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The correlation between second-hand tobacco smoke exposure and biofilm formation in chronic rhinosinusitis

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Introduction and purpose

Tobacco smoke is a major health concern globally. Due to tobacco epidemic, approximately 8 million people died as a result of cigarette smoking in 2020 alone, where 1.2 million were caused by non-smokers inhaling second-hand smoke. Chronic rhinosinusitis (CRS) is an inflammatory condition that has a significant health and economic impact worldwide. Despite its great burden on the health-care system and patients' quality of life, the variety of therapy options for CRS is currently limited. Tobacco-induced biofilm formation may contribute to the refractory nature of many respiratory diseases reported in smokers and second-hand smokers, due to increased resistance to antibiotics. The aim of this study is to present that exposure to household passive smoking may induce the formation of nasal biofilms.

Brief description of the state of knowledge

Chronic bacterial infections involving biofilms have recently been recognised as a factor in CRS pathogenesis. The presence of biofilms on the mucosa of CRS patients is associated with more severe pre-operative disease, persistent postoperative symptoms, ongoing mucosal inflammation, and infections following endoscopic sinus surgery. Tobacco smoke and CRS

have been associated because of the immunosuppressive and irritating effects of tobacco smoke on sinonasal epithelial cells. Smoking uptake and cessation have been shown to affect microbial communities, with smokers having not just distinct microbial communities, but also a higher frequency of possible pathogens in those communities.

Summary

Biofilms may play a significant role in the development of chronic rhinosinusitis. The impact of tobacco smoke on biofilm development could have major implications not only for CRS but also for other respiratory infections.

Key words: tobacco smoking; tobacco smoke pollution; sinusitis; biofilm; children;

Introduction and purpose

Chronic rhinosinusitis (CRS) is an inflammatory condition that has a significant health and economic impact worldwide (1). According to the European Position Paper on Rhinosinusitis and Nasal Polyps 2020, it is defined as continuing inflammation of the nasal cavity and sinuses for more than 12 weeks (2). Despite its great burden on the health-care system and patients' quality of life, the variety of therapy options for CRS is currently limited. This is largely due to a lack of knowledge of the processes underlying disease pathology. There is also evidence that bacterial biofilms adversely affect treatment outcomes in patients with CRS.

Bacterial biofilms are a complex formation of bacteria that can attach to a variety of biological and non-biological surfaces (3). Bacteria cells are enclosed in a self-produced polysaccharide matrix, and when compared to planktonic bacteria, they have a different phenotype and genotype (4,5). Biofilms are known to be related with various infections, including chronic rhinosinusitis, otitis media, and pneumonia. Worth mentioning is that biofilms have inherent resistance to the human immune system and antimicrobial drugs (4,6).

The effects of smoke on the microbiome have rarely been studied. Increasing data suggests that cigarette smoke encourages the production of biofilms in a variety of pathogenic bacteria (7–10). Tobacco-induced biofilm formation may contribute to the refractory nature of many respiratory diseases reported in smokers and second-hand smokers, due to increased resistance to antibiotics (5).

Tobacco smoke is a major health concern globally. Due to tobacco epidemic, approximately 8 million people died as a result of cigarette smoking in 2020 alone, where 1.2 million were caused by non-smokers inhaling second-hand smoke (11). In 2020, tobacco was used by 22.3 percent of the global population (11). Inhaling smoke or tobacco products

produced by others is known as passive or second-hand smoking (PS) (6). The non-smoker breathes sidestream smoke from the cigarette's burning tip, as well as mainstream smoke inhaled and expelled by the smoker. More than 4000 chemicals, including more than 60 known carcinogens, are emitted into the atmosphere as a result of environmental tobacco smoke (12,13). However, there is currently inadequate knowledge regarding how tobacco smoke induces the formation of bacterial biofilms.

This research employs a non-systematic review and analysis of the scientific literature. Databases including PubMed and Scopus were searched through.

The aim of this study is to present that exposure to household passive smoking may induce the formation of nasal biofilms.

Description of the state of knowledge

Chronic bacterial infections involving biofilms have recently been recognised as a factor in CRS pathogenesis (3,5,14,15). The presence of biofilms on the mucosa of CRS patients is associated with more severe pre-operative disease, persistent postoperative symptoms, ongoing mucosal inflammation, and infections following endoscopic sinus surgery (16–18). Chronic rhinosinusitis recalcitrance fits the characteristics of a biofilm infection. Staphylococcus aureus (S. aureus) plays an important role in CRS due to its virulence and the ability to form biofilms. Because S. aureus colonises the mucosa of the nose and the lower respiratory tract, it will be exposed to cigarette smoke during the course of the disease (14). Biofilms allow bacteria to evade host defences and release planktonic bacteria on purpose, which can lead to implantation and colonisation of new areas (19). Many recent investigations have suggested that bacterial biofilms, in particular staphylococcal biofilms, of the paranasal sinus mucosa may play a role in persisting refractory CRS (19–22). However, the significance of bacteria in the pathogenesis of CRS as a major or aggravating cause is still unclear.

