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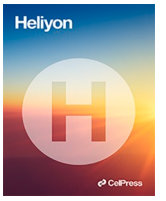

Research article

Exposure to mixture particulate contaminants in the air and the risk of oral cancer: An updated systematic review and meta-analysis

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ABSTRACT

The mixture of contaminants in the air (e.g., $PM_{2.5}$, smoke) is a part of air pollutants that has become a hot environmental issue. Previous epidemiological studies have reported the relationship between wood smoke and $PM_{2.5}$ exposure and oral cancer, but findings have been inconsistent. Therefore, this work designed to find out the relationship between mixture contaminants in air exposure and oral cancer. Fourteen studies were included through research in three databases before February 2024. Before analysis, the Newcastle-Ottawa Scale was applied to examine the quality of all selected studies. Then, the meta-analysis was carried out by meta-regression analysis, sensitivity analysis and subgroup analysis. The results showed that exposure to $PM_{2.5}$ may have a positive association with oral cancer (pooled OR = 1.13, 95 % confidence interval: 1.06, 1.20). In contrast, no significant association was found between indoor air pollution and oral cancer. However, the result of the subgroup analysis indicated there is a significant association of indoor air pollution and oral cancer in developing countries (pooled OR = 2.5, 95 % confidence interval: 1.7, 3.6). In addition, the heterogeneity among studies of indoor air pollution exposure and oral cancer may caused by studies carried out in developed countries according to the subgroup and meta-regression analyses. In conclusion, the studies about indoor air pollution exposure and oral cancer are discrepant. The effects of mixed air contaminants for people's health are not simple and more studies are demanded to find out it in the future.

1. Introduction

Recently, air contamination is a hot spot in the field of environmental issues. With the development of industrialization, urbanization, and population, anthropogenic activities, including industrial and vehicular emissions, use of solid biomass fuel, and agricultural residue burning in neighbouring rural areas caused the aggravation of air pollution [1]. Evidence suggests that human can be affected by the toxic substances present in the air through inhalation and dermal, and airborne particulate matter has been considered as carcinogenic by International Agency for Research on Cancer [2]. So far, studies have showed air pollution has a positive association with lung cancer, breathing trouble, pneumonia, and bronchitis [2–4]. Among various air contaminants, the mixture of particulate

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contaminants has significant effects. Heating and cooking in homes with solid fuels with solid fuels produce a mixture of particulates, leading to indoor air pollution [5]. Indead *Ramanakumar* et al. found the women who heated or cooked in homes by wood or coal stoves more prone to lung cancer than other people [6]. In addition, as a heterogeneous mixture of solid particles contaminants and liquid droplets, fine particulate matter is increasingly accepted as a kind of hazardous component of air pollution, especially $PM_{2.5}$ (particles with diameters 2.5 μm and smaller) [7]. Previous studies have found $PM_{2.5}$ exposure can increase cardiovascular mortality and morbidity [8].

Up to now, cancer has posed serious threats to people's health because of its high morbidity and mortality. Oral cancer as the sixth most common cancer is still a serious global public health threat [9]. Research has found that alcohol consumption, tobacco use, viral infection, occupational exposure, immune deficits, and family heredity are associated with oral cancer [9]. In addition, environmental contaminants including particulate matter [10] and the burning of solid fuels [11], are related to the occurrence of oral cancer. Although previous studies have investigated the association between $PM_{2.5}$ or burning of solid fuels exposure and oral cancer, consensus and certain relationships have not been found in these studies. A cohort study of Turner et al. suggested that there was no relationship between $PM_{2.5}$ exposure and oral cancer [12]. In another cohort study, Ku et al. suggested that exposure to $PM_{2.5}$ was positively correlated with oral cancer [13]. In terms of burning solid fuels, Franco et al. indicated that exposure to smoke from fossils fuel is related to oral cancer [14], whereas *Dietz* et al. did not [15]. The inconsistencies in the results may have been caused by many diverse sample sizes, object, and study designs.

