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Effect of environmental tobacco smoke on oral pigmentation: A systematic review

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Review Article

Abstract

BACKGROUND AND AIM: Oral pigmentation is a condition in which the color of oral mucosa such as gingival mucosa changes. Some exogenous and endogenous factors may lead to oral pigmentation. Secondhand smoke (SHS) or environmental tobacco smoke (ETS) is indirect smoking of an active smoker's exhalation that can lead to cardiovascular, respiratory system, and some oral diseases. The aim of this review study is to assess the effect of SHS on oral pigmentation.

METHODS: Data in this study were collected from PubMed, MEDLINE, and Scopus databases with the Medical Subject Headings (MeSH) keywords (Passive Smoking, Secondhand Smoke, Environmental Tobacco Smoke, Smoke Pollution, Involuntary Smoking, and Pigmentation) in the English language among the studies conducted in the period of 1990 to 2019. All records were imported into the EndNote software and duplicate articles were removed. The titles and abstracts of all records were pre-screened and among the articles remained, the relevant ones were selected for review based on the inclusion and exclusion criteria. To assess the quality of the studies, the Strengthening the Reporting Observational Studies in Epidemiology (STROBE) checklist was used.

RESULTS: Based on the STROBE checklist, the quality of the studies was assessed and finally, seven studies were included in the review, with six of them conducted about children and young adults and one about women. 6 (85.7%) articles showed a strong correlation between ETS and oral pigmentation and 1 (14.2%) showed no correlation.

CONCLUSION: ETS probably was correlated to the oral pigmentation.

KEYWORDS: Passive Smoking; Mouth Pigmentation; Tobacco Smoking; Environmental Tobacco Smoke Pollution

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Pigmentation in the oral cavity has been defined as a change in the color of the oral mucosa mainly caused by the melanin-induced pigmentation which may lead to esthetic problems.[1] Becker expressed the presence of melanocytes in the epithelium for the first time.[2] The presence of melanocytes in the gingival tissue was later identified by Laidlaw and Cahn.[3] Melanocytes are present in the basal layer of epithelium and if stimulated by stimuli such as nicotine and benzopyran in cigarette smoke, they increase the production of melanosomes containing melanin, hence causing oral pigmentation. Melanin is derived from tyrosine during biochemical processes.[4,5] Oral pigmentation is the result of either pathologic or physiologic factors. The pathologic factors are divided into two main categories: exogenous factors and endogenous factors, with exogenous agents including amalgam tattoo, lead poisoning, antimalarial drugs, and cigarette consumption. The endogenous factors are Peutz-Jeghers syndrome (PJS), Addison's disease, hormonal disorders, and physiological factors.[6] Oral pigmentation occurs in various areas such as palate, ventral

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surface of the tongue, labial mucosa, and attached gingiva, with the attached gingiva being the most common place.[7] Oral pigmentation in dark-skin individuals is more prevalent compared to the white-skin ones.[8] It is also less common among Europeans in comparison to the Asians. Indians have the highest percentage of oral pigmentation in Asia.[7,9] There is a close relationship between oral pigmentation and smoking, thus there is a close relation between the number of years of quitting smoking and the reduction of pigmentation. Moreover, increased pigmentation in the oral cavity has been reported due to an increase in the number of cigarettes consumed.[5,10] Passive smoking or environmental tobacco smoke (ETS) is the inhalation of a smoker's cigarette smoke.[11] ETS causes numerous health problems for humans: Exposure to ETS can cause respiratory diseases, lung cancer, cardiovascular diseases (CVDs), and death.[5,12,13] Exposure to ETS through the placenta has also been reported. It can also cause behavioral abnormalities and early childhood cancers.[14] Other complications such as orofacial cleft, short clinical crowns (SCC), and dental caries can all be attributed to ETS.[15] ETS-induced vasoconstriction reduces blood flow to tissues so it may lead to tissue damage.[16] The aim in this review study is to evaluate the effect of ETS on oral pigmentation.

Methods

Studies in this systematic review were assessed by systematic searching in the online databases PubMed, Scopus, and MEDLINE. All keywords were checked with the Medical Subject Headings (MeSH) database. The search phrase was ("passive smoking OR secondhand smoke (SHS) OR environmental tobacco smoke (ETS) OR Smoke Pollution OR Involuntary Smoking") AND ("pigmentation"). The searches were limited to the published and peer-reviewed articles in the English language from 1990 to 2019. The inclusion criteria were full-text articles in the English language, articles related to pigmentation in the oral cavity, and human subjects. The exclusion criteria included unpublished articles, non-peer-reviewed articles, conference reports, case reports, and letter to editors. All data were imported into the EndNote software and duplicate studies were deleted. Two reviewers (PF, SR) pre-screened the title and abstract of all search records independently. The articles that did not meet the inclusion criteria and were irrelevant to the study were excluded. Disagreement about eligibility was resolved by a discussion between the two reviewers. If the articles met the inclusion criteria, their full-text versions were obtained for assessment. Two reviewers (PF, RN) evaluated the remaining full-text articles based on the inclusion criteria. The irrelevant articles according to the title, abstract, and body text were excluded.

