Rhinology

The effect of passive smoking on bacterial colonisation of the upper airways and selected laboratory parameters in children

L'effetto del fumo passivo sulla colonizzazione batterica delle alte vie aeree e su determinati parametri di laboratorio nei bambini

G. BUGOVA[1], M. JANICKOVA[2], B. UHLIAROVA[3], R. BABELA[4], M. JESENAK[5]

[1] Department of Otorhinolaryngology, Head and Neck Surgery, Comenius University in Bratislava, Jessenius Faculty of Medicine in Martin, University Hospital, Martin, Slovakia; [2] Department of Stomatology and Maxillofacial Surgery, Comenius University in Bratislava, Jessenius Faculty of Medicine in Martin, Martin, Slovakia; [3] Department of Otorhinolaryngology, FD Roosevelt Faculty Hospital, Banska Bystrica, Slovakia; [4] Institute of Healthcare Disciplines, St. Elisabeth University, Bratislava, Slovakia; [5] Department of Paediatrics, Comenius University in Bratislava, Jessenius Faculty of Medicine in Martin, University Hospital, Martin, Slovakia

SUMMARY

Exposure to tobacco smoke is associated with a higher risk of respiratory tract diseases. The aim of this study was to determine the influence of passive smoking on selected characteristics of children with adenoid hypertrophy. Sixty-one children with adenoid hypertrophy were enrolled in the prospective study. Differences in bacterial colonisation of middle nasal meatus and nasopharynx and changes in selected laboratory immune and inflammatory markers according to the tobacco smoke exposure were analysed. Exposure to tobacco smoke was associated with significantly higher colonisation of pathogenic bacteria and polymicrobial growth of pathogenic bacteria (≥ 2 bacteria) in middle nasal meatus compared to non-exposed children (P = 0.045, P = 0.032, respectively). Identification of pathogenic bacteria in the middle nasal meatus did not correlate with isolation of pathogenic bacteria in the nasopharynx in either group of children. Parameters of humoral immunity in serum, IgA and IgG, were detected at higher concentrations in children exposed to tobacco smoke (P = 0.047, P = 0.031, respectively). Differences in selected parameters of cellular immunity in peripheral blood according to passive smoking were not observed. Tobacco smoke exposure is related to increased colonisation by pathogenic bacteria in middle nasal meatus and elevation of IgA and IgG in peripheral blood, but does not seem to influence markers of cellular immunity parameters in children with adenoid hypertrophy. Avoidance of passive smoking could be recommended as a universal preventive strategy against microbial colonisation of the upper airways and development of various inflammatory diseases in children, e.g. adenoid hypertrophy.

KEY WORDS: Mucosal microbiota • Passive smoking • Pathogenic bacteria • Upper airways • Immunity

RIASSUNTO

L'esposizione al fumo di sigaretta è associato ad un alto rischio di sviluppare malattie del tratto respiratorio. L'obiettivo di questo studio è stato quello di determinare l'influenza del fumo passivo su determinate caratteristiche dei bambini con ipertrofia adenoidea. Sessantuno bambini con ipertrofia adenoidea sono stati arruolati in questo studio prospettico. Sono stati analizzati differenze nella colonizzazione batterica del meato medio e del nasofaringe e cambiamenti di determinati parametri di laboratorio immunologici e di marker dell'infiammazione in relazione all'esposizione al fumo di tabacco. L'esposizione al fumo è stata associata in maniera significativa alla colonizzazione di batteri patogeni e alla crescita polimicrobica di batteri patogeni (≥ 2 batteri) nel meato nasale medio, rispetto ai bambini non esposti (P = 0,045, P = 0,032, rispettivamente). L'identificazioene di batteri patogeni nel meato medio non è stata accompagnata all'isolamento di batteri patogeni nel nasofaringe di entrambi i gruppi di bambini. Parametri sierici dell'immunità umorale, quali IgA e IgG, sono risultati notevolmente più elevati nei bambini esposti (P = 0,047, P = 0,031, rispettivamente). Tuttavia non sono state trovate differenze nei parametri sierici riguardanti l'immunità cellulare. In conclusione l'esposizione al fumo di tabacco sembra essere correlata ad un incremento della colonizzazione da parte di batteri patogeni del meato medio e ad un aumento delle IgA e delle IgG nel sangue periferico, mentre non sembra influenzare i markers dell'immunità cellulare nei bambini con ipertrofia adenoidea. Evitare il fumo passivo dovrebbe essere raccomandato come strategia preventiva universale contro la colonizzazione microbica delle alte vie aeree e lo sviluppo di svariate malattie infiammatorie dei bambini, come ad esempio l'ipertrofia adenoidea.