To date, review articles have summarized that indoor air pollution and traffic-related air pollution exposure are related to cancer, which may be due to oxidative damage to DNA [16]. For example, the generation of reactive oxygen species and oxidative damage to DNA which are positively correlated with cancer are related to air contamination exposure. Systematic reviews of air pollution and oral cancer are relatively few. Although there may be a duplication of pollutants between outdoor air pollutants and indoor air pollutants, the burning of solid fuels indoor is one of the majority reason for mortality and disease in developing countries [17]. In addition, $PM_{2.5}$ and PM_{10} are important components of air pollutants that affect both air quality and students' health [18]. However, $PM_{2.5}$ has become the preferred indicator because of its more significant health impacts in most studies [19]. We used $PM_{2.5}$ and the burning of solid fuel replace common pollutants of outdoor and indoor air pollution, respectively. Therefore, to further understand the association between oral cancer and a mixture of particulate contaminants in the air, including $PM_{2.5}$ and smoke produced by the burning of solid fuels, 14 published articles was used to perform a systematic review and quantitative meta-analysis. Furthermore, significant confounding factors (e.g., location and economic status) were investigated to find out the source of heterogeneity among these studies. In addition, we further explored the source of heterogeneity by meta-regression and sensitivity analyses.

2. Methods

This work used Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria as general principles [20].

2.1. Search strategy

The literature search was carried out in three databases (Web of Science, Embase, and PubMed electronic) for articles related to air pollution and oral cancer published up to February 2024 by two reviewers, respectively. The search strategy was as follows: "fine particulate matter", "$PM_{2.5}$", "indoor air pollution", "household air pollution", "wood smoke", "wood dust", "coal usage", "charcoal usage", and "oral cancer", "oral tumor", "oral neoplasms", "mouth neoplasms", and"oral carcinoma". The literature search mainly relied on human research that published in English. Furthermore, the references of included articles were checked manually by more than one reviewer for further identification.

2.2. Study selection criteria

The selected studies were required to match the following inclusion criteria (i) original epidemiological studies (e.g., cohort and case-control studies): investigated the relationship of air pollution and oral cancer (which were diagnosed by clinic doctors); (ii) the participants of the studies were exposed to the air pollution, including $PM_{2.5}$ and indoor pollution; and (iii) studies reported quantitative estimates for the relationship of $PM_{2.5}$ and indoor air pollution exposure and oral cancer, including OR, relative risk (RR), or hazard ratio (HR) with 95 % confidence intervals (CIs). The studies were removed according to the exclusion criteria: (i) in vitro studies, in vivo studies and animal studies; (ii) studies with duplicated data; (iii) studies for which data could not be extracted and (iv) studies of low quality. All studies were evaluated by three reviewers, independently. The disagreements were discussed again until an agreement was reached.

2.3. Quality assessment

Three reviewers used the 9-star Newcastle-Ottawa Scale (NOS) to evaluate the quality of all selected studies, respectively. The NOS assessment items of the NOS items included: selection (0-4stars), comparability (0-2stars), and outcome or exposure (0-3stars). Studies with a score of more than 6 were defined as high quality, and only the studies that obtained high scores will be used for further analysis.

2.4. Data extraction

All the included studies were examined carefully by three researchers to independently extract the following information: first authors, year of publication, locations, research methods, number of subjects, exposure, information sources, statistics, adjusted variables, and effect sizes about the relationship of air pollution and oral cancer. As six studies reported ORs, four studies reported RRs and three studies reported HRs, ORs with 95 % CIs were applied for further investigation. Furthermore, the disagreements among the reviewers were discussed again to obtain the same results.

2.5. Statistical analysis

The association between exposure to a mixture of particle contaminants in the air and oral cancer was investigated by calculating pooled effect estimates via a random-effects model meta-analysis. To facilitate statistical analysis, ORs with 95 % CIs were reported as main effect estimates in this study, and other effect estimates, including RRs and HRs, were transformed into ORs ($RR = OR/[1 - P_0 \times (1 - OR)]$, P_0 is incidence rate; $HR = OR$) [21,22]. The effect of heterogeneity across studies were assessed by calculating the I^2 statistic. Low, medium, and high heterogeneity were judged by I^2 values of 25 %, 50 %, and 75 %, respectively. If heterogeneity was present, the reasons were investigated by using subgroup and sensitivity analyses. In addition, the leave-one-out approach was applied in sensitivity analyses to assess the stability of the results. If the I^2 was above 50 %, a meta-regression analysis was applied to determine the source of variation. In addition, publication bias assessment was inappropriate because the number of included studies was less than ten [23]. The meta-analysis was carried out by the Stata 14.0 by setting α as 0.05.

