The air we breathe: a study into the impact of historical socioeconomic changes on the respiratory health of past Dutch populations (ca. 470-1850 CE)

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CHAPTER 2

Clinical background and paleopathology of respiratory disease

Building on previous paleopathological research on respiratory disease, this study centers on three specific conditions: chronic maxillary sinusitis, infectious middle ear disease, and pleural inflammation. To better understand differences in respiratory disease prevalence rates in archaeological populations, a thorough understanding of modern clinical data on each of these conditions is essential. Thus, the next three sections provide a clinical background on sinusitis, infectious middle ear disease, and pleural inflammation, respectively. Each section covers the anatomy of the organs and tissues affected by inflammation, the pathogenesis and etiology of each disease, as well as the clinical evidence for inflammation-related bone changes. Finally, this chapter concludes with a concise overview of existing bioarchaeological research on respiratory diseases, establishing the foundation for the analytical framework of this dissertation.

2.1. Chronic maxillary sinusitis

In the clinical practice, maxillary sinusitis is defined as the inflammation of the mucous membrane lining the maxillary sinuses (Slavin et al., 2005, S21). Symptoms are varied, but typically include nasal congestion, headache, cough, and purulent nasal discharge. However, depending on the etiology and complications, they may also include fever, fatigue, dental pain, facial pressure, and/or earache (Brook, 2009). Epidemiological studies currently report a global prevalence of chronic sinusitis (defined as symptoms lasting 8 weeks or longer) ranging from 5% to 12% (de Loos et al., 2019; Slavin et al., 2005). In the Netherlands specifically, approximately 500,000 individuals are diagnosed with chronic sinusitis each year, making it one of the most prevalent health conditions among the Dutch population (KNO, 2010).

2.1.1. Anatomy of the sinuses

The paranasal sinuses consist of eight air-filled cavities located on both sides of the facial region of the skull whose primary function is to act as the first line of defense against potential pathogens (Figure 2.1) (Rosen & Pletcher, 2016). The inner surface of each sinus is lined with the mucosa (or mucous membrane), a layer of ciliated soft tissue interspersed by goblet cells producing mucus (Brook, 2009; Mularczyk & Welch, 2024). The main function of mucus (a thin liquid consisting of water, salts, and immune cells) is to trap pathogens and particulate matter inhaled during breathing. If the paranasal sinus system functions properly, both mucus and any trapped particles are then effectively drained into the nasal cavity through a small, tubular opening known as the ostium (Brook, 2009).

The maxillary sinus, the largest of the paranasal sinuses, is situated bilaterally within the maxillary bone. The maxillary bone forms the majority of the sinus cavity, with the exception of the roof, which is formed by the orbit floor (Mularczyk & Welch, 2024). Unlike the frontal and sphenoid sinuses, the maxillary sinus is present at birth and keeps increasing in size until approximately the age of 18 years, expanding vertically, horizontally, and anteroposteriorly (Lorkiewicz-Muszyńska et al., 2015).

The growth of the maxillary sinus occurs unevenly during childhood. The first phase of rapid development lasts for 2–3 years after birth, followed by a slower growth phase that extends until approximately 7–8 years of age and into puberty (Lorkiewicz-Muszyńska et al., 2015). Once the maxillary sinus is fully formed (i.e., it has reached a volume of ca. 15-20mL), remodeling and expansion of the sinus floor into the alveolar bone of the maxilla may continue, particularly in response to tooth loss (Brook, 2009).

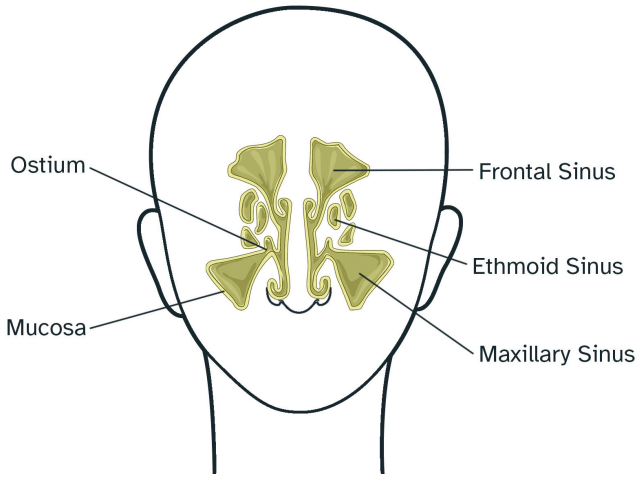

Figure 2.1. Diagram showing the coronal view of the paranasal sinuses. Aspects of diagrams adapted from Servier Medical Art library. Licensed for use by CC BY 4.0.

2.1.2. Pathogenesis and etiology

From a physiological standpoint, sinusitis develops when the ostium becomes blocked, hindering the proper drainage of mucus. The ostium of the maxillary sinus is particularly susceptible to blockage, as its position high on the sinus medial wall hinders gravitational drainage (Ah-See & Evans, 2007; Wald, 1995). The accumulation of mucus creates an ideal environment for the proliferation of bacteria and viruses, which in turn exacerbates inflammation of the mucosa and, consequently, the underlying bone surface (Dykewicz & Hamilos, 2010; Kocak et al., 2002; Slavin et al., 2005).