PAROLE CHIAVE: *Microbiota • Fumo passivo • Batteri patogeni • Alte vie respiratorie • Immunità*

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Introduction

Exposure to environmental tobacco smoke (ETS) is associated with a variety of health effects, including cancer, cardiovascular diseases and/or respiratory illnesses. Tobacco is also a major burden to people who do not smoke. As developing individuals, children are particularly vulnerable to the negative effects of second-hand smoke (SHS). Furthermore, they are unable to influence their own degree of exposure. Worldwide, at least 40% of children are regularly exposed to SHS predisposing them to upper and lower respiratory infections as well as asthma [1 2]. Moreover, long-term exposure to ETS creates a state of permanent inflammation and an imbalance in the lipid profile that leads to lipid accumulation in the blood vessels of the heart and aorta. Children with long-term exposure to ETS may have an elevated risk for the development of premature coronary artery disease [3]. Simonetti et al. [4] found that in healthy children, parental smoking is an independent risk factor for higher blood pressure, adding to other familial and environmental risk factors.

Mucosal microbiota of the upper airways is very important for health and diseases and its development begins suddenly after the birth. Changes in airway microbiota are associated with acute and chronic consequences. There are many positive factors influencing the correct composition of microbiota, e.g. breastfeeding [5]. On the other hand, negative changes of mucosal microbiota may contribute to the development of pathological conditions such as sinusitis, otitis media or chronic airway inflammation [6]. Passive smoking changes the microbial colonisation of the airways [7]. It has been shown that nasopharyngeal microbiota is related to the frequency of upper respiratory infection and sinusitis and can be a determinant for infection spread to the lower airways [6 8 9]. It has been suggested that commensal colonisation may interfere with pathogen colonisation [10]. Adenoids serve as a bacterial reservoir for upper airway infections and their removal is followed by changes in upper airway microbiota and a decrease in the rate of respiratory infections [11].

In our prospective study, we aimed to evaluate the modifying effect of passive smoking on selected parameters in children with adenoid hypertrophy indicated for endoscopic adenotomy due to mechanic upper airway obstruction or recurrent respiratory tract infections. Moreover, we studied the effects of passive smoking on the composition of upper airway mucosal microbiota in the nasopharynx and middle nasal meatus.

Materials and methods

Design of the study

The prospective study was conducted with 61 children divided into 2 groups according to tobacco smoke exposure. All children enrolled in the study were scheduled to endoscopic adenotomy for adenoid hypertrophy (due to mechanic obstruction of upper airways or recurrent respiratory tract infections) at the Department of Otorhinolaryngology, Head and Neck Surgery, Comenius University, Jessenius Faculty of Medicine, University Hospital in Martin, Slovakia.

Children treated with systemic or local antibiotics within the 2 weeks before enrolment, recent respiratory infection, increased level of C-reactive protein and those with recurrent tonsillitis were excluded from the study.

Smoking exposure was recorded according to the questionnaires. Differences in bacterial colonisation of the middle nasal meatus and nasopharynx and changes in humoral and cellular immunity according to tobacco smoke exposure were analysed.

The study was approved by the Ethics Committee of Jessenius Medical Faculty, Comenius University in Martin, Slovakia (EK 1515/2014). An informed consent form was signed by all parents of participating children.

Smoking exposure

Only exposure to parental smoking was recorded as parents were considered to be the closest individuals caring for the child. The level of exposure when smokers were other than the parents (e.g. grandparent or sibling) and parent was a non-smoker could not be evaluated and those cases were excluded from the study. Exposure to smoke other than tobacco smoke was not recorded. All the children were from urban areas.