3. Results

3.1. Study results

798 articles were searched in the three databases based on the PRISMA guidelines (Fig. 1). Five hundred and seventeen articles were excluded because of the duplicate. After sifting titles and abstracts, 251 articles that were not related to the topic were excluded. Then, sixteen full-text articles were removed, among which 5 studies were reviews, 2 studies were meta-analysis reviews, one study was an animal study, one study was a letter, one study had overlapping information with another study, 4 studies lacked data and 2 studies can not match the selection criterion. 14 studies were finally selected in this work to further investigate the association of exposure to mixed particle contaminants in air and oral cancer [11–14,24–31].

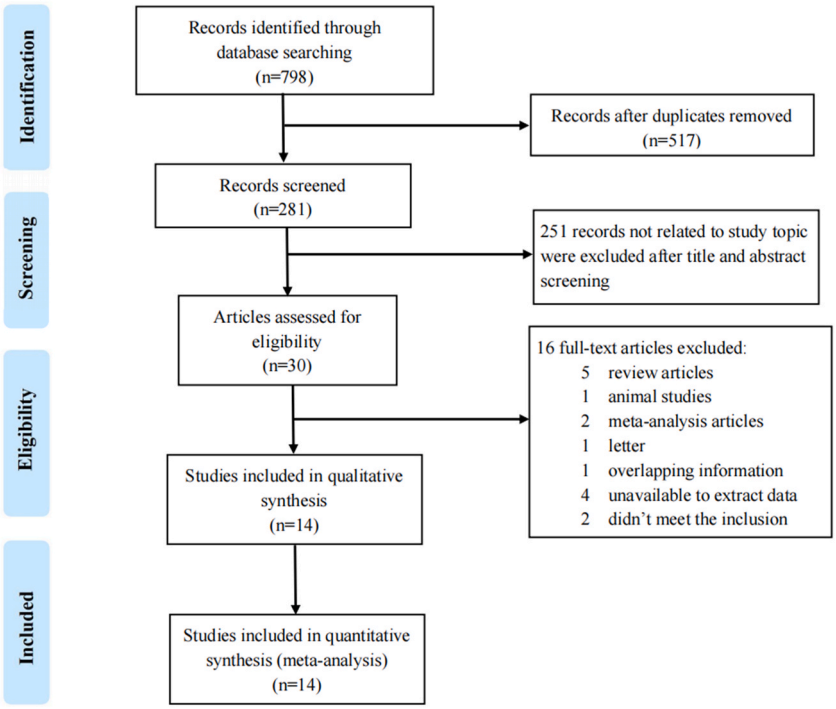

Fig. 1. Flow chart of study search strategy based on the PRISMA guidelines.

3.2. Study characteristics

For 14 included studies, 7 evaluated the relationship of $PM_{2.5}$ exposure and oral cancer, and 7 reported the relationship of indoor air pollution exposure and oral cancer. All the studies included in this work were based on cohort (n = 7) and case-control (n = 7) studies. These studies reported from 1982 to 2019 were carried out in Europe, Asia, North America, and South America with 200–3686729 participants. Most of studies used conditional logistic regression and the Cox proportional hazards model to assess the

Table 1
Characteristics of thirteen studies included in the systematic review and meta-analysis.

