022; Shah & Kaltsakas, 2023). Meta-analyses show that obese patients are almost three times more likely to develop ARDS than non-obese patients(X. Zhang i in., 2021). They are also more likely to require invasive mechanical ventilation or advanced support, such as extracorporeal membrane oxygenation (ECMO), during severe respiratory illness. Emerging evidence suggests that extreme obesity is an independent predictor of long-term symptoms of post-viral fatigue and dyspnoea after severe ARDS(Heubner i in., 2022). Furthermore, obese patients recovering from severe illness often experience prolonged impairment in lung function and exercise capacity compared to their non-obese counterparts (Vimercati i in., 2021).

However, some studies highlight the complexity of these relationships. The so-called 'obesity paradox' suggests that moderate obesity may not always increase—and may sometimes decrease—mortality among critically ill patients with ARDS or on ECMO support. This effect may be confounded by differences in age and comorbidity, or by selection bias(Dana i in., 2021).

7. Discussion

The reviewed evidence shows that obesity amplifies respiratory disease through both mechanical and inflammatory/metabolic mechanisms. Excess fat mass reduces lung and chest wall compliance, decreases functional residual capacity and increases airway resistance, particularly when lying down, thereby promoting airway narrowing and collapse(Cesanelli i in., 2024). At the same time, hypertrophic adipose tissue creates chronic, low-grade systemic inflammation involving elevated levels of IL-6, TNF-α, IL-1, leptin, and other mediators. This impairs innate immunity and favours exaggerated inflammatory responses in the airways and lung parenchyma(Luzi & Radaelli, 2020).

In asthma, these mechanisms support the concept of an obese-asthma phenotype characterised by more persistent disease, frequent exacerbations, a reduced response to inhaled glucocorticoids, small-airway dysfunction and spirometric improvement following weight loss(Reyes Noriega i in., 2023). In OSA, central and visceral adiposity around the pharynx, neck and thorax narrows the upper airway and destabilises ventilatory control; visceral fat indices also correlate more strongly with apnoea–hypopnoea severity than BMI alone(Lee i in., 2024).

Regarding SARS-CoV-2, multiple meta-analyses and large cohorts consistently link obesity with a higher risk of hospitalisation, ICU admission, ARDS, mechanical ventilation and death, with a clear dose–response relationship from class I to class III obesity(Cai i in., 2021). Obesity also increases susceptibility to ARDS and long-term post-COVID symptoms, although an 'obesity paradox' has been described in some ARDS/ECMO cohorts(X. Zhang i in., 2021).

8. Conclusions

Obesity significantly alters the physiology of the respiratory system and promotes chronic inflammation, making it a major modifying factor in disease progression for many respiratory conditions. Mechanically, excess adipose tissue in the chest and abdominal regions reduces the compliance of the lungs and chest wall, decreases functional residual capacity, and increases airway resistance, particularly when lying on the back and during sleep. This restrictive pattern, combined with diaphragmatic displacement and airway narrowing, promotes reduced airflow, airway collapse, and hypoventilation. Metabolically, hypertrophic adipocytes and infiltrating macrophages in visceral fat induce a state of chronic, low-grade systemic inflammation ("meta-inflammation"), with elevated levels of cytokines and adipokines (e.g., IL-6, TNF-α, leptin, HMGB1), which impair innate immunity, remodel the airways and vascular system, and predispose individuals to chronic lung diseases and severe infections.

In asthma, obesity is associated with a distinct, difficult-to-treat phenotype. Obese individuals are more likely to suffer from persistent asthma, experience more frequent exacerbations, show a reduced response to inhaled glucocorticoids, and exhibit spirometric signs of small airway dysfunction. Weight loss improves FEV_1, FVC, mean and peak flow rates, highlighting the mechanical impact of excess fat on airway obstruction and confirming that weight loss is a therapeutic goal.