Cultivation studies

Middle nasal meatus and nasopharyngeal swab specimens were obtained under endoscopic control by using sterile cotton-wool swabs and transported in Stuart's transport medium to the microbiological laboratory within 2 to 4 hours. The swab was inoculated on Sheep blood agar (Columbia Bio-Rad, Bratislava, Slovakia) and Chocolate agar with bacitracin disc, Mac Conckey agar (Bio-Rad, Bratislava, Slovakia), and placed into a 7% CO_2 incubator at 37° C. Plates were examined after 18 to 24 hours of incubation. The incubation was further extended to 48 hours to detect slow-growing microbes. Identification of colonies at a genus or species level was based upon typical colony morphology by subculture, Gram stain, standard

rapid tests (catalase, pyrrolidonyl aminopeptidase - PYR and oxidase tests), identification by latex agglutination tests and biochemical tests. All pathogenic strains were tested for their susceptibility to antimicrobial agents using the agar diffusion method (by EUCAST) with commercial discs (Oxoid).

Examination of immune parameters in peripheral blood

Various parameters and markers of cellular and humoral immunity were evaluated. Venous blood samples were drawn from a peripheral arm vein, collected into an evacuated tube and treated with either EDTA or sodium heparin. The sampling was performed on the day before surgery. White and red components of the blood count were examined by sampling with an 18-parameter haematological ANALYSER Beyer Advia 60 (Siemens AG, Munich, Germany) using the company's reagents. Differential counts of leukocytes, as well as subpopulations of lymphocytes and NK cells, were assessed immediately after sampling. Cells were counted on the flow cytometer FC500 (Beckmann Coulter, Brea, CA, USA) after staining with monoclonal antibodies (Immunotech, Prague, Czech Republic) according to the manufacturer's instructions. A four-colour fluorescent protocol was used with the following composition: $CD4^+CD19$-FITC/$CD8^+CD16^+CD56$-PE/CD3-PC5/CD45-PC7. The absolute counts of all cell populations were calculated from the examined blood count. The following populations, based on the $CD45^+$ leukocyte gate, were quantified from the CD4 vs. side scatter (SS) cytogram: lymphocytes (low SS), T lymphocytes ($CD3^+$), T helpers ($CD3^+CD4^+$), T cytotoxic cells ($CD3^+8^+$), B lymphocytes ($CD19^+$) and NK cells ($CD3\text{-}8^+16^+56^+$). Absolute and relative counts of NK cells were also examined on the cytometer using separate analysis parameters (CD8-FITC/$CD16^+CD56$PE/CD-3PC5/CD45PC7). Serum four immunoglobulin isotypes (IgG, IgA, IgM, total IgE) were also examined.

Statistical analysis

Frequencies of categorical data were tabulated and evaluated with a chi-square test using Yates's correction. For other data, median and interquartile range was calculated and tested with the Kruskal-Wallis or Mann-Whitney tests. The statistical analysis was performed with STATISTICA Cz 10. All conclusions were based on a significance level of $P < 0.05$.

Results

Among the 61 children in this study, 23 (37%) were exposed to SHS (14 male, 9 female, mean age 5.5 ± 3 years, range 2-16). Thirty-eight (63%) children had non-smoking parents (29 male, 9 female, aged 5.2 ± 2.8 years, range 2-16). Bacterial growth was present in 50 of the 61 samples (82%) from the middle nasal meatus and 60 of the 61 samples (98%) from the nasopharynx. Microorganisms isolated from the middle nasal meatus and nasopharynx are presented in Tables I and II. Coagulase-negative *Staphylococcus species, Corynebacterium species, Streptococcus viridans and Neisseria species* are considered as commensals of the upper respiratory tract. Other species of identified bacteria in our study were considered as pathogens. The pathogenic bacteria in our patients were present in 29 samples (58%) in the middle nasal meatus and 43 (72%) in the nasopharynx (P = 0.133). We found significantly more intense colonisation by pathogenic bacteria in the middle nasal meatus in children exposed to SHS than in children with non-smoking parents (P = 0.045). Polymicrobial growth of pathogens (defined as detection of at least 2 or more pathogens from one sampling site) in the middle nasal meatus was also significantly increased in children exposed to SHS (P = 0.032) (Fig. 1A). In the nasopharynx, there were no significant differences in the presence of commensals, pathogenic bacteria, or polymicrobial growth between children exposed and not exposed to tobacco smoke (Fig. 1B). In children exposed to SHS, *Streptococcus pneumoniae* was significantly more often isolated from the middle nasal meatus and nasopharynx (P = 0.031, P = 0.004, respectively). Other pathogens, such as *Staphylococcus aureus*, *Haemophilus influenzae* and *Moraxella catarrhalis,* were more often colonised in the middle nasal meatus in children exposed to SHS (P = 0.031, P = 0.001, P = 0.05, respectively) (Table I). Those differences were not observed in the nasopharynx. Identification of pathogenic bacteria in the middle nasal meatus did not correlate with isolation of pathogenic bacteria in the nasopharynx in either group of children. Gram-negative pathogens were isolated significantly more often from the middle nasal meatus in children exposed to SHS compare to gram-positive bacteria (P = 0.018). There were no significant differences in the presence of gram-positive and gram-negative bacteria in the nasopharynx between children exposed and not exposed to tobacco smoke (P = 0.723).