References	Locations	Study periods	Designs	Sample sizes	Exposures	Information sources	Statistics	Adjusted variables
Franco et al. [14]	Brazil	1986–1988	Case-control	232	Indoor air pollution	Questionnaire	Conditional logistic regression	Age, sex, study site, and admission period.
Dietz et al. [15]	Germany	1989–1992	Case-control	500	Indoor air pollution	Interview	Chi-squared test	Age, sex, tobacco and alcohol consumption.
Pintos et al. [27]	Brazil	1987–1989	Case-control	2352	Indoor air pollution	Interview	Conditional logistic regression	Age, gender, hospital, admission period, tobacco and alcohol.
Vlajinac et al. [30]	Serbia	1998–2000	Case-control	200	Indoor air pollution	Interview	Conditional logistic regressions	Age, gender, place of residence, education, BMI, smoking, alcohol consumption and family history of oropharyngeal cancers.
Sapkota et al. [11]	Russia, Romania, Poland, Hungary, Slovakia, the Czech Republic	2003	Case-control	1313	Indoor air pollution	Hospital	Multivariate logistic regression	Country, age, sex, BMI, tobacco PY, alcohol, and consumptions of dairy red meat, fruits and vegetables.
Purdue et al. [28]	Sweden	1971–2001	Cohort	30440	Indoor air pollution	Health examinations	Poisson regression	Age, smoking status, snuff use.
Jayaprakash et al. [26]	USA	1982–1998	Case-control	1763	Indoor air pollution	Hospital	Unconditional logistic regression	Age, pack years of smoking, BMI, smoking status, education, alcoholic drinks and year of enrolment.
Chu et al. [24]	China	2012–2013	Case-control	482659	$PM_{2.5}$	Health Administration	Logistic regression	$PM_{10-2.5}$, SO_2, O_3, age, betel quid chewing, and smoking
Yu et al. [7]	Brazil	2010–2018	Cohort	1768668	$PM_{2.5}$	Brazil Mortality Information System	Quasi-Poisson regression	Sex, GDP, regions.
Shin et al. [29]	South Korea	2002–2015	Cohort	87608	$PM_{2.5}$	National Health Insurance Statistic	Cox Proportional Hazard	Age, sex, health insurance premium, employee status, smoking status, smoking period, smoking amount, frequency of alcohol consumption and physical activity, die, BMI, and family history of any cancer.
Turner et al. [12]	USA	1982–2004	Cohort	623048	$PM_{2.5}$	Questionnaire	Cox Proportional Hazard	Age, race/ethnicity, gender, education, marital status, BMI, smoking status, cigarettes per day, duration of smoking, age started smoking, passive smoking, diet, alcoholic drinks, industrial exposures; occupation dirtiness index.
Coleman et al. [10]	USA	1987–2014	Cohort	635539	$PM_{2.5}$	Public National Health Interview Survey	Cox Proportional Hazards	Age, sex, race/ethnicity, income, education, marital status, BMI, smoking, urban/rural, census regions, and survey year
Ku et al. [13]	China	2006–2016	Cohort	3686729	$PM_{2.5}$	Health Promotion Administration	Logistic regression	Sex, age, betel quid chewing and cigarette smoking.

relationship of mixture contaminants in air and oral cancer. As for some serious confounding factors, such as age, sex, smoking status, alcohol consumption, have been adjusted in a majority of studies (Table 1). Tables S1 and S2 presented the Quality assessment of 14 selected studies. 14 studies were defined as high-quality studies based on the NOS. These included studies that did not receive the full mark and lost stars because of no clear description of the items mainly including the definition of controls, non-response rate, and adequacy of follow-up of cohorts.

3.3. The association between indoor air pollution exposure and oral cancer

7 studies provided valid data that were applied to investigate the association of indoor air pollution and oral cancer. Among these studies, 5 studies used the OR and one study used RR. A study found that exposure to the smoke from wood stoves could increase the risk of oral cancer (OR = 2.7; 95 % CI: 1.8–4.2) [27], and another showed little evidence (OR = 2.3, 95 % CI: 0.96–5.7) [30]. However, 5 studies indicated there was no statistically significant relationship of indoor air pollution and oral cancer. According to forest plot, indoor air pollution exposure and oral cancer had no statistical significance (OR = 1.4, 95 % CI: 0.81–2.3), but showed obvious heterogeneity ($I^2 = 76.6$ %, $p < 0.001$) (Fig. 2).

To determine the source of heterogeneity, meta-regression and subgroup analyses by various locations and economic statuses were performed. Based on the subgroup analysis of location, the pooled OR of the studies performed in South America was 2.3 (95 % CI: 1.3–4.2) with no significant heterogeneity ($I^2 = 29$ %, $p = 0.24$) (Fig. 3a). In addition, another subgroup analysis of economic status showed there was a positive relationship of indoor air pollution exposure and oral cancer in developing countries without heterogeneity (OR = 2.3, 95 % CI: 1.7–3.4; $I^2 = 0.00$ %, $p = 0.49$) (Fig. 3b). The subgroup analysis showed that studies performed in Europe ($I^2 = 54$ %, $p = 0.09$) and developed countries ($I^2 = 63$ %, $p = 0.04$) may be the reason of heterogeneity of these studies. The meta-regression analysis indicated the variability among studies of exposure to indoor air pollution and oral cancer could not be attributed to the location ($I^2 = 50$ %, $p = 0.12$) (Table 2). However, different economic statuses may be the reason for the heterogeneity ($I^2 = 47$ %, $p = 0.04$) (Table 2). In conclusion, it was clear that the main source of the heterogeneity was from these studies conducted in developed countries. According to the sensitivity analysis, the results showed that the results were robust regardless of which article was removed (Fig. S1). Therefore, the main reason for the heterogeneity may be caused by the studies conducted in developed countries.

