CHEST

Beyond BMI
Obesity and Lung Disease

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The worldwide prevalence of obesity has increased rapidly in the last 3 decades, and this increase has led to important changes in the pathogenesis and clinical presentation of many common diseases. This review article examines the relationship between obesity and lung disease, highlighting some of the major findings that have advanced our understanding of the mechanisms contributing to this relationship. Changes in pulmonary function related to fat mass are important, but obesity is much more than simply a state of mass loading, and BMI is only a very indirect measure of metabolic health. The obese state is associated with changes in the gut microbiome, cellular metabolism, lipid handling, immune function, insulin resistance, and circulating factors produced by adipose tissue. Together, these factors can fundamentally alter the pathogenesis and pathophysiology of lung health and disease.

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The world is facing an unprecedented obesity epidemic, with the highest prevalence occurring in developed countries. More than one-third of the US adult population is obese and another one-third is overweight,[1] but most troubling is that 32% of US children and adolescents are either overweight or obese.[2] Obesity is a major risk factor for the development of a number of respiratory diseases, including asthma, pulmonary hypertension, sleep apnea, obesity hypoventilation syndrome, pneumonia, and ARDS,[3] and it complicates the pathogenesis of other diseases such as COPD. These outcomes occur because obesity profoundly alters normal lung homeostasis through a combination of mass loading and hormonal, metabolic, inflammatory, neurologic, and dietary factors.

The present review highlights some of the major findings that have advanced our understanding of mechanisms linking obesity, as well as its associated metabolic disorders, to common lung diseases.

The Mechanical Effects of Obesity on Lung Function

Obesity directly alters the mechanical properties of the lungs and chest wall through the accumulation of fat in the mediastinum and in the abdominal and thoracic cavities.[4] This action elevates the diaphragm and also limits its downward excursion, causing pleural pressure to increase[5] and functional residual capacity to decrease (Fig 1).[6] These effects are substantial: functional residual capacity is

ABBREVIATIONS: SCFA = short-chain fatty acid; TNF-α = tumor necrosis factor-alpha; Treg = regulatory T cell

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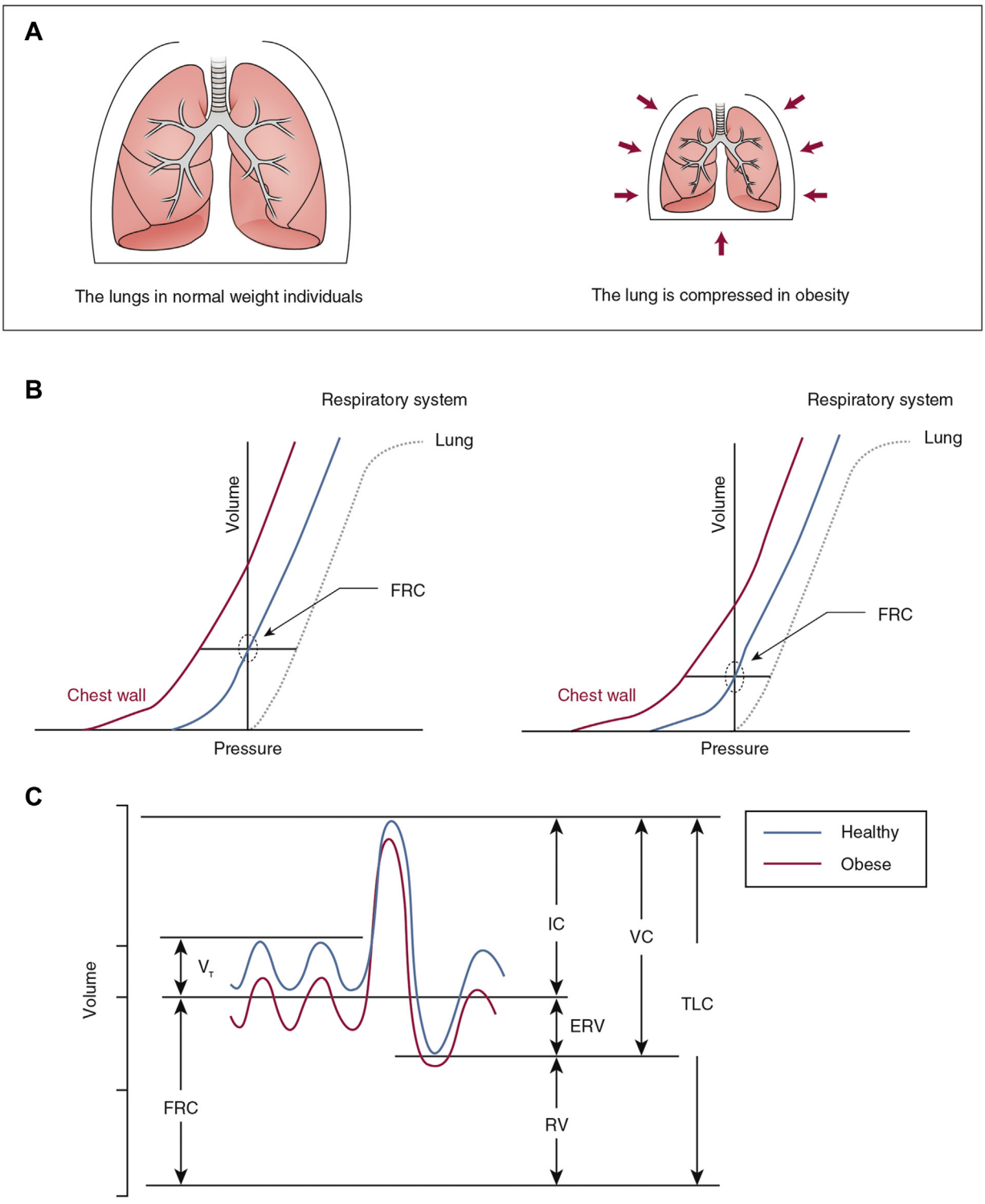

Figure 1 – *A-C, Mechanical effects of lung compression in obese individuals compared with normal-weight individuals. The volume of the chest compartment is invariably decreased in obesity (A), which lowers the operating volume of the lungs (B). Consequently, the FRC and the ERV are reduced, but all other subdivisions of lung volume are remarkably well preserved (C). ERV = expiratory reserve volume; FRC = functional residual capacity; IC = inspiratory capacity; TLC = total lung capacity; VC = vital capacity; V_T = tidal volume.*

reduced by 10%, 22%, and 33% in overweight, mildly obese, and severely obese subjects without asthma, respectively. The mechanical effects of obesity are also reflected in significant decrements in the compliance of the lungs, chest wall, and entire respiratory system,[7] which likely contribute to the