Analysing the differences in selected immunological parameters according to the exposure to tobacco smoke, a possible association with passive smoking was found. Significant higher levels of IgA and IgG in peripheral blood were detected in children exposed to SHS compared to children whose parents were non-smokers (P = 0.047, P = 0.031, respectively). Differences in the markers of cellular immunity were not found (Table III).

Table I. Bacterial species isolated from middle nasal meatus.

Bacteria	SHS exposed (n = 18)	SHS non-exposed (n = 32)	P
Gram-positive			
Coagulase-negative Staphylococcus	5 (28%)	10 (31%)	0.667
Corynebacterium species	4 (22%)	7 (30%)	0.621
Streptococcus viridans	3 (16%)	7 (30%)	0.136
Streptococcus pneumoniae	4 (22%)	1 (3%)	0.031
Streptococcus agalactiae	0	0	NA
Streptococcus β-hemolyticus	1 (5%)	0	0.361
Staphylococcus aureus	4 (22%)	1 (3%)	0.031
Staphylococcus aureus MRSA	0	0	NA
Gram-negative			
Neisseria species	2 (11%)	3 (9%)	0.599
Haemophilus influenzae	9 (50%)	2 (6%)	0.001
Moraxella catarrhalis	6 (33%)	4 (12%)	0.051
Polymicrobial growth*	6 (33%)	1 (3%)	0.032

SHS: second hand smoke; MRSA: methicillin-resistant Staphylococcus aureus; n: number of patient; NA: not applicable.
** Detection of ≥ 2 pathogenic bacteria from one sampling site in one patient.*

Table II. Bacterial species isolated from nasopharynx.

Bacteria	SHS exposed (n = 23)	SHS non-exposed (n = 37)	P
Gram-positive			
Coagulase-negative Staphylococcus	1 (4%)	4 (11%)	0.379
Corynebacterium species	1 (4%)	1 (3%)	0.693
Streptococcus viridans	18 (78%)	26 (70%)	0.703
Streptococcus pneumoniae	8 (35%)	2 (5%)	0.004
Streptococcus agalactiae	0	1 (3%)	0.617
Streptococcus β-hemolyticus	1 (4%)	0	0.383
Staphylococcus aureus	6 (26%)	9 (24%)	0.576
Staphylococcus aureus MRSA	0	0	NA
Gram-negative			
Neisseria species	7 (30%)	15 (40%)	0.607
Haemophilus influenzae	10 (43%)	15 (40%)	0.521
Moraxella catarrhalis	5 (22%)	7 (19%)	0.791
Polymicrobial growth*	8 (35%)	14 (38%)	0.978

SHS: second hand smoke; MRSA: methicillin-resistant Staphylococcus aureus; n: number of patient; NA: not applicable.
** Detection of ≥ 2 pathogenic bacteria from one sampling site in one patient.*

Discussion

Exposure to tobacco smoke has many harmful effects on the health status of exposed children. In our prospective study in a group of children with adenoid hypertrophy, we analysed the influence of passive smoking on mucosal microbiota and selected laboratory parameters. We were able to detect various significant changes and differences associated with exposure to tobacco smoke. Passive smoking was associated with increased colonisation with pathogenic bacteria and polymicrobial growth in the upper airways. Due to chronic stimulation of mucosal immunity, children exposed to tobacco smoke had higher serum levels of IgG and IgA, although no changes were observed in the cellular part of immunity.