3.4. The association between $PM_{2.5}$ exposure and oral cancer

7 studies explored the relationship of exposure to $PM_{2.5}$ and oral cancer, among which 3 used HR, 3 used RR, and one used OR. Among these studies, 5 studies found that $PM_{2.5}$ was significantly positively related to oral cancer (OR = 1.4, 95 % CI: 1.2–1.7; RR = 1.2, 95 % CI: 1.0–1.4; HR = 1.2, 95 % CI: 1.0–1.4; HR = 1.1, 95 % CI: 1.1–1.1; RR = 1.04, 95 % CI: 1.01–1.07) [10,13,24,31,32].

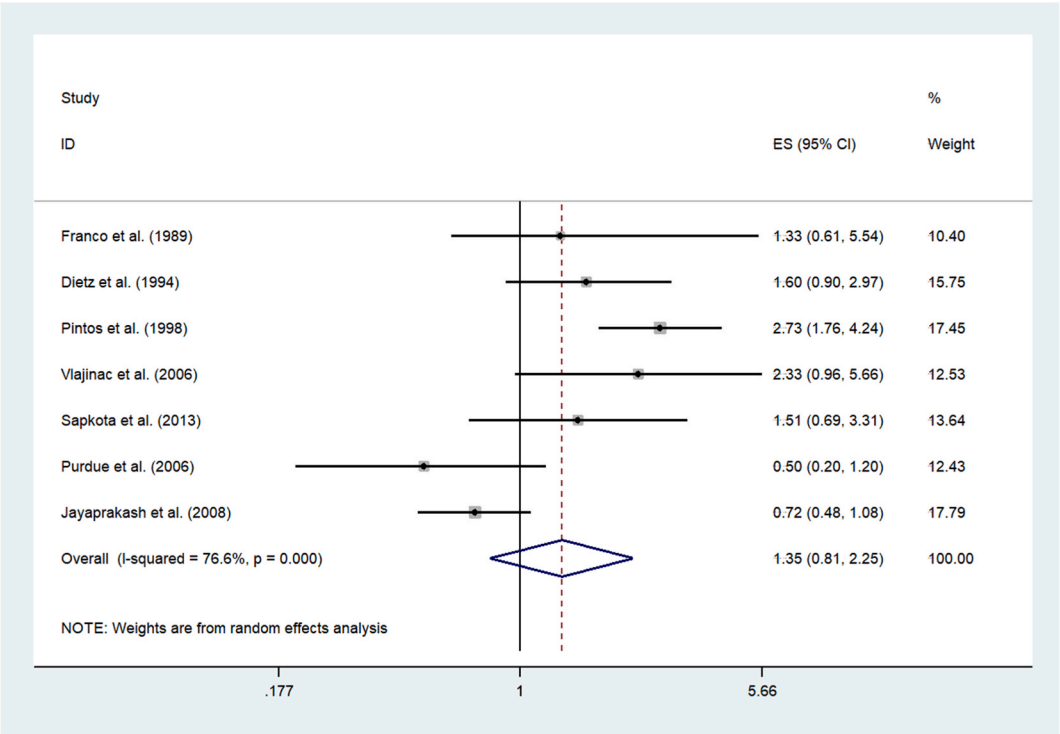

Fig. 2. Forest plots of indoor air pollution exposure and oral cancer.