Although much is known about the deleterious consequences of tobacco smoke on humans, little is known about its influence on microbiota, particularly that found in the upper respiratory system (19). The nose and paranasal sinuses are exposed to significantly higher levels of particle pollution than the lower respiratory tract (19). Tobacco smoke and CRS have been associated because of the immunosuppressive and irritating effects of tobacco smoke on sinonasal epithelial cells (6). Bioactive components of tobacco smoke triggered the formation of S. aureus biofilms through an oxidant-dependent mechanism, according to Kulkarni et al. (10). Smoking uptake and cessation have been shown to affect microbial communities, with smokers having not just distinct microbial communities, but also a higher frequency of possible

pathogens in those communities (5,23,24). There is solid evidence that children are especially vulnerable to the effects of SE. It's conceivable that secondhand smoking exposure promotes bacteria adhere better to the nasopharyngeal mucosa in children whose parents smoke (23).

In the work of Antunes et al., it was investigated how the presence of repeated exposure to tobacco smoke influences biofilm formation. For three days, bacterial cultures were repeatedly exposed to cigarette smoke. The production of biofilms was examined 20 hours after each exposure. After 1 exposure to smoke, there was no change in biofilm formation, but the biofilm mass developed after 2 and 3 exposures (5). This study showed that tobacco smoke induces an increase in biofilm mass in vitro.

In study by Lacoma et al., the impact of cigarette smoke on specific virulence phenotypes important in S. aureus pathogenesis was investigated. The findings show that tobacco smoke causes S. aureus to adopt virulence characteristics associated with long-term infection, such as increased biofilm formation, increased invasiveness, and intracellular persistence (25).

In work by Le Shi et al., they found that nicotine can enhance S. aureus biofilm formation. S. aureus established a biofilm with a protective phenotype by changing gene expression in the unfavourable circumstances of tobacco smoke. As a result, the bacteria become encased in a protective biofilm, making them more resistant to elimination (14).

In study by Goldstein-Daruech et al., they reported that repetitive in vitro exposure to whole tobacco smoke induces biofilm formation in bacteria isolated from the sinonasal cavities of patients with CRS (19). Bacterial cultures were obtained from CRS patients, with and without tobacco exposure, demonstrating mucopurulent sinonasal secretions on nasal endoscopic exam. Bacteria were exposed in vitro to the smoke of 5 cigarettes over 3 hours. Bacteria isolated from tobacco exposure patients showed a strong biofilm formation during in vitro tobacco smoke challenge compared to bacteria isolated from non-smoked patients (19). Moreover, bacteria from patients exposed to smoke can revert to a non-biofilm phenotype when not exposed to tobacco smoke (19). Chronic or repeated exposure to smoke would alter the bacteria's ability to form biofilm.

In the study by Elwany et al., they performed identification of biofilms for report about development of nasal biofilms in children exposed to household passive smoking (6). A questionnaire study among parents and an objective study of cotinine in the urine of children confirmed the exposure of children to passive smoking. Of the study group, 11 samples of nasal mucosa developed biofilms at different stages of the biofilm life cycle, which 10 grew Staphylococcus aureus (S. aureus). With longer periods of exposure to cigarette smoke, the

chances of biofilm formation increased. In children who developed nasal biofilms, the average length of passive smoking exposure was 112.6 months. (± 15.6). Children of the study group who developed nasal biofilms were exposed to tobacco smoke for significantly longer duration than children who did not develop biofilms, moreover children of heavy smokers, (those who smoke greater than or equal to 25 or more cigarettes a day) developed nasal biofilms more frequently than other children (6).

The cited studies indicate that exposure to tobacco smoke has an influence on biofilm formation. It seems that changes in bacterial biofilm are directly proportional to the duration of passive smoke exposure. It is worth noting that bacteria's potential to form biofilm can be impaired by chronic or recurrent exposure to smoke. It is hypothesised that cigarette smoke inducted biofilm formation improve bacterial viability and boost adaptability to the harsh nasal environment, thereby contributing to persistent infection. Moreover, biofilm development by tobacco smoke may also contribute to the resistant nature of many respiratory infections in smokers, because biofilm microorganisms have been shown to have higher antibiotic resistance. Middle ear effusion, chronic rhinosinusitis, lower respiratory tract infections, asthma, low birth weight, and sudden infant death syndrome have all been linked to environmental smoke exposure in children. Moreover, in comparison to children whose parents have never smoked, they are more likely to have adenotonsillectomy (26). Because of their rapid breathing rates, children are especially vulnerable to the negative consequences of PS (6,12,27). As a result of exposure to tobacco smoke, active and passive smokers may suffer from an increased incidence of sinus infections due to biofilm formation. As can be seen in the study by Elwany et al.(6), biofilms grew as a result of prolonged tobacco smoke exposure and increasing parental smoking. It is important to pay attention to the dangers of smoking, both for parents who smoke and for their children.

Summary

In some patients, biofilms may play a significant role in the development of chronic rhinosinusitis. The impact of tobacco smoke on biofilm development could have major implications not only for CRS but also for other infections such as otitis media, chronic obstructive pulmonary disease, bronchitis, and pneumonia. However, more research is needed, with a greater number of cases and controls, to fully understand and prove this process. This review should encourage people not to smoke and current smokers to give up smoking, especially if there are children in their environment.

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