In obstructive sleep apnea, obesity—especially central and visceral obesity—is a key etiological factor. Fat deposition around the neck, throat, trunk, and abdomen mechanically narrows the upper airways and elevates the diaphragm, increasing the apnea-hypopnea index. Measures of abdominal and visceral fat (waist-to-hip ratio, LAP, VAI) correlate more strongly with OSA severity than BMI alone. OSA is very common in obese individuals and is independently associated with hypertension, cardiovascular disease, arrhythmias, pulmonary hypertension, metabolic disorders, daytime sleepiness, and reduced quality of life.

In the case of COVID-19, obesity significantly increases the risk of severe disease, including higher rates of hospitalization, ICU admissions, mechanical ventilation, ARDS, and deaths, with a clear dose-response relationship across different obesity classes. Meta-analyses and large cohort studies consistently indicate an increased likelihood and relative risk of mortality, particularly in cases of severe and extreme obesity and among younger adults. Overall, obesity emerges as a key, modifiable factor determining both the development and progression of serious respiratory diseases. Through interrelated mechanical, metabolic, and immunological pathways, it increases the prevalence of diseases, worsens their severity, complicates treatment, and hinders recovery, particularly in the case of asthma, OSA, and COVID-19, while increased physical activity and weight loss are documented prognostic factors for exacerbations and the onset of respiratory diseases.

The authors declare that they have no conflict of interest.

This research received no external founding.

All authors contributed to the study conception, literature review, and manuscript preparation. All authors read and approved the final version of the manuscript.