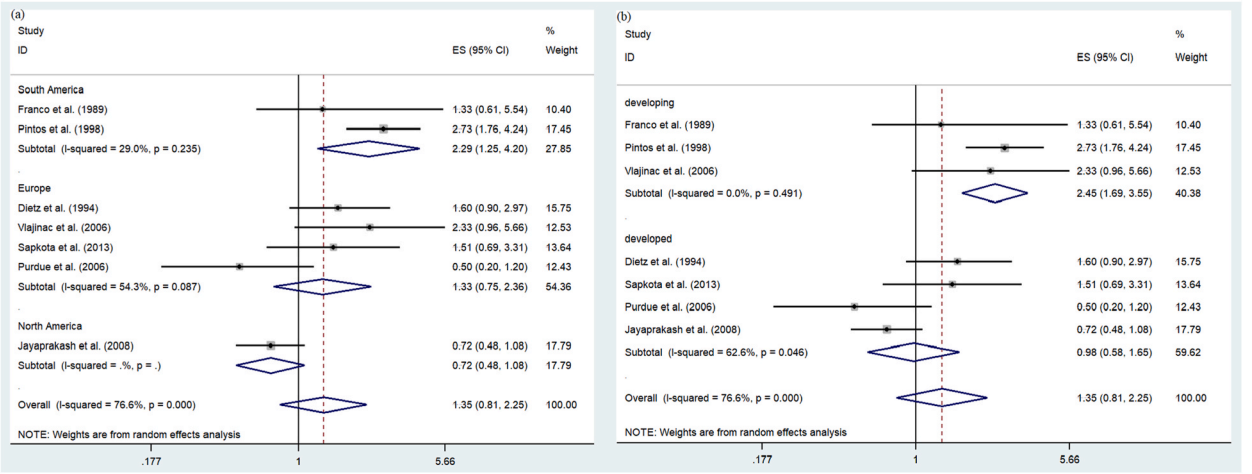

Fig. 3. Forest plots of indoor air pollution exposure and oral cancer in different location (a) and economic status (b).

Table 2
Results of the association between solid contaminants in air exposure and oral cancer.

Subgroup	No.	Subgroup analysis	Meta-regression		Heterogeneity	
		OR (95 % CI)	I^2 (%)	*p*-value	I^2 (%)	*p*-value
Indoor air pollution	7	1.35 (0.81, 2.3)			76	0.0
Location			50	0.12		
South America	2	2.3 (1.3, 4.2)		Reference group	29	0.24
Europe	4	1.3 (0.75, 2.4)		0.38	55	0.09
North America	1	0.72 (0.48, 1.1)		0.15	–	–
Economic status			47	0.035		
Developed	4	0.98 (0.58, 1.7)		Reference group	63	0.04
Developing	3	2.5 (1.7, 3.6)		0.07	0.00	0.50
$PM_{2.5}$	7	1.2 (1.1, 1.3)			45	0.11
Location			56	0.19		
South America	1	1.2 (1.1, 1.5)		Reference group	–	–
Asia	2	1.25 (1.00, 1.55)		0.95	68	0.05
North America	4	1.08 (0.99, 1.17)		0.46	32	0.23
Economic status			52	0.02		
Developed	4	1.07 (1.00, 1.14)		Reference group	13	0.33
Developing	3	1.2 2(1.06, 1.40)		0.27	71	0.03

However, two other studies indicated there was no significant relationship between $PM_{2.5}$ and oral cancer. The pooled OR from these 6 studies of $PM_{2.5}$ exposure and oral cancer was 1.2 (95 % CI: 1.1–1.3) without statistically significant heterogeneity ($I^2 = 45$ %, $p = 0.11$) (Fig. 4).

Although there was no significant heterogeneity in these studies of the relationship of $PM_{2.5}$ exposure and oral cancer, further investigation were conducted. According to the subgroup analyses, these studies investigated in developing countries ($I^2 = 71$ %, $p = 0.03$) and Asia ($I^2 = 68$ %, $p = 0.05$) showed significant heterogeneity (Figs. S2 and S3). Based on meta-regression analysis, the location ($I^2 = 56$ %, $p = 0.19$) may cause the variability of studies about $PM_{2.5}$ exposure and oral cancer, but economic status might have caused the difference ($I^2 = 52$ %, $p = 0.02$) (Table 2). The results were robust regardless of which article was removed based on the sensitivity analysis (Fig. S4).

4. Discussion

14 studies were included in this study to find out the association between mixed air contaminants exposure and oral cancer. All selected studies were identified as high quality based on the NOS. Among the 7 case-control studies, 5 studies lost their score because without description of non-response rate, and one study lacked a description of the definition of control and non-response rates. Among the 7 cohort studies, 2 studies did not present the adequacy and time of follow-up, and two studies lacked a description of the adequacy of follow-up. Fortunately, all included studies have adjusted these major confounding factors, such as age, sex, tobacco use, and alcohol consumption, which could decrease the selection bias to some extent.