Second-hand tobacco smoke consists of exhaled smoke

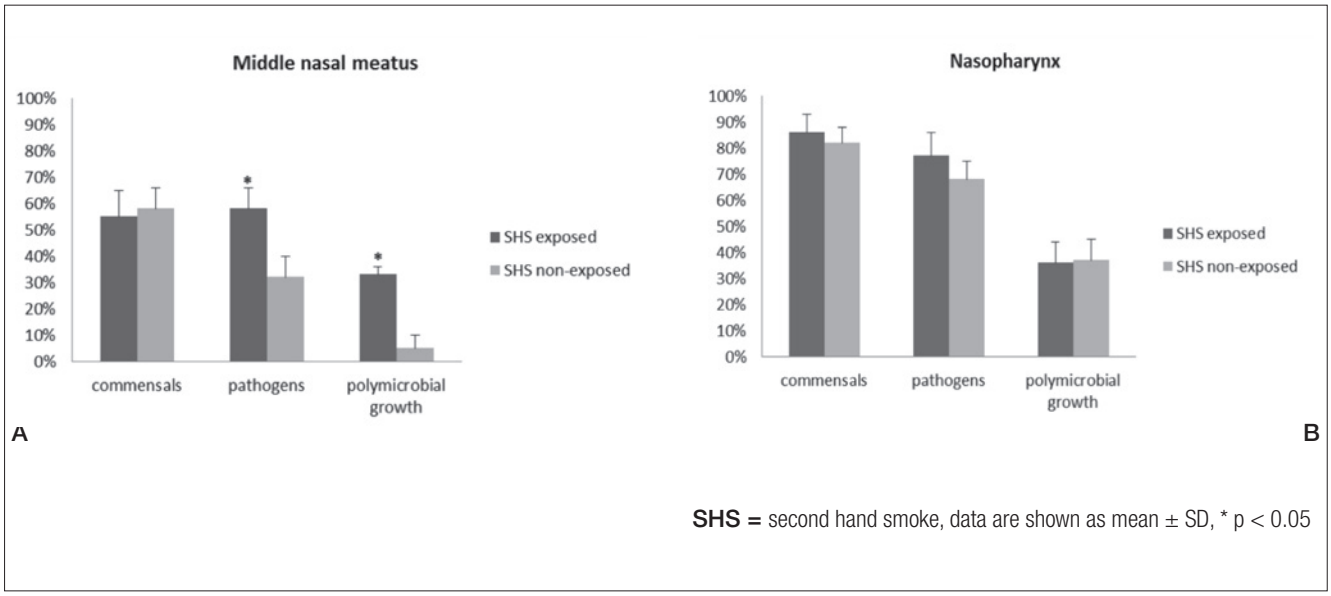

Fig. 1. Differences in microbial isolation and tobacco smoke exposure.

Table III. Differences in humoral and cellular immunity according to the second hand smoke exposure. Data are shown as mean ± SD.

Immune parameter	SHS exposed	SHS non-exposed	P
IgG [g/L]	9.87 ± 2.56	8.43 ± 1.79	0.031
IgA [g/L]	1.13 ± 0.39	0.86 ± 0.46	0.047
IgM [g/L]	0.95 ± 0.38	0.89 ± 0.38	0.477
C3 [g/L]	1.16 ± 0.19	1.16 ± 0.22	0.952
C4 [g/L]	0.25 ± 0.13	0.21 ± 0.07	0.249
IgE [IU/L]	58.94 ± 76.73	32.09 ± 29.63	0.566
Leucocytes [10^6/L]	7.77 ± 1.46	7.80 ± 2.82	0.340
Neutrophils [10^6/L]	3.15 ± 1.08	3.04 ± 0.83	0.979
Lymphocytes [10^6/L]	3.66 ± 0.84	3.84 ± 2.15	0.548
Monocytes [10^6/L]	0.65 ± 0.19	0.70 ± 0.27	0.603
Eosinophils [10^6/L]	0.39 ± 0.36	0.25 ± 0.15	0.566
Basophils [10^6/L]	0.05 ± 0.05	0.05 ± 0.05	0.862
$CD3^+$ T lymphocytes [10^9/L]	2508.77 ± 701.03	2589.93 ± 146.70	0.430
$CD19^+$ B lymphocytes [10^9/L]	627.38 ± 185.84	656.72 ± 345.82	0.786
$CD3^+CD4^+$ T lymphocytes [10^9/L]	1330.46 ± 377.87	1374.03 ± 645.44	0.596
$CD3^+CD8^+$ T lymphocytes [10^9/L]	1026.69 ± 399.34	990.52 ± 694.42	0.169
$CD15^+CD56^+$ NK cells [10^9/L]	462.69 ± 242.76	527.66 ± 438.57	0.849
CD4:CD8 ratio	1.39 ± 0.32	1.48 ± 0.47	0.751