REFERENCES

1. Alterki, A., Abu-Farha, M., Al Shawaf, E., Al-Mulla, F., & Abubaker, J. (2023). Investigating the relationship between obstructive sleep apnoea, inflammation and cardio-metabolic diseases. *International Journal of Molecular Sciences, 24*(7), 6807. https://doi.org/10.3390/ijms24076807
2. Bantulà, M., Roca-Ferrer, J., Arismendi, E., & Picado, C. (2021). Asthma and obesity: Two diseases on the rise and bridged by inflammation. *Journal of Clinical Medicine, 10*(2), 169. https://doi.org/10.3390/jcm10020169
3. B. H., S., G., D., Sawal, A., & Balwir, T. P. (2023). Anatomical basis of obstructive sleep apnoea: A review of randomized controlled trials. *Cureus*. https://doi.org/10.7759/cureus.44525
4. Cai, Z., Yang, Y., & Zhang, J. (2021). Obesity is associated with severe disease and mortality in patients with coronavirus disease 2019 (COVID-19): A meta-analysis. *BMC Public Health, 21*(1), 1505. https://doi.org/10.1186/s12889-021-11546-6
5. Cesanelli, L., Cesanelli, F., Degens, H., & Satkunskiene, D. (2024). Obesity-related reduced spirometry and altered breathing pattern are associated with mechanical disadvantage of the diaphragm. *Respiratory Physiology & Neurobiology, 325*, 104267. https://doi.org/10.1016/j.resp.2024.104267
6. Dana, R., Bannay, A., Bourst, P., Ziegler, C., Losser, M.-R., Gibot, S., Levy, B., Audibert, G., & Ziegler, O. (2021). Obesity and mortality in critically ill COVID-19 patients with respiratory failure. *International Journal of Obesity, 45*(9), 2028–2037. https://doi.org/10.1038/s41366-021-00872-9
7. De Leeuw, A. J. M., Oude Luttikhuis, M. A. M., Wellen, A. C., Müller, C., & Calkhoven, C. F. (2021). Obesity and its impact on COVID-19. *Journal of Molecular Medicine, 99*(7), 899–915. https://doi.org/10.1007/s00109-021-02072-4
8. Deng, H., Duan, X., Huang, J., Zheng, M., Lao, M., Weng, F., Su, Q., Zheng, Z., Mei, Y., Huang, L., Yang, W., Xing, X., Ma, X., Zhao, W., & Liu, X. (2023). Association of adiposity with risk of obstructive sleep apnea: A population-based study. *BMC Public Health, 23*(1), 1835. https://doi.org/10.1186/s12889-023-16695-4
9. Dixon, A. E., & Peters, U. (2018). The effect of obesity on lung function. *Expert Review of Respiratory Medicine, 12*(9), 755–767. https://doi.org/10.1080/17476348.2018.1506331
10. Drager, L. F., Togeiro, S. M., Polotsky, V. Y., & Lorenzi-Filho, G. (2013). Obstructive sleep apnea. *Journal of the American College of Cardiology, 62*(7), 569–576. https://doi.org/10.1016/j.jacc.2013.05.045
11. Esmaeili, N., Gell, L., Taranto-Montemurro, L., Messineo, L., Imler, T., Sands, S., Yee, J., Cronin, J., Wellman, A., White, D. P., Heinzer, R., Redline, S., & Azarbarzin, A. (2024). Prevalence of obesity in obstructive sleep apnea within a large community-based cohort of middle-aged/older adults. *SLEEP, 47*(Supplement_1), A372–A372. https://doi.org/10.1093/sleep/zsae067.0866
12. Fang, C., Jiang, Z., Su, X., & Fan, W. (2025). The association between body mass index and asthma in children: A cross-sectional study from NHANES 1999 to 2020. *Scientific Reports, 15*(1), 9448. https://doi.org/10.1038/s41598-025-92619-z
13. Fitch, A. K., Malhotra, S., & Conroy, R. (2024). Differentiating monogenic and syndromic obesities from polygenic obesity: Assessment, diagnosis, and management. *Obesity Pillars, 11*, 100110. https://doi.org/10.1016/j.obpill.2024.100110
14. Gasa, M., López-Padrós, C., Monasterio, C., Salord, N., Mayos, M., Vilarrasa, N., Fernandez-Aranda, F., Montserrat, J. M., & Dorca, J. (2019). Anthropometrical phenotypes are important when explaining obstructive sleep apnea in female bariatric cohorts. *Journal of Sleep Research, 28*(5), e12830. https://doi.org/10.1111/jsr.12830
15. Gottlieb, D. J., & Punjabi, N. M. (2020). Diagnosis and management of obstructive sleep apnea: A review. *JAMA, 323*(14), 1389. https://doi.org/10.1001/jama.2020.3514
16. Green, M., Arora, K., & Prakash, S. (2020). Microbial medicine: Prebiotic and probiotic functional foods to target obesity and metabolic syndrome. *International Journal of Molecular Sciences, 21*(8), 2890. https://doi.org/10.3390/ijms21082890
17. Güngör, K., Gülbay, B., & Acican, T. (2025). Investigating the effectiveness of nonpolysomnography diagnostic methods used in sleep disorders in predicting the diagnosis of obstructive sleep apnea syndrome. *Tuberk Toraks, 73*(2), 102–111. https://doi.org/10.5578/tt.2025021111
18. Guzmán-Ruiz, R., Tercero-Alcázar, C., López-Alcalá, J., Sánchez-Ceinos, J., Malagón, M. M., & Gordon, A. (2021). The potential role of the adipokine HMGB1 in obesity and insulin resistance: Novel effects on adipose tissue biology. *Molecular and Cellular Endocrinology, 536*, 111417. https://doi.org/10.1016/j.mce.2021.111417
19. Haber, R., Ghezzawi, M., Puzantian, H., Haber, M., Saad, S., Ghandour, Y., El Bachour, J., Yazbeck, A., Hassanieh, G., Mehdi, C., Ismail, D., Abi-Kharma, E., El-Zein, O., Khamis, A., Chakhtoura, M., & Mantzoros, C. (2024). Mortality risk in patients with obesity and COVID-19 infection: A systematic review and meta-analysis. *Metabolism, 155*, 155812. https://doi.org/10.1016/j.metabol.2024.155812