Inflammation of the mucosa (commonly triggered by a viral or bacterial upper respiratory tract infection or an allergic reaction) is one of the most common causes of ostial blockage, as the swollen tissue resulting from inflammation can lead to a narrowing of the ostium (Brook, 2009; Dykewicz & Hamilos, 2010; Slavin et al., 2005). Various other factors can predispose an individual to mucosal inflammation or ostium obstruction, such as craniofacial anomalies (e.g., cleft palate), physiological variations, nasal blockages (e.g., polyps and tumors), nasal injuries or fractures, cystic fibrosis, inherited and/or acquired immune deficiencies, specific infectious diseases

(e.g., leprosy and tuberculosis), and asthma (Choi et al., 2012; Kaya et al., 2017; Le et al., 2016). While determining the impact of most of these factors on historical sinusitis prevalence rates is challenging, according to Lewis and colleagues (1995) many of these factors (e.g., congenital disease, tumors, immune deficiencies) are expected to have had minimal influence, as individuals suffering from such conditions were unlikely to survive into adulthood. Conversely, it is likely that pre-existing upper respiratory infections, allergies, and poor air quality may have been among the greatest contributors to sinusitis in certain time periods (Davies-Barrett, 2018).

In the case of poor air quality, it is well established today that airborne particulates and pollutants can irritate the mucosa and, subsequently, trigger inflammation (e.g., Leland et al., 2022; Trevino, 1996). This not only includes chemicals from industrial activities, but also tobacco smoke and the domestic burning of biomass fuels, both of which have been associated with chronic sinusitis (Hoover et al., 1997; Lieu & Feinstein, 2000). Additionally, environmental factors such as inadequate ventilation of living and/or working spaces, overcrowding, low humidity, and poor hygienic conditions increase the risk of sinusitis due to heightened exposure to viral and bacterial pathogens (e.g., Dykewicz & Hamilos, 2010; Hoover et al., 1997).

Another common cause of sinusitis is the invasion of the sinus by pathogens originating from dental infections (i.e., odontogenic sinusitis) (Patel & Ferguson, 2012). The roots of the permanent maxillary molars lie just beneath the floor of the maxillary sinus, separated by only a thin layer of bone. This close proximity allows dental infections to easily spread into the sinus cavity, leading to maxillary sinusitis (Patel & Ferguson, 2012). Nowadays, most odontogenic sinusitis cases are attributed to complications arising from modern dental healthcare procedures (Arias-Irimia et al., 2010). However, dental caries can still result in sinusitis by infecting the dental pulp and causing inflammation of the surrounding tissues (Mehra & Jeong, 2009). Chronic abscesses may also lead to resorption of the maxillary bone between the tooth apex and the sinus, forming a direct pathway for infection known as oroantral fistula (Mehra & Jeong, 2009).

2.1.3. Bone involvement

The first clinical evidence of bone involvement in maxillary sinusitis in humans was presented by Tovi and colleagues (1992), who documented a series of histological case studies highlighting a significant periosteal reaction and bone formation within some of the sinuses they analyzed. Most recently, several reports of bone involvement in chronic sinusitis have come from observations of sinus wall thickening through computed tomography, leading to the development of clinical scoring systems which evaluate the extent of bone involvement (Georgalas, 2013; Leung et al., 2016; Snidvongs et al., 2013).

Table 2.1. Skeletal changes categories considered as evidence of chronic sinusitis, as presented by Boocock and colleagues (1995, denoted by *) and Merrett and Pfeiffer (2000, denoted by †). Adapted from Davies-Barrett (2018, Table 3.1; 3.2).

Skeletal change category	Description
Pitting*	Fine clusters of pits (i.e., bone resorption) often found in association with other types of bone change.
Spicules*	Small outgrowths of bone (2-4 mm) located on top of the periosteal surface of the sinus. Clusters of spicules are common and often observed in the form of stellate plates of bone of varying dimensions. These are among the most frequently observed bone changes in previously published bioarchaeological studies on sinusitis.
Remodeled spicules*	Remodeling of spicules into the sinus walls.
White pitted bone*	Localized areas of bone exhibiting extensive pitting, characterized by a white coloration.
Plaque†	Layers of smooth or dense, yet porous bone, with varying thickness.
Lobules†	Smooth, rounded masses of bone located on the periosteal surfaces of the sinus.
Cysts†	Hemispherical depressions in the bone presenting a smooth interior surface and a smooth rim of bone deposition.