To evaluate the quality of the final studies, the Strengthening the Reporting Observational Studies in Epidemiology (STROBE) checklist[17] was used. This checklist includes 22 items and each item was allocated a score of 1 in this study. The studies with a total score of less than 15 were excluded and the ones scoring 15 to 22 were included in the review (Table 1). Eventually, for the remaining studies, the data regarding the references, year of publication, study design, sample size, intervention, and the main outcomes were extracted and imported into the Excel software. The search process is depicted in a flowchart demonstrated in figure 1.

Results

The study selection procedure in this systematic review has been detailed in figure 1. Initially, 20 articles were found after removing duplicate records. Finally, 7 articles were included in the systematic review after the quality assessment. The main data of each study is illustrated in table 1. 6 studies were conducted on children and young adults and 1 study was about women.

Table 1. Information of the studies included in the review

References	Study Design	Sample Size	Intervention	Main outcomes	Score in the STROBE checklist
Hanioka et al.[5]	Case-control study	59 patients	Two independent examiners evaluated gingival pigmentation via oral photographs MIS was used to determine gingival pigmentation	Children who had smoker parent(s) showed more gingival pigmentation	18
Hajifattahi et al.[18]	Case-control study	400 patients	Case and control groups matched in terms of skin color. Oral pigmentation of the two groups was recorded. MIS was used to determine gingival pigmentation.	Oral pigmentation was seen in 61% of the control group (non-exposed) and 75% of the case group (exposed) (P < 0.005)	19
Sridharan et al.[8]	Cross-sectional study	153 patients	Urinary cotinine biomarker was obtained for participants. GPI and intra-oral photographs were used to evaluate gingival pigmentation. MIS was used to determine gingival pigmentation.	Increase in the urinary cotinine in all exposed participants More gingival pigmentation in passive smokers (P < 0.050)	17
Yadav et al.[19]	Cross-sectional study	117 patients	Participants were divided into 2 groups (group 1: 10-14 years; group 2: 15-21 years). MIS was used to determine gingival pigmentation.	17.24% of group 1 showed no pigmentation (MIS 0), while 5.08% pigmentation was observed in group 2 (P < 0.001) On the other hand, 38.98% of group 2 showed band-like hyperpigmentation (MIS2) vs 17.24% in group 1.	17
Moravej-Salehi et al.[6]	Retrospective cohort study	100 patients	Clinical examination was carried out to evaluate gingival pigmentation. MIS was used to determine gingival pigmentation	54% of passive smokers vs. 28% of controls showed gingival pigmentation (P < 0.050) House floor area was correlated with gingival pigmentation (P < 0.025)	17
Ponnaiyan et al.[9]	Cross-sectional study	200 patients	GPI was used for extension of gingival pigmentation and DOPI was used for the intensity of gingival pigmentation.	Intensity and extension of oral pigmentation were more observed in passive smokers (P < 0.001).	17
Hasmun et al.[15]	Case-control study	44 patients	Gingival pigmentation was assessed using MIS.	Gingival melanin index was the same in children exposed and unexposed to ETS	16

ETS: Environmental tobacco smoke; MIS: Melanin Index Score; GPI: Gingival pigmentation Index; DOPI: Dummett-Gupta oral pigmentation index; STROBE: Strengthening the Reporting Observational Studies in Epidemiology

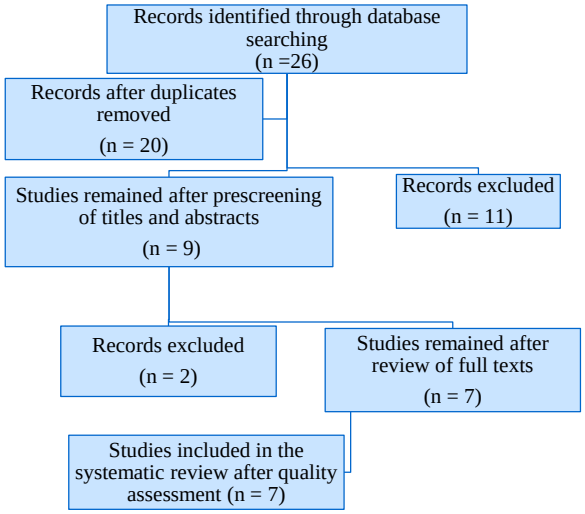

Figure 1. Flowchart of studies considered in the review

The sample sizes varied from 44 to 400. The oldest and newest studies were carried out in 2005 and 2017, respectively. Among these 7 studies, 3, 3, and 1 were case-control, cross-sectional, and retrospective cohort studies, respectively. In the oral cavity, the most common site discussed was gingival tissue. All ETS-exposed individuals had at least one smoker parent or husband. Melanin Index Score (MIS) or Gingival Pigmentation Index (GPI) that are the same, were used in all studies to evaluate the oral (or gingival) pigmentation.

