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The Immuno-metabolic Lung: Linking Obesity, Inflammation and Respiratory Disease Trajectories

Yagita Aaronson*

Department of Molecular Medicine, University Hospital Federation (FHU) Oncoage, 06204 Nice, France

Introduction

The convergence of metabolic and immune pathways in the context of respiratory health represents a burgeoning field of inquiry. The term "immuno-metabolic lung" encapsulates this intersection, emphasizing how systemic metabolic imbalances, such as those stemming from obesity, can profoundly influence lung physiology and pathophysiology. Obesity, a global epidemic, not only alters adipose tissue function and systemic metabolism but also exerts a considerable impact on immune system dynamics. Emerging research elucidates that obesity-induced chronic low-grade inflammation can exacerbate or even initiate respiratory disorders. Understanding the immuno-metabolic nexus in pulmonary diseases is essential for developing targeted therapies and predictive models that accommodate the complexities introduced by metabolic dysregulation [1].

Obesity is characterized by excessive accumulation of adipose tissue, which acts not only as an energy reservoir but also as an active endocrine organ. Adipose tissue secretes a variety of adipokines and cytokines such as Tumor Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6) and leptin, which collectively contribute to a pro-inflammatory state. This low-grade, chronic inflammation can disrupt immune homeostasis and promote systemic immune activation [2].

Description

Multiple pathways mediate the deleterious effects of obesity on lung health. Mechanical factors, such as increased abdominal fat, reduce lung volumes and impair respiratory mechanics. However, biochemical and immunological factors are increasingly recognized as central to the immuno-metabolic lung phenotype. Obesity-induced inflammation can alter airway reactivity, mucus production and lung tissue remodeling. Adipokines like leptin and adiponectin directly affect lung epithelial cells and immune cell behavior within pulmonary tissues. Moreover, obesity can impair the integrity of the alveolar-capillary barrier, facilitating increased susceptibility to infections and environmental insults. Obesity-related asthma is a distinct phenotype characterized by late onset, lower response to corticosteroids and increased neutrophilic inflammation. Elevated levels of leptin and reduced adiponectin may modulate airway hyperresponsiveness and inflammation. Obstructive Sleep Apnea (OSA) is highly prevalent among obese individuals due to fat deposition around the pharyngeal airway. Intermittent hypoxia in OSA triggers systemic inflammation and oxidative stress, creating a feedback loop that worsens metabolic dysfunction [3].

***Address for Correspondence:** Yagita Aaronson, Department of Molecular Medicine, University Hospital Federation (FHU) Oncoage, 06204 Nice, France, E-mail: aaronsonyagita@ait.fr*

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NF-κB Pathway is key transcription factor is activated by inflammatory stimuli and regulates the expression of various cytokines and adhesion molecules. Obesity enhances NF-κB signaling, promoting lung inflammation. mTOR and AMPK are nutrient-sensing pathways modulate cellular metabolism and immune cell function. mTOR activation in obesity supports pro-inflammatory T cell responses, while AMPK activation has anti-inflammatory effects. Leptin signals through the JAK/STAT pathway, influencing T cell differentiation and cytokine production. Dysregulation of this pathway contributes to chronic inflammation in the lungs. Excess nutrient intake leads to ER stress in both adipose and lung tissues. This stress promotes inflammation via the unfolded protein response (UPR) and can impair epithelial barrier integrity. The gut-lung axis has emerged as a critical mediator in the immuno-metabolic interface. Gut dysbiosis in obesity leads to increased intestinal permeability and systemic translocation of endotoxins (e.g., lipopolysaccharides), which can reach the lungs and activate immune responses. Moreover, short-chain fatty acids (SCFAs) produced by gut microbiota can modulate pulmonary immune responses, highlighting a bidirectional communication system that influences respiratory outcomes [4].

Targeting the immuno-metabolic interface opens new avenues for the treatment and prevention of obesity-associated respiratory diseases. Targeting specific cytokines or signaling pathways (e.g., IL-6 inhibitors, NF-κB blockers) may reduce lung inflammation. Drugs that improve insulin sensitivity (e.g., metformin) or reduce ER stress (e.g., chemical chaperones) may have dual benefits in managing systemic and pulmonary inflammation. Probiotics, prebiotics and dietary modifications to restore gut microbiota balance could mitigate systemic inflammation and improve lung health. Lifestyle interventions, bariatric surgery and pharmacological treatments aimed at weight reduction have shown improvements in asthma control and lung function. Understanding individual variations in immuno-metabolic responses may allow for tailored interventions, combining genomics, metabolomics and microbiomics. The low-grade, chronic inflammation can disrupt immune homeostasis and promote systemic immune activation. The resulting milieu is one in which immune cells, such as macrophages, neutrophils and T cells, exhibit altered functionality, potentially exacerbating inflammatory conditions in distant organs, including the lungs [5].

Conclusion

The concept of the immuno-metabolic lung offers a comprehensive framework to understand how metabolic health influences respiratory outcomes. Obesity, through its multifaceted effects on immune regulation and inflammation, significantly alters the trajectory of various lung diseases. Integrating metabolic profiling into respiratory medicine could enhance diagnostic precision, inform personalized treatments and improve patient outcomes. As global obesity rates continue to climb, addressing the immuno-metabolic underpinnings of lung disease is not only a scientific imperative but also a public health priority.

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Conflict of Interest

None.

References

1. Park, Min-Jung, Sung-Hee Lee, Eun-Kyung Kim and Eun-Jung Lee, et al. "Interleukin-10 produced by myeloid-derived suppressor cells is critical for the induction of Tregs and attenuation of rheumatoid inflammation in mice." *Sci Rep* 8 (2018): 3753.

2. Liu, Cui Hua, Haiying Liu and Baoxue Ge. "Innate immunity in tuberculosis: Host defense vs. pathogen evasion." *Cell Mol Immunol* 14 (2017): 963-975.

3. Brighenti, Susanna and Diane J. Ordway. "Regulation of immunity to tuberculosis." *Tuberculosis and the Tubercle Bacillus* 4 (2017): 73-93.

4. Olson, Oakley C., Hyunjung Kim, Daniela F. Quail and Emily A. Foley, et al. "Tumor-associated macrophages suppress the cytotoxic activity of antimitotic agents." *Cell Rep* 19 (2017): 101-113.

5. De Palma, Michele and Claire E. Lewis. "Macrophage regulation of tumor responses to anticancer therapies." *Cancer Cell* 23 (2013): 277-286.

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