Paleopathologists, on the other hand, have examined a broader spectrum of skeletal evidence, including bone resorption, minor bone formations on the sinus surface, and more pronounced alterations in sinus wall morphology (Table 2.1) (Boocock et al., 1995; Merrett & Pfeiffer, 2000). While most of these criteria lack a solid clinical foundation, the presence of spicule bone formations (Figure 2.2, B) has been recently identified as a possible evidence of heterotopic mucosal ossification[2] (Ahn et al., 2017; Meyers et al., 2019). Moreover, it is likely that the pronounced periosteal reaction identified by Tovi and colleagues (1992) is a primary factor contributing to the thickening of the sinus walls noted in various radiographic studies (e.g., Georgalas, 2013; Leung et al., 2016). This type of bone formation (encompassing the bioarchaeological categories of 'white pitted bone' and 'plaque') (Figure 2.2, C) is possibly indicative of a subperiosteal reaction stemming from inflammation, a phenomenon commonly observed in other regions of the skeleton in paleopathology (Ortner, 2003).

2 Heterotopic ossification is a pathological process with varying levels of morbidity, characterized by the abnormal formation of bone in muscle and soft tissues. In clinical settings, the term heterotopic ossification refers to bone development at any location outside the skeletal system, including sites such as muscles, tendons, ligaments, subcutaneous tissue, skin, vascular walls, or any other connective tissues (Ahn et al, 2017; Meyers et al., 2019).

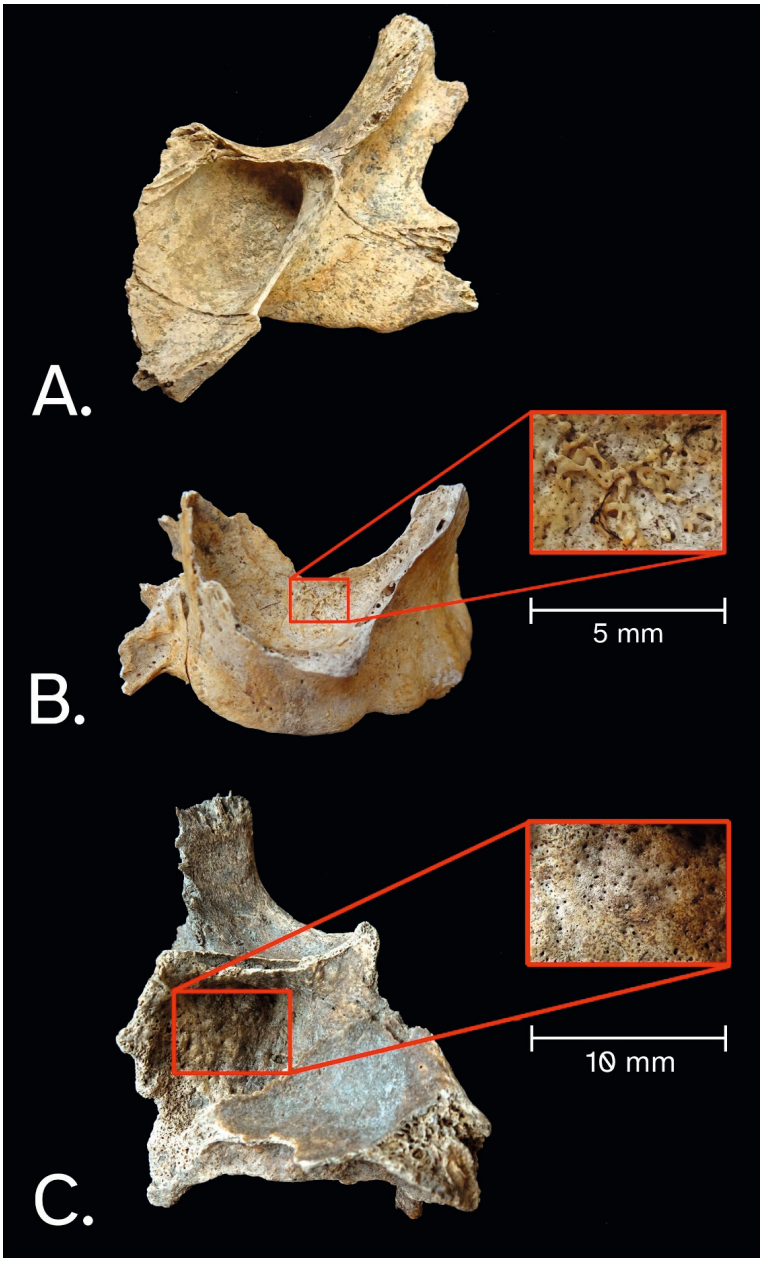

Figure 2.2. Maxillary sinus walls, viewed macroscopically. A: Sinus showing no pathological alteration. Note the smooth walls and the presence of a vascular channel impression on the inferior portion; B: Bone spicules on the medial wall; and C: Thick lamellar bone formation on the distal and anterior sinus walls. Photographs by M. Casna.