20. Hakala, K., Stenius-Aarniala, B., & Sovijärvi, A. (2000). Effects of weight loss on peak flow variability, airways obstruction, and lung volumes in obese patients with asthma. *Chest, 118*(5), 1315–1321. https://doi.org/10.1378/chest.118.5.1315
21. Hendren, N. S., De Lemos, J. A., Ayers, C., Das, S. R., Rao, A., Carter, S., Rosenblatt, A., Walchok, J., Omar, W., Khera, R., Hegde, A. A., Drazner, M. H., Neeland, I. J., & Grodin, J. L. (2021). Association of body mass index and age with morbidity and mortality in patients hospitalized with COVID-19: Results from the American Heart Association COVID-19 Cardiovascular Disease Registry. *Circulation, 143*(2), 135–144. https://doi.org/10.1161/CIRCULATIONAHA.120.051936
22. Heubner, L., Petrick, P. L., Güldner, A., Bartels, L., Ragaller, M., Mirus, M., Rand, A., Tiebel, O., Beyer-Westendorf, J., Rößler, M., Schmitt, J., Koch, T., & Spieth, P. M. (2022). Extreme obesity is a strong predictor for in-hospital mortality and the prevalence of long-COVID in severe COVID-19 patients with acute respiratory distress syndrome. *Scientific Reports, 12*(1), 18418. https://doi.org/10.1038/s41598-022-22107-1
23. KavithaGiri, N. L., Mani, M. S., Ahamed, S. Y., & Sivaraman, G. (2021). Evaluation of central obesity, increased body mass index, and its relation to oropharyngeal airway space using lateral cephalogram in risk prediction of obstructive sleep apnea. *Journal of Pharmacy and Bioallied Sciences, 13*(Suppl 1), S549–S554. https://doi.org/10.4103/jpbs.JPBS_566_20
24. Kim, B., & Choi, J. H. (2023). Weight loss for obstructive sleep apnea: Pharmacological and surgical management. *Journal of Rhinology, 30*(1), 1–5. https://doi.org/10.18787/jr.2023.00010
25. Kim, Y., Moonie, S., Yoo, J. W., & Chung, T.-H. (2025). Class III obesity as a risk factor for persistent asthma. *Respiratory Care, 70*(1), 100–107. https://doi.org/10.1089/respcare.11934
26. Koka, V., De Vito, A., Roisman, G., Petitjean, M., Filograna Pignatelli, G. R., Padovani, D., & Randerath, W. (2021). Orofacial myofunctional therapy in obstructive sleep apnea syndrome: A pathophysiological perspective. *Medicina, 57*(4), 323. https://doi.org/10.3390/medicina57040323
27. Kompaniyets, L., Goodman, A. B., Belay, B., Freedman, D. S., Sucosky, M. S., Lange, S. J., Gundlapalli, A. V., Boehmer, T. K., & Blanck, H. M. (2021). Body mass index and risk for COVID-19-related hospitalization, intensive care unit admission, invasive mechanical ventilation, and death—United States, March–December 2020. *MMWR. Morbidity and Mortality Weekly Report, 70*(10), 355–361. https://doi.org/10.15585/mmwr.mm7010e4