Ig: immunoglobulin; C3 and C4: parts of complement system; NK cells: natural killer cells.

as well as side-stream smoke that is released from the burning cigarette between inhalations, which has a very similar composition. In children, exposure to smoking is associated with upper and lower respiratory tract diseases, such as acute otitis media, asthma, wheezing, cough, bronchitis, pneumonia and impaired pulmonary function [12]. Both smoking and exposure to tobacco smoke in the household are associated with carriage of bacteria, such as *Neisseria meningitidis* [13]. In children, the intensity of exposure to environmental smoking correlates with

respiratory infection rates, especially if the parent smokes in the same room as the child [14]. In young children, *Streptococcus pneumoniae, Haemophilus influenzae* and *Moraxella catarrhalis* are the most common bacterial pathogens that cause respiratory infections, such as acute otitis media and pneumonia, as well as invasive infections, such as bacteraemia and meningitis [7]. Adenoid hypertrophy is a common pathological condition in early childhood and is frequently associated with upper respiratory tract obstruction (leading to e.g. obstructive sleep apnoea syndrome) [15] and recurrent respiratory tract infections. Based on our results, passive smoking had a negative influence on selected parameters in children with adenoid hypertrophy, and could therefore represent an avoidable factor that can complicate the clinical status of these children.

Colonisation of organisms is the first step toward development of respiratory diseases. Therefore, recognition of risk factors for colonisation is important. However, data are sparse with regards to the influence of smoking on rates of nasal and nasopharyngeal colonisation with these organisms in children and adults. In the present study, we demonstrated that children exposed to smoking by parents had a significantly higher rate of pathogenic bacteria (*Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis* and *Staphylococcus aureus*) carriage than did children who were not exposed to tobacco smoke. The increased carriage rate in individuals exposed to tobacco smoke was seen in the middle nasal meatus, but not in nasopharyngeal specimens. The differences between these two sites were found also by Rawling et al. (2013) [16]. Increased nasal colonisation by pathogenic bacteria may be a predisposing factor to additional spread, resulting in lower respiratory tract infections. This could be further increased during coinfection with respiratory viruses, such as influenza viruses, in children [17]. In a recent study, it was shown that colonisation with *Staphylococcus* may be also beneficial to some extent, since it counteracts otopathogens [10]. Despite the fact that we did not study the effect of bacterial colonisation and passive smoking on the frequency of respiratory infections, our findings might explain previous reports demonstrating that children exposed to environmental tobacco smoke more frequently experience respiratory infections, such as acute otitis media and pneumonia [7 14]. Increased polymicrobial growth of pathogens in the upper respiratory tract in children exposed to SHS compared with children born to non-smoking parents found in our study also confirmed the role of tobacco smoke in the pathogenesis of respiratory tract infections in those children in early childhood.

The mechanism by which smoking and passive exposure to tobacco smoke is associated with carriage of potentially pathogenic bacteria is not fully understood. One of the possible explanations is based on mucociliary transport alterations. Several studies have shown that cigarette smoke significantly reduces the ciliary beat of respiratory epithelial cells both *in vitro* [18] and *in vivo* [19]. Moreover, Tamashiro et al. (2009) found that exposure of tobacco smoke impaired ciliogenesis in a dose-dependent manner in murine sinonasal epithelial cell culture [20]. Cigarette smoking is also associated with profound changes in the mechanisms of mucous production. Chronic exposure to tobacco smoke causes respiratory epithelium metaplasia with increased number and size of goblet cells and, consequently, increased mucous secretion in the upper respiratory tract. Furthermore, tobacco smoke also inhibits interleukin 8 and human β-defensin in sinonasal epithelial cell cultures derived from patients with CRS [21]. These findings suggest that cigarette smoke may have a suppressive function on sinonasal innate immunity. Taken together, more intense colonisation of upper respiratory tract by pathogenic bacteria might be a result of impaired local defence mechanisms of respiratory mucosa. Moreover, respiratory pathogens (e.g. *Streptococcus pneumoniae*) are associated with increased adherence to respiratory epithelial cells in chronic exposure to cigarette smoke [22].