2.2. Infectious middle ear disease

Infectious Middle Ear Disease (IMED) is a group of infections (i.e., chronic otitis media, recurrent acute otitis media, and chronic tubal dysfunction) occurring in the middle ear. Today, ear infections are among the most prevalent pediatric conditions, with approximately 80% of children experiencing at least one episode by the age of ten (Schilder et al., 2016). Although primarily considered a childhood condition, IMED is also prevalent in adults. For example, in the Netherlands, acute otitis media alone has an overall incidence of 5.3 cases per 1,000 person-years among individuals aged 15 and older (Rijk et al., 2021). IMED typically manifests with symptoms such as ear pain, fever, fluid drainage from the ears, reduced hearing, and, in some instances, temporary deafness (Briddell et al., 2018; Rettig & Tunkel, 2018). While these infections often resolve without medical intervention, they can result in various complications, including permanent hearing loss, speech delays, and an increased susceptibility to further respiratory infections (Aslam & Al-Qahtani, 2021).

2.2.1. Anatomy of the middle ear

The middle ear, a partially air-filled structure housed within the temporal bone, is one of three main sections of the ear, along with the outer and inner ear (Figure 2.3). It extends from the tympanic membrane to the lateral wall of the inner ear, with its primary function being the transmission of sound vibrations to the cochlea (Mansour et al., 2019).

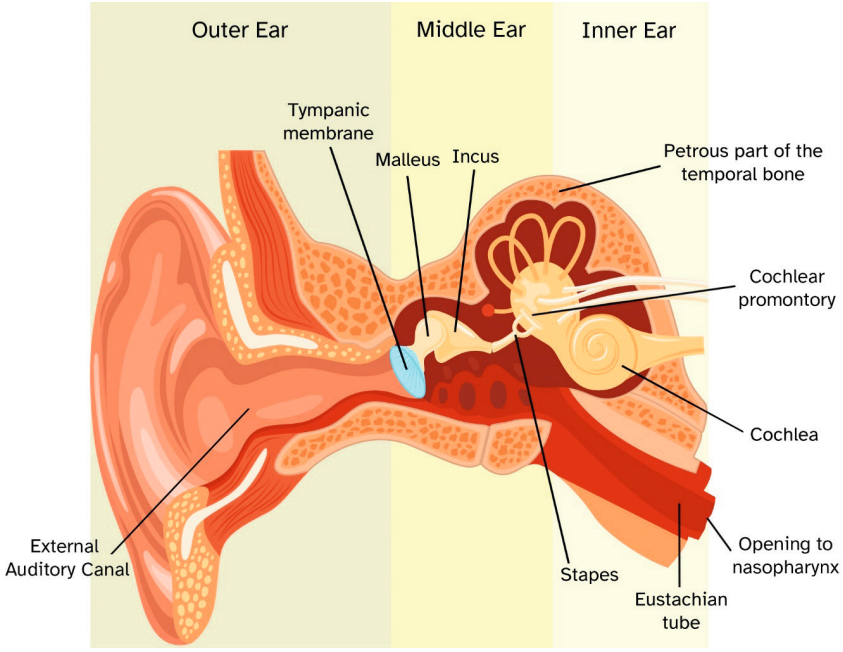

Figure 2.3. Simplified anatomy of the ear. Diagram adapted from macrovector / Freepik.

The middle ear mainly consists of the tympanic cavity, located medially to the tympanic membrane and distally to the cochlea. The tympanic cavity contains the ear ossicles (malleus, incus, and stapes) which, oscillating with the vibration of the tympanic membrane, help transmit sound waves to the inner ear (Mansour et al., 2019). Extending from the anterior wall of the middle ear towards the lateral wall of the nasopharynx there is the Eustachian tube, a fibrocartilaginous duct with the function to help equalizing the pressure between the atmosphere and the middle ear, as well as to facilitate fluid drainage (Proctor, 1967). Similar to the sinus walls, the Eustachian tube is lined with a ciliated mucous membrane, which helps to clear mucus from the middle ear by sweeping it toward the nasopharynx (Mansour et al., 2019).

The development of the middle ear starts as early as the fourth week of intrauterine life (Mansour et al., 2019). Most structures within the middle ear (e.g., the ossicles and the tympanic cavity) cease to grow at around 20 weeks in-utero, when full adult proportions are attained (Mansour et al., 2019). The Eustachian tube, in contrast, continues to grow after birth up until early adolescence, extending in length and gradually shifting diagonally to accommodate craniofacial development (Magro et al., 2021; Proctor, 1967).

2.2.2. Pathogenesis and etiology

According to clinical literature, IMED is closely linked to upper respiratory tract infections (e.g., common colds, sinusitis, and pharyngitis), with the latter being necessary for the initial onset of the former (Thornton et al., 2020). In fact, infections of the upper respiratory tract can lead to swelling of the Eustachian tube and/or the surrounding membranes, impeding fluid drainage and providing a favorable environment for viral and bacterial growth, leading to tissue inflammation (Rijk et al., 2021; Schilder et al., 2016).