28. Krishnan, A., Hamilton, J. P., Alqahtani, S. A., & Woreta, T. A. (2021). A narrative review of coronavirus disease 2019 (COVID-19): Clinical, epidemiological characteristics, and systemic manifestations. *Internal and Emergency Medicine, 16*(4), 815–830. https://doi.org/10.1007/s11739-020-02616-5
29. Lee, S., Ryu, S., Lee, G. E., Redline, S., & Morey, B. N. (2024). Risk of sleep apnea is associated with abdominal obesity among Asian Americans: Comparing waist-to-hip ratio and body mass index. *Journal of Racial and Ethnic Health Disparities, 11*(1), 157–167. https://doi.org/10.1007/s40615-022-01507-z
30. Lempesis, I. G., & Georgakopoulou, V. E. (2023). Implications of obesity and adiposopathy on respiratory infections: Focus on emerging challenges. *World Journal of Clinical Cases, 11*(13), 2925–2933. https://doi.org/10.12998/wjcc.v11.i13.2925
31. Li, R., Wang, J., Zhu, F., Li, R., Liu, B., Xu, W., He, G., Cao, H., Wang, Y., & Yang, J. (2018). HMGB1 regulates T helper 2 and T helper17 cell differentiation both directly and indirectly in asthmatic mice. *Molecular Immunology, 97*, 45–55. https://doi.org/10.1016/j.molimm.2018.02.014
32. Li, X., Wang, T., Jin, L., Li, Z., Hu, C., Yi, H., Guan, J., Xu, H., & Wu, X. (2023). Overall obesity not abdominal obesity has a causal relationship with obstructive sleep apnea in individual level data. *Nature and Science of Sleep, 15*, 785–797. https://doi.org/10.2147/NSS.S422917
33. Lin, C., & Lin, C. (2012). Work of breathing and respiratory drive in obesity. *Respirology, 17*(3), 402–411. https://doi.org/10.1111/j.1440-1843.2011.02124.x
34. Listyoko, A. S., Okazaki, R., Harada, T., Inui, G., & Yamasaki, A. (2024). Impact of obesity on airway remodeling in asthma: Pathophysiological insights and clinical implications. *Frontiers in Allergy, 5*, 1365801. https://doi.org/10.3389/falgy.2024.1365801
35. Liu, W., Song, S., Zhang, H., & An, C. (2024). A comparative analysis of anthropometric indices for diagnosing obstructive sleep apnea among American adults. *Scientific Reports, 14*(1), 29578. https://doi.org/10.1038/s41598-024-81191-7
36. Luong, S., Lezama, L., & Khan, S. (2024). Diagnosis and management of obstructive sleep apnea: Updates and review. *Journal of Otorhinolaryngology, Hearing and Balance Medicine, 5*(2), 16. https://doi.org/10.3390/ohbm5020016
37. Luzi, L., & Radaelli, M. G. (2020). Influenza and obesity: Its odd relationship and the lessons for COVID-19 pandemic. *Acta Diabetologica, 57*(6), 759–764. https://doi.org/10.1007/s00592-020-01522-8
38. Malhotra, A., Grunstein, R. R., Fietze, I., Weaver, T. E., Redline, S., Azarbarzin, A., Sands, S. A., Schwab, R. J., Dunn, J. P., Chakladar, S., Bunck, M. C., & Bednarik, J. (2024). Tirzepatide for the treatment of obstructive sleep apnea and obesity. *New England Journal of Medicine, 391*(13), 1193–1205. https://doi.org/10.1056/NEJMoa2404881