Discussion

In this study, 7 articles performed to evaluate the effect of ETS on oral pigmentation were reviewed. These studies showed that ETS has a strong relationship with oral pigmentation.[5,6,8,9,15,18,19] In a study by Hanioka et al.[5] in Japan, gingival pigmentation was evaluated using oral photographs taken by a digital camera among 59 children in the age range of 6 to 16 years old. In this study, the smoker parents were identified using oral photographs. Two examiners independently reviewed the photographs and classified gingival pigmentation based on MIS considering 3 main scores of 0, 1, and 2 indicating no pigmentation, solitary unit of pigmentation, and continuous band-like hyperpigmentation, respectively. In this study, children who had smoking parent(s) showed more gingival pigmentation. Furthermore, in this study, different lesions similar to the ETS-induced pigmentation like melanoma, antimalarial drug pigmentations, and some others were mentioned, but there was no standard criterion for this distinction. A standard scale may be necessary for differentiation between these lesions. There was no gender predilection in oral pigmentation. Additionally, continuous band-like hyperpigmentation was more obvious than the solitary forms in the subjects but surprisingly, the number of smoking parents in children with a solitary pattern of pigmentation was more in comparison to the children with continuous pattern of pigmentation. Maybe further studies need to be performed to solve this contrast. Skin color matching should be considered by researchers for more reliable results.[5] In a retrospective cohort study in Iran, researchers evaluated 200 children (10 to 11 year-old) with no gender predilection in oral pigmentation. They used a standard scale for the classification of skin colors and matched them. They used the MIS scale in order to assess oral pigmentation. More gingival pigmentation was observed in ETS-exposed children ($P < 0.005$). Fair skin children were more susceptible to oral pigmentation in comparison to the dark skin children. Besides, the anterior surface of the jaws was the most common site for oral pigmentation.[18]

In a study in India, the same oral pigmentation evaluation method (oral photographs taken by a digital camera) was used as the one used in the study by Hanioka et al.[5] It seems that the method in this study was more reliable compared to the similar method carried out in Japan given the use of standard lighting and backdrop conditions. They also obtained urine samples from their subjects to assess the cotinine level (a metabolite of nicotine) and used the MIS scale in order to assess oral pigmentation like previous studies. They also highlighted the

number of years of exposure of the subjects to ETS that may play a key role in the prevalence of oral pigmentation.[8] Yadav et al. carried out a study similar to the studies carried out in Japan and India and used the same methods. They also prooved that there is a logical relationship between the number of cigarette packs smoked by parents and the gingival pigmentation.[19] The only study on women was conducted in Iran by Moravej-Salehi et al.,[6] which showed that pigmentation in buccal mucosa was very rare and the most commonplace for oral pigmentation was the labial surface of the jaws. They showed a connection between house floor area and oral pigmentation in women. Ponnaiyan et al.[9] used a different method for evaluating the gingival pigmentation called Dummett-Gupta oral pigmentation index (DOPI) which contains 4 main degrees as 0, 1, 2, and 3 indicating pink, mild brown, moderate brown or mixed brown and pink, and deep brown or blue, respectively Hasmun et al. in New Zealand evaluated children (1-5 year-old) and showed quite different results from the previous studies and demonstrated that the mean melanin index was the same in both ETS-exposed and unexposed children. This finding may originate from the lack of years of exposure in these children.

The main limitation in the present study was the insufficient number of articles and high heterogeneity in the studies in terms of the number of years of exposure and number of cigarettes smoked in the subjects' environment. Further studies are needed to solve these heterogeneities and to consider women and adult men. Moreover, it is essential to perform more investigations on other areas involved in the oral cavity.

Conclusion

Oral pigmentation is a color-changing condition that occurs in the oral cavity in different areas such as the labial surface of the jaws, buccal mucosa, and gingival tissue and may also lead to unhealthy outcomes such as esthetic problems. Based on the studies, ETS may cause pigmentation in the oral cavity, especially in children and women. Practitioners should inform families about this problem to prevent oral pigmentation caused by ETS and to prevent the associated systematic diseases.

Conflict of Interests

Authors have no conflict of interest.

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