The etiology of IMED is highly similar to that of sinusitis (Wald, 2011). Predisposing factors may include Eustachian tube dysfunction, immunodeficiencies, anatomical abnormalities of the palate, and chronic vitamin deficiencies (Cetinkaya & Topsakal, 2023). Furthermore, in children specifically, a shorter, horizontally-oriented Eustachian tube can impede fluid drainage in case of swelling, contributing to a higher incidence of ear infections among pediatric populations (Massa et al., 2015). The living environment is also considered to be a major impacting factors on the development of IMED. Chronic exposure to air pollution, particularly nitrogen dioxide produced by the combustion of fuels such as coal, can alter the middle ear mucosa and cause the widening of vascular spaces (i.e., swelling of the Eustachian tube walls), potentially obstructing normal fluid drainage (Lee et al., 2023). Additionally, air pollutants have been observed to decrease ciliary action, further impairing fluid clearance in the middle ear (Park et al., 2014).

2.2.3. Bone involvement

In IMED, bone involvement is primarily linked to inflammation in the tympanic cavity and to the accumulation of fluid following episodes of infection and inflammation (e.g., Floreanova et al., 2020; Friedmann, 1957; Wiatr et al., 2020). In past osteoarchaeological studies, these changes have often been referred to as otitis media (e.g., Casna et al., 2023; Goycoolea et al.,

2019; Krenz-Niedbała & Łukasik, 2017; Qvist & Grøntved, 2001). However, because any observed changes within the ear canal are inherently non-specific, it should be noted that it is not possible to identify the exact type of ear infection that affected an archaeological individual. Therefore, the term IMED is more appropriate in this context.

Bone involvement in IMED has been extensively studied in both clinical and archaeological contexts, with various studies focusing on different bony structures (e.g., ear ossicles, mastoid cells, tympanic canal, cochlear promontory) (e.g., Casna et al., 2024; Friedmann, 1957; Krenz-Niedbała & Łukasik, 2017; Primeau et al., 2018). The majority of these studies focus on the tympanic cavity, and are clinically associated with the formation of cholesteatoma, a destructive, non-cancerous yet expansive growth in the middle ear, linked to chronic episodes of IMED (e.g., Jung & Chole, 2002; Kärjä et al., 1976; Wiatr et al., 2020). The chronic inflammation associated with cholesteatoma triggers the release of both cytokines (signaling proteins that help control inflammation) and osteoclasts, ultimately leading to bone resorption within the tympanic cavity (Jung & Chole, 2002). This process has been observed to actively impact the ear ossicles, resulting in a range of consequences that can significantly influence the patient's symptoms and clinical outcomes (Kärjä et al., 1976; Wiatr et al., 2020).

In palaeopathology, the erosion of the ear ossicles has been the primary focus of most studies investigating ear infections (e.g., Collins, 2019; Krenz-Niedbała & Łukasik, 2017, 2020; Qvist & Grøntved, 2000). Bone changes have been observed to vary, ranging from small pits affecting less than 25% of the bone surface to extensive pathological alterations covering the majority of the ossicular surface (Figure 2.4).

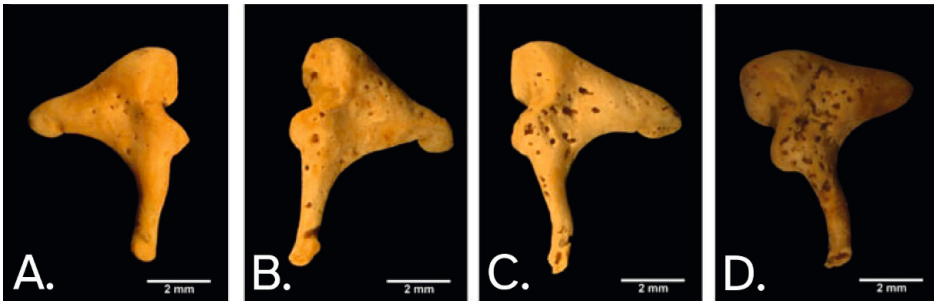

Figure 2.4. Classification of severity of ear bones erosion as presented by Krenz-Niedbała and Łukasik (2017, Figure 2). A: No erosion present; B: Small pits affecting less than 25% of the bone surface; C: Pits present in 25–50% of the affected bone area; and D: More than half of the bone surface pathologically altered. Adapted from Krenz-Niedbała and Łukasik (2017). License Number 1532926-1.

Although the observation of ear ossicle erosion has proven to be a reliable and straightforward method for analyzing IMED in past populations, it is important to acknowledge that ossicles are frequently absent from archaeological skeletal remains. Additionally, their delicate nature makes them highly susceptible to taphonomic damage, further complicating their preservation and

analysis (Bruintjes, 1990; Ortner, 2003). For this reason, recent bioarchaeological research has shifted focus towards changes to other bony structures within the tympanic cavity as potential indicators of IMED. One such structure is the cochlear promontory, located on the medial wall of the tympanic cavity, which has been increasingly recognized for its diagnostic potential in the study of ear infections in past populations (Figure 2.5) (Casna, et al., 2024; Collins, 2019; Floreanova et al., 2020; Roumelis, 2007). Although clinical data on the involvement of the cochlear promontory during chronic episodes of IMED remains limited, some evidence suggests that bone changes in this region may be associated with the presence of cholesteatoma, in a manner similar to the pathological alterations observed in ear ossicles (e.g., McGinn & Chole, 1991). Most recently, bony alterations of the cochlear promontory as presented by Floreanova and colleagues (2020) were recently analyzed in relation to hypopneumatization of the mastoid process (an indicator of childhood IMED)[3] in 50 archaeological individuals (Casna et al., 2024). The findings revealed a significant correlation between mastoid hypopneumatization and promontory bone changes, further reinforcing the role of the cochlear promontory in chronic IMED episodes (Casna et al, 2024).