The results of this study suggested that a mixture of indoor air contaminants exposure did not influence oral cancer, while there were significant associations between $PM_{2.5}$ exposure and oral cancer. Previous epidemiological research have shown that viral factors

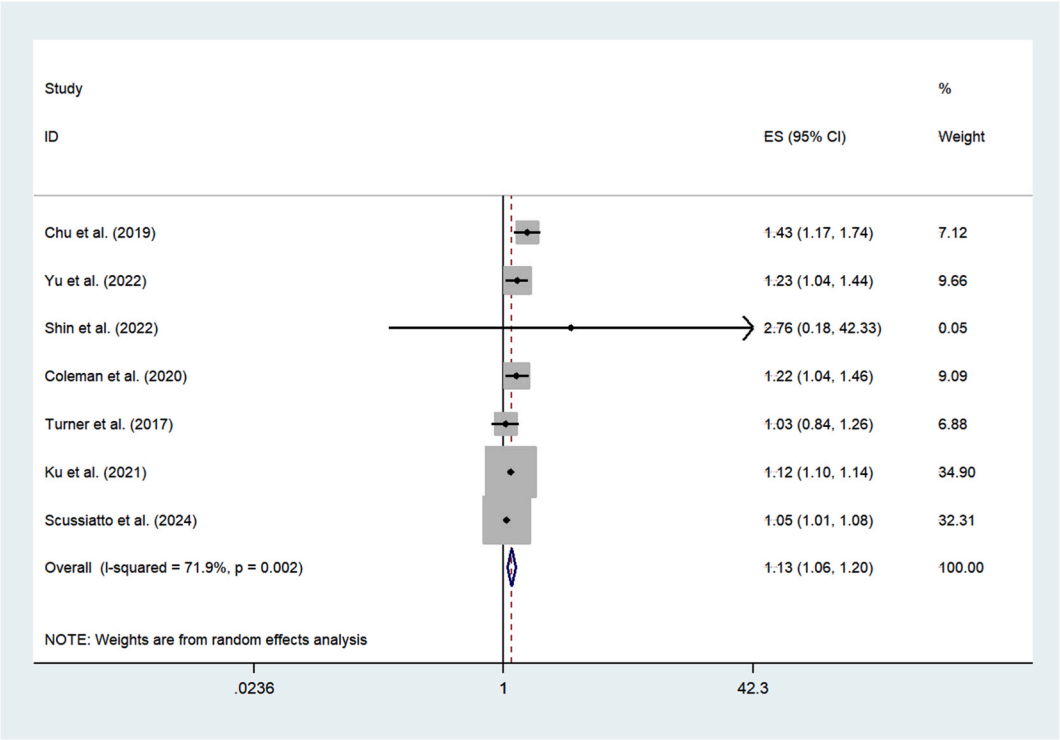

Fig. 4. Forest plots of $PM_{2.5}$ exposure and oral cancer.

including tobacco use, alcohol consumption, virus infection, familial risk, genetic factors, and immune deficits, are risk factors for oral cancer [9]. Evidence from previous epidemiological studies indicated that air pollution can lead to many diseases, including respiratory and cardiovascular diseases [33,34]. Based on previous research, human exposure to air pollution may cause oral cancer [35,36]. However, the results of the association between indoor air pollution and $PM_{2.5}$ exposure and oral cancer are inconsistent, which may be caused by diverse study populations, exposure sources, races or ethnicities, nationality, and study designs. Furthermore, age has been adjusted in all studies, and sex, alcohol intake, and cigarette consumption have been adjusted in most studies. The diverse adjustments may be another reason for these inconsistencies.

Air pollution, especially a mixture of contaminants in the air, has been verified as a risk factor of many oral diseases. An epidemiologic study found that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration showed a positive relationship with cleft palate alone (OR = 1.4, 95 % CI: 1.1–1.9) [37]. *Pintos* et al. suggested a positive association between the consumption of wood stoves for heating and cooking and oral cancer (OR = 2.7, 95 % CI: 1.8–4.2) [27]. In addition, a case-control study indicated that wood dust exposure was associated with oral cancer to some extent (OR = 2.3, 95 % CI: 0.96–5.7) [30]. However, other studies have no founding between indoor air pollution and oral cancer. Our meta-analysis also did not find obvious relationship between indoor air pollution and oral cancer, although the results showed remarkable heterogeneity.

Subgroup analyses were applied to identify the sources of heterogeneity, revealing that the heterogeneity may have been caused by the studies conducted in developed countries and Europe. The results of meta-regression analysis indicated different economic statuses can cause the heterogeneity. However, the sensitivity analysis results suggested that the pooled OR value did not substantially after omitting any study. In summary, the results indicated that the main source of heterogeneity was studies conducted in developed countries, which may have been caused by the various latitudes of these countries, leading to different usage amounts of solid fuel. Additionally, there was a positive relationship between indoor air pollution and oral cancer in developing countries (OR = 2.5, 95 % CI: 1.7–3.6). Biomass fuels, such as firewood and sawdust, are the major solid fuels in most developing countries owing to their low cost and wide availability [38,39]. Therefore, we speculated that because more solid fuels are used in developing countries, resulting in indoor air pollution is a risk factor for oral cancer in developing countries.