This study also demonstrated that gram-negative bacteria were significantly more often isolated from the middle nasal meatus in children exposed to SHS compared to non-exposed children. Similar to our results, Ertel et al. (1991) showed that the respiratory system of adult smokers is preferentially colonised by gram-negative bacilli. This is explained by better resistance of gram-negative bacteria to cigarette smoke compared to gram-positive ones [23].

On the other hand, only *Streptococcus pneumoniae* was found more often in the nasopharynx in children exposed to SHS compared to non-exposed children. Differences in other pathogens according to tobacco exposure were not observed. One explanation for this finding is the study population. All children in the present study underwent surgical treatment due to adenoid hypertrophy. Pathogens such as *Haemophilus influenzae, Streptococcus pneumoniae* and *Moraxella catarrhalis* are most commonly detected bacteria in adenoid tissue [24]. Therefore, we assume that colonisation by pathogens was up-regulated due to adenoid hypertrophy, and thus the possible differences between tobacco exposure and pathogens in nasopharynx in children with adenoid hypertrophy could not be manifested.

In our study, we observed an effect of passive smoking on markers of systemic humoral immunity (IgG and IgA), which were increased in children regularly exposed to tobacco smoke compared to non-exposed children. There

are only a few reports on the effects of passive and active smoking on humoral mucosal and systemic immunity in the literature. It has been shown that smokers may have increased [25] but also decreased concentrations of IgA in saliva [26]. Exposure of cigarette smoke supports the activation of innate immunity (e.g. Toll-like receptors, neutrophils) with possible effects on parameters of adaptive humoral immunity [27]. On the other hand, tobacco smoke increases the susceptibility of respiratory mucosa to various viruses and bacteria [28], which can contribute to the recurrent respiratory infections, chronic stimulation of mucosal immune system and its hypertrophy. Hypertrophic lymphoid tissue in the upper airways is associated with increased levels of saliva IgA [29]. Maternal smoking increases chronic upper respiratory symptoms and saliva IgA levels in children [30]. The increased levels of IgA and IgG in peripheral blood observed in our children might be therefore attributed to the chronic stimulation of lymphoid tissue of upper airways due to recurrent respiratory infections, which can be the result of exposure to passive smoke. Moreover, this activation of humoral immunity can also be supported by chronic inflammation induced by exposure to tobacco smoke. Passive smoking was also shown to be a risk factor for the development of adenoid hypertrophy in another study [31]. Recently, it was shown that adenoid tissue is related to a Th-2 deviated immune response. This could aggravate the harmful effect of passive smoking on mucosal immunity and microbial colonization [32]. It was further shown that smoking induces nasopharyngeal lymphoid hyperplasia, and therefore passive smoking could also be considered as a risk factor for development of adenoid hypertrophy [33]. Another important contributing mechanism of passive smoking is the induction of persistent oxidative stress and endothelial dysfunction [34].

Conclusions

Tobacco smoke exposure is related to increased colonisation by pathogenic bacteria in the middle nasal meatus, but not in the nasopharynx. Identification of bacteria from the middle nasal meatus did not correlate with isolation of pathogenic bacteria from the nasopharynx. The upper respiratory tract is preferentially colonised by gram-negative bacteria in children exposed to second hand smoke. Chronic passive smoking alters the composition of upper airway mucosal microbiota and thus contributes to the development of several pathological conditions. Exposure to tobacco smoke leads to elevation of IgA and IgG in peripheral blood, but does not influence markers of cellular immunity in children. Therefore, it can be suggested that avoidance of passive smoking represents a universal strategy for prevention of different inflammatory conditions in children and could be recommended as a standard part of complex management of children with recurrent respiratory tract infections and adenoid hypertrophy.

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Conflict of interest statement

None declared.

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Address for correspondence: Milos Jesenak, Department of Paediatrics, Jessenius Faculty of Medicine, Comenius University in Bratislava, Kollarova 2, 036 59 Martin, Slovakia. Tel. +421 43 4203 959. Fax +421 43 4222 678. E-mail: jesenak@gmail.com