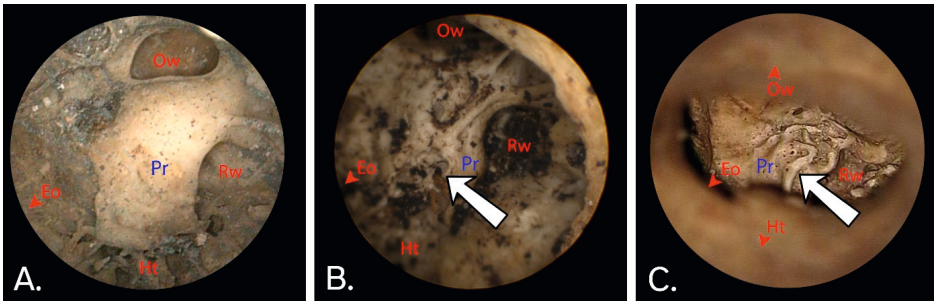

Figure 2.5. Endoscopic view of the cochlear promontory (Pr) as seen by Casna and colleagues (2024, Figure 1). Surrounding structures were labelled for anatomical reference: Ht = Hypotympanum; Eo = Eustachian orifice; Rw = Round window niche; Ow = Oval window. A: No remodeling is present; B: Disruption of the promontory surface integrity in the form of bone erosion (white arrow); and C: Bone growth in the form of bony spicules formation located superiorly from the round window. Licensed for use by CC BY 4.0.

2.3. Pleural inflammation

Pleural inflammation is clinically defined as the inflammation of the pleurae, the membranes that surround the lungs and line the chest cavity (Broaddus, 2022). It is among the most common complications of lower respiratory tract infections, and typically manifest as sharp chest pain while breathing, with associated shortness of breath, cough, and possibly fever (Wrightson &

3 Mastoid pneumatization refers to the process by which air cells develop within the mastoid portion of the temporal bone, creating a system of air-filled spaces connected to the middle ear. This process only occurs during childhood and stops permanently in cases of chronic middle ear infections, resulting in the permanently incomplete development of these air cells (Aoki et al., 2010; Mansour et al., 2019).

Maskell, 2012). Other causes include pneumonia, pulmonary embolism, autoimmune disorders, and lung cancer (Fenster et al., 2022).

2.3.1. Anatomy of the lower respiratory tract

The lower respiratory tract begins at the larynx and continues through the trachea, which divides into the left and right bronchi to supply air to the lungs (Figure 2.6) (Gibson, 2003). The main bronchi branch into smaller divisions, which extends to the bronchioles, leading into the lung parenchyma, were the alveolar ducts are housed (Gibson, 2003). Aside from the mediastinum (which contains structures like the heart and esophagus), the lungs occupy most of the thoracic cavity (Marieb & Hoehn, 2008). The apex of each lung is located beneath the clavicle, while the base rests on the diaphragm (Gibson, 2003).

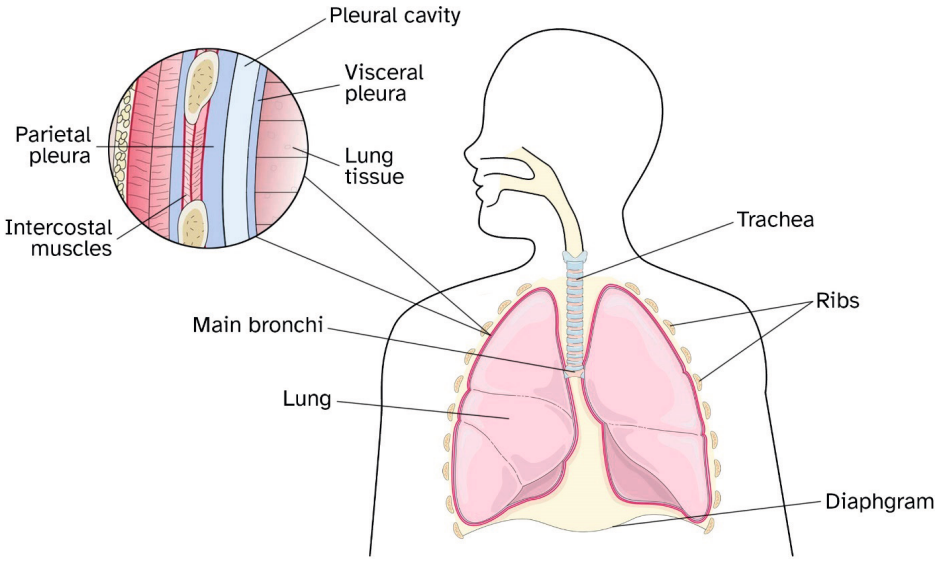

Figure 2.6. Simplified diagram of the lower respiratory tract, featuring a cross-sectional view of the thoracic wall (upper-left corner) that highlights the relevant anatomical relationships between the ribs, pleural membranes, and lungs. Aspects of the diagram adapted from Freepik and Servier Medical Art library. Licensed for use by CC BY 4.0. Cross-sectional anatomy of the thoracic wall reproduced from Hicks (2000, Figure 9.4). License Number 1537985-1.