39. Marcelin, G., Silveira, A. L. M., Martins, L. B., Ferreira, A. V. M., & Clément, K. (2019). Deciphering the cellular interplays underlying obesity-induced adipose tissue fibrosis. *Journal of Clinical Investigation, 129*(10), 4032–4040. https://doi.org/10.1172/JCI129192
40. McNicholas, W. T., & Korkalainen, H. (2023). Translation of obstructive sleep apnea pathophysiology and phenotypes to personalized treatment: A narrative review. *Frontiers in Neurology, 14*, 1239016. https://doi.org/10.3389/fneur.2023.1239016
41. Melén, E., Zar, H. J., Siroux, V., Shaw, D., Saglani, S., Koppelman, G. H., Hartert, T., Gern, J. E., Gaston, B., Bush, A., & Zein, J. (2024). Asthma inception: Epidemiologic risk factors and natural history across the life course. *American Journal of Respiratory and Critical Care Medicine, 210*(6), 737–754. https://doi.org/10.1164/rccm.202312-2249SO
42. Miethe, S., Karsonova, A., Karaulov, A., & Renz, H. (2020). Obesity and asthma. *Journal of Allergy and Clinical Immunology, 146*(4), 685–693. https://doi.org/10.1016/j.jaci.2020.08.011
43. Pelosi, P., Croci, M., Ravagnan, I., Tredici, S., Pedoto, A., Lissoni, A., & Gattinoni, L. (1998). The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. *Anesthesia & Analgesia, 87*(3), 654–660. https://doi.org/10.1097/00000539-199809000-00031
44. Peters, U., Dixon, A. E., & Forno, E. (2018). Obesity and asthma. *Journal of Allergy and Clinical Immunology, 141*(4), 1169–1179. https://doi.org/10.1016/j.jaci.2018.02.004
45. Poly, T. N., Islam, M. M., Yang, H. C., Lin, M. C., Jian, W.-S., Hsu, M.-H., & Li, Y.-C. J. (2021). Obesity and mortality among patients diagnosed with COVID-19: A systematic review and meta-analysis. *Frontiers in Medicine, 8*, 620044. https://doi.org/10.3389/fmed.2021.620044
46. Popkin, B. M., Du, S., Green, W. D., Beck, M. A., Algaith, T., Herbst, C. H., Alsukait, R. F., Alluhidan, M., Alazemi, N., & Shekar, M. (2020). Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships. *Obesity Reviews, 21*(11), e13128. https://doi.org/10.1111/obr.13128
47. Porsbjerg, C., Melén, E., Lehtimäki, L., & Shaw, D. (2023). Asthma. *The Lancet, 401*(10379), 858–873. https://doi.org/10.1016/S0140-6736(22)02125-0
48. Reyes Noriega, N., Del-Río-Navarro, B. E., Berber, A., De Jesús Romero Tapia, S., & Molina Díaz, D. J. M. (2023). Effect of obesity on lung function in the pediatric and adult populations with asthma: A review. *Journal of Clinical Medicine, 12*(16), 5385. https://doi.org/10.3390/jcm12165385
49. Savin, I. A., Zenkova, M. A., & Sen'kova, A. V. (2023). Bronchial asthma, airway remodeling and lung fibrosis as successive steps of one process. *International Journal of Molecular Sciences, 24*(22), 16042. https://doi.org/10.3390/ijms242216042
50. Shah, N. M., & Kaltsakas, G. (2023). Respiratory complications of obesity: From early changes to respiratory failure. *Breathe, 19*(1), 220263. https://doi.org/10.1183/20734735.0263-2022
51. Singh, R., Rathore, S. S., Khan, H., Karale, S., Chawla, Y., Iqbal, K., Bhurwal, A., Tekin, A., Jain, N., Mehra, I., Anand, S., Reddy, S., Sharma, N., Sidhu, G. S., Panagopoulos, A., Pattan, V., Kashyap, R., & Bansal, V. (2022). Association of obesity with COVID-19 severity and mortality: An updated systematic review, meta-analysis, and meta-regression. *Frontiers in Endocrinology, 13*, 780872. https://doi.org/10.3389/fendo.2022.780872
52. Sood, A. (2009). Altered resting and exercise respiratory physiology in obesity. *Clinics in Chest Medicine, 30*(3), 445–454. https://doi.org/10.1016/j.ccm.2009.05.003
53. Suzukawa, M., Koketsu, R., Baba, S., Igarashi, S., Nagase, H., Yamaguchi, M., Matsutani, N., Kawamura, M., Shoji, S., Hebisawa, A., & Ohta, K. (2015). Leptin enhances ICAM-1 expression, induces migration and cytokine synthesis, and prolongs survival of human airway epithelial cells. *American Journal of Physiology-Lung Cellular and Molecular Physiology, 309*(8), L801–L811. https://doi.org/10.1152/ajplung.00365.2014
54. Vimercati, L., De Maria, L., Quarato, M., Caputi, A., Gesualdo, L., Migliore, G., Cavone, D., Sponselli, S., Pipoli, A., Inchingolo, F., Scarano, A., Lorusso, F., Stefanizzi, P., & Tafuri, S. (2021). Association between long COVID and overweight/obesity. *Journal of Clinical Medicine, 10*(18), 4143. https://doi.org/10.3390/jcm10184143
55. Zhang, J., Zhang, L., Zhang, S., Yu, Q., Xiong, F., Huang, K., Wang, C.-Y., & Yang, P. (2017). HMGB1, an innate alarmin, plays a critical role in chronic inflammation of adipose tissue in obesity. *Molecular and Cellular Endocrinology, 454*, 103–111. https://doi.org/10.1016/j.mce.2017.06.012
56. Zhang, X., Lewis, A. M., Moley, J. R., & Brestoff, J. R. (2021). A systematic review and meta-analysis of obesity and COVID-19 outcomes. *Scientific Reports, 11*(1), 7193. https://doi.org/10.1038/s41598-021-86694-1