

Biology 2020, 9, 318 6 of 16 The sinuses are cavities around the nose where inflammations may occur such as acute or recurrent sinusitis. Sinusitis can affect both the ethmoidal, sphenoidal, and frontal sinuses. The sinuses are usually an individual microbiome area but also can not be contaminated by nasal and oral cavity microbiota, usually under accumulation of secretions with the main bacteria involved, *Pneumococcus*, *Haemophilus* and *Streptococcus*. Finally, rhinitis, pharyngitis and tonsillitis caused by a heterogeneous group that includes viruses and bacteria (Hemolytic β streptococcus is the most common cause and covers 15% of cases) [32–37]. Quite often, however, these infections are due to rhinovirus, adenovirus, infectious mononucleosis virus, coronavirus (such as the new pandemic SARS-CoV-2). SARS-CoV-2 in the upper airway tract can lead to the complication of hyposmia/anosmia and hypogeusia/ageusia interacting directly with neural tissues or via the immune system. In fact, according to some hypotheses, such symptoms are linked to the neuronal cells' impairment or to ischemic harm of the central nervous system, but also to an increase in Interleukin-6 [38–42]. Otitis is an acute infection of the middle ear and occurs most often in children and it is mainly due to *Pneumococcus*, *Haemophilus* and *Staphylococcus* [5,22,43]. Several factors can increase or decrease the risk of appearance of a lung disease. The composition of the pulmonary microbiota depends on three main factors: (a) microbial immigration (micro-aspiration, inhalation of microorganisms, direct mucosal dispersion), (b) microbial elimination capacity (cough, muco-ciliary clearance, innate and adaptive immunity), (c) regional growth conditions (nutritional availability, temperature, O₂ tension, local microbial competition, concentration and activity of inflammatory cells). The reduction in the microbial elimination capacity both increases regional growth conditions and creates dysbiosis, and therefore leads to a high risk of lung disease (Figure 2). These modifications facilitate the formation of niches that favor the growth and increase in *Prevotella* and *Veillonella* capable of inducing inflammation in the airways through the production of neutrophils and lymphocytes. They therefore lead to dysbiosis of the microbiota, inflammation, and lung damage. It is assumed that the modified pulmonary microbiota loses its protective capacity and may play a potential role in the pathogenesis of chronic lung diseases, or in asthma, cystic fibrosis, chronic obstructive pulmonary disease, broncho-dysplasia, and idiopathic pulmonary fibrosis [44–47]. Inflammation of the lungs resulting from infection during childhood is associated with the development of asthma. Thus, microbiota dysbiosis of the airways could be the basis for the susceptibility and progression of chronic lung disease. Most newborns first colonize with *Staphylococcus* or *Corynebacterium* before stable colonization with *Alloiococcus* or *Moraxella*: the link between bacterial colonization of the airways in children and the onset of asthma later in life is noted. Infants whose pharynx has been colonized by *Streptococcus pneumoniae*, *Haemophilus influenzae* or *Moraxella catarrhalis* since the beginning of their life have increased asthma risk. These same bacteria are constantly associated with the worsening of asthma, such as COPD. However, exposure to a wider range of germs seems to have a protective effect on the development of asthma in children by activating the innate immune system. This finding will support the hypothesis that asthma caused by a lack of microbial exposure at the beginning of life has consequent effects on the development of the immune system. Epidemiological research has consistently shown that a rich microbial environment in early childhood provides protection against the development of asthma,

suggesting the need to understand the extent and nature of the normal microbiota of the airways. Another study showed that two-month age streptococcal colonization was a strong predictor of asthma later in life