In living individuals, the lungs are encased in a serous membrane known as the visceral pleura, which closely adheres to their surface. This is paralleled by the parietal pleura, a corresponding membrane that lines the inner chest wall (Broaddus, 2022). Between these two layers lies the pleural cavity, a fluid-filled space that facilitates smooth movement, enabling the lungs to expand and contract seamlessly within the chest during breathing (Broaddus, 2022). Overlaying the parietal pleura are the visceral surfaces of the ribs and portions of the intercostal muscles, which play a crucial role in expanding and contracting the chest cavity during breathing (Marieb & Hoehn, 2008).

2.3.2. Pathogenesis

As previously discussed, pleural inflammation can arise from various pathological processes. When linked to infections in the lungs (e.g., pneumonia, bronchitis), pleural inflammation occurs due to the accumulation of inflammatory cells and fluid within the alveoli, which mobilize to neutralize pathogens (Wilkinson & Woodhead, 2004). The spread of pathogens and inflammatory cells from the lung tissue to the pleura initiates inflammation, often resulting in pleural effusion (i.e., an abnormal accumulation of fluid within the pleural cavity) (Kroegel & Antony, 1997).

Pleural effusion and pleural inflammation are clinically closely linked, as inflammation of the pleural membranes often leads to the accumulation of excess fluid in the pleural cavity, with the effusion further exacerbating the inflammatory response by creating pressure and impairing lung function (Broaddus, 2022). In fact, pleural effusion is a frequent complication of various lung infections, particularly in pneumonia (where it occurs in about 50% of cases), and tuberculosis (Broaddus & Light, 2022; Roy et al., 2021). In tuberculosis specifically, the effusion and inflammation are thought to result from the rupture of a subpleural granuloma,[4] releasing tuberculous proteins into the pleural cavity and provoking an immune response (Kroegel & Antony, 1997). Among non-infectious causes, congestive heart failure is a common contributor, as increased pulmonary capillary pressure forces pulmonary fluid into the pleural space, typically affecting both sides of the chest (Broaddus & Light, 2022). Similarly, pleural effusion (and subsequent inflammation) can develop in up to 50% of patients with pulmonary embolism due to the heightened permeability of lung capillaries, leading to fluid migration into the pleural space (Light, 2013). Neoplastic-related pleural effusions are also prevalent, accounting for 22% of cases, often originating from metastases of cancers such as lung, breast, gastrointestinal, genitourinary, or lymphoma (Gonnelli et al., 2024). In cases of malignant pleural effusion, fluid accumulation and associated inflammation result from cancerous obstruction of parietal lymphatic channels, which impairs lymphatic drainage and increases pleural fluid production (Fashoyin-Aje & Brahmer, 2020; Gonnelli et al., 2024).

2.3.3. Etiology

As pleural inflammation can result from a wide range of diseases, its etiology is complex and multifactorial. Factors such as age, infectious disease prevalence rates, access to prior healthcare, and the geographic or demographic context of the population significantly influence its underlying causes (Fashoyin-Aje & Brahmer, 2020; Light, 2013; Wrightson et al., 2012). However, similarly to sinusitis and IMED, exposure to particulate air pollution, both indoor and outdoor smoke, and overcrowded living conditions have been shown to increase the likelihood of developing infectious diseases commonly associated with pleural inflammation, such as

4 The human body's typical immune response to tuberculosis infection involves the recruitment of macrophages and other immune cells to encapsulate the mycobacterial foci that have adhered to alveolar cells. This process leads to the formation of granulomas, i.e. cellular masses that serve to isolate, rather than eliminate, the bacteria from the rest of the system (Guirado & Schlesinger, 2013; Walter & Daley, 2012).

pneumonia, tuberculosis, and cancer (Kyung & Jeong, 2020; van der Poll & Opal, 2009; Wilkinson & Woodhead, 2004).

Both indoor and outdoor air pollution have been extensively studied in the field of respiratory medicine, with sufficient data now available to detail the specific physiological impacts of various pollutants on the respiratory system (e.g., Balmes & Holm, 2022; Kuschner & Blanc, 2022). In most cases, air pollution has been widely observed to exacerbate asthmatic symptoms (Tran et al., 2023). Furthermore, individuals with pre-existing respiratory conditions have been observed to exhibit increased susceptibility to infections, as pollutants can impair both lung development and overall respiratory function (Maung et al., 2022). Chronic exposure to particulate air pollution can also induce cellular changes in the airways, increasing the risk of lung cancer (Almetwally et al., 2020). It is estimated that approximately 16% of lung cancer deaths are attributable to air pollution, making it among the greatest contributors to the global burden of respiratory diseases (WHO, 2016).

2.3.4. Bone involvement

In clinical reports of lower respiratory tract and lung disease, the presence of new bone formation on the visceral surfaces of ribs has been only sporadically linked to pleural inflammation (e.g., Asnis & Niegowska, 1997; Eyler et al., 1996; Guttentag & Salwen, 1999; Simon, 1973). Studies analyzing skeletal remains with known causes of death have revealed a significant correlation between new bone formation on the visceral surfaces of ribs and pulmonary tuberculosis (e.g., Matos & Santos, 2006; Roberts et al., 1994; Santos & Roberts, 2001, 2006). However, similar lesions were also identified in 15–36% of individuals who died from other pulmonary conditions (e.g., pneumonia, bronchitis, and emphysema), suggesting that the bone formation may have a more non-specific origin, rather than being exclusively linked to a single disease (Davies-Barrett et al., 2019). As previously discussed, chronic exposure to particulate pollution can lead to interstitial lung disease, which may trigger ongoing inflammation and fibrosis (i.e., the development of fibrous connective tissue in response to persistent inflammation) in the lungs (Gold et al., 2000; Ross & Murray, 2004; Sirajuddin & Kanne, 2009). According to Davies-Barrett and colleagues (2019) this process may, in return, trigger the formation of new bone on the surface of the ribs in direct contact with the parietal pleura.

Building on this hypothesis, Davies-Barrett and colleagues (2019) conducted a systematic investigation of new bone formation on the visceral surface of ribs in archaeological skeletal remains, developing a method to distinguish inflammatory bone formation from other types of bony alterations (e.g., fracture calluses, soft tissue attachment markers, and normal anatomical variations). Their study identified key characteristics of bone formation associated with pleural inflammation, including irregular, diffuse woven bone patches and porous remodeling of lamellar bone deposited atop the original cortical surface (Figure 2.7) (Davies-Barrett et al., 2019). The criteria established by Davies-Barrett et al. (2019) have since become a standardized method for recording pleural inflammation in archaeological skeletal remains, providing a consistent framework for diagnosing this condition in past populations.

Figure 2.7. Visceral surfaces of ribs, viewed macroscopically. A: Rib showing no pathological alteration.; and B: Inflammatory periosteal reaction in the form of woven bone formation on the rib neck and angle. Photographs by M. Casna.

2.4. The bioarchaeology of respiratory disease

Over the past two decades, the bioarchaeological investigation of respiratory disease has expanded significantly, with an increasing number of publications featuring both case studies and population-level analyses (e.g., Coutinho-Nogueira et al., 2022; Goycoolea et al, 2019; Lee et al., 2024; Riccomi et al., 2021; Vilumets et al., 2024). In archaeological populations, the increased occurrence of the bone changes discussed in this chapter is usually interpreted as a sign of prolonged exposure to irritants, such as particulate matter from indoor fires (e.g., Sundman & Kjellström, 2013), industrial emissions (e.g., Boyd, 2020), or overcrowded living environments (e.g., Casna et al., 2021), all of which are characteristic of urbanized societies. Additionally, respiratory diseases have also been used as proxies to explore the impact of climate change on

human health, further highlighting the multifaceted and complex etiology of these conditions (Davies-Barrett et al., 2023).

Decades of bioarchaeological research into respiratory disease have highlighted their unique role in understanding historical living environments (Lee et al., 2024; Roberts, 2016). However, more recently, concerns have been raised about existing interpretations, as findings often appear contradictory across different studies. For instance, when investigating chronic maxillary sinusitis in medieval Yorkshire, Lewis and colleagues (1995) observed that urban populations exhibited higher prevalence rates than their rural counterparts. Similarly, Davies-Barrett and colleagues (2023) reported higher rates of pleural inflammation in urban populations compared to rural groups in the Middle Nile Valley over an extended time period (2500 BCE–1500 CE). However, several other studies comparing urban and rural settings have identified no significant differences in respiratory diseases, indicating that variations in living and working conditions may not have consistently influenced respiratory health across different historical and environmental contexts (e.g., Bernofsky, 2010; Casna et al., 2021; Krenz-Niedbała & Łukasik, 2016). These discrepancies may suggest that, while the bioarchaeological study of respiratory disease offers valuable insights, our understanding of the complex interactions between environmental, social, and biological factors influencing health currently remains incomplete (Betsinger & DeWitte, 2021). As bioarchaeology and paleopathology gain increasing recognition as essential tools for investigating the past alongside historical and archaeological sources, it becomes imperative to broaden the scope of paleopathological analysis by incorporating a wider diversity of contexts in the study of respiratory disease. To contribute to this effort, the following chapters will analyze prevalence rates of respiratory disease in diverse living conditions, encompassing various geographic regions, time periods, and socioeconomic environments. This approach will provide a comprehensive overview of how diverse risk factors influenced different Dutch populations across time and space, enriching our knowledge of the complex interplay between health and environment throughout human history.

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