Some systematic reviews have described the association of cigarette consumption or heavy metals and oral cancer, but reviews about $PM_{2.5}$ and oral cancer are rare [40]. One case-control study suggested that higher levels of ambient $PM_{2.5}$ were considered a risk factor occurrence of oral cancer (OR = 1.4, 95 % CI: 1.2–1.7) [24]. There are, of course, some studies indicating no relationship of $PM_{2.5}$ exposure and oral cancer. In this meta-analysis, a positive association of exposure to $PM_{2.5}$ and oral cancer has been found with no statistically significant heterogeneity. $PM_{2.5}$ has been verified as an inducement of many biological events, including inflammation, oxidative damage, and cellular dysfunction [16,41]. Furthermore, previous research has shown that micronuclei frequency, as a biomarker of DNA damage, could be influenced by the increased $PM_{2.5}$ exposure [42], which could increase the risk of oral cancer.

Some studies have attempted to explain the mechanisms about the relationship of exposure to indoor air pollution and oral cancer.

The burning of solid fuels can produce many chemicals, including some known human carcinogens (e.g., polycyclic aromatic hydrocarbons and aldehydes) [43,44]. In addition, epidemiological evidence has shown that the increase of DNA adducts in human blood cells and the increase of mutagenicity in human urine were related to exposure to the emission from solid fuels [45]. In vitro experiments have shown found that exposure to wood smoke could enhance oxidative damage to purines in terms of formamidopyrimidine-DNA glycosylase sites and generation of reactive oxygen species [46,47]. Therefore, indoor air pollution caused by burning of solid fuels could increase the risk of oral cancer to some extent.

The main strengths of this meta-analysis are as follows: First, the relationship of exposure to mixture contaminants (e.g., $PM_{2.5}$) in air and oral cancer is firstly summarized in this work. Second, most included studies have adjusted major confounding factors (e.g., age, sex, cigarette use and alcohol consumption), which reduced the bias. Third, we carried out subgroup analyses of diverse economic status and locations because of the effect of various factors in different studies. Fourth, the outcome (oral cancer) of all included studies was diagnosed by clinical doctors, which reduced the information bias. Last, relevant findings of our meta-analysis may be useful for exploring the potential risks of air mixture contaminants exposure for oral health, reducing the hazards of mixture contaminants in the air in advance, and providing directions for future investigations.

This study has several limitations. First, the studies about indoor air pollution exposure and oral cancer are few, which may have caused potential bias. Second, there was potential heterogeneity in the studies about indoor air pollution exposure and oral cancer ($I^2 = 76$ %, $p < 0.01$), which may influence the generalization of the meta-analysis results. Third, for the reason of lack of relevant data, the subgroup analysis of different sexes was not performed, which limited the comprehensiveness of the study. Last, although most studies have adjusted the major confounding factors, the lack of consistent confounding adjustments may still cause the incorrect assessment of the actual relationship of air mixture contaminants exposure and oral cancer.

5. Conclusions

In summary, a meta-analysis of 14 studies was conducted to find out the possible relationships of exposure to mixed air contaminants and oral cancer. The meta-analysis results suggested a statistically significant relationship of oral cancer and $PM_{2.5}$ exposure (pooled OR = 1.13, 95 % CI: 1.06, 1.20). In addition, the result of the subgroup analysis indicated there is a significant relationship of indoor air pollution and oral cancer in developing countries (pooled OR = 2.5, 95 % CI: 1.7, 3.6), indicating that the usage of solid fuels is a major issue in developing countries. Although there are some studies about the relationship of indoor air pollution and oral cancer, the evidence could not provide definitive conclusions. In addition, epidemiological evidence to date is still insufficient to confirm the potential toxicities of indoor air pollution for oral health. Therefore, further investigation are demanded to verify the relationship of indoor air pollution exposure and oral cancer and evaluate the potential toxicity and mechanism of indoor air pollution on oral health.

Data availability statement

All data are already provided in manuscript and supplementary materials, further requests can be directed to the corresponding authors. Study Registration: PROSPERO CRD42024580548.

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CRediT authorship contribution statement

Hui Zhan: Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Dong Liu:** Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Zhuoma Deji:** Writing – review & editing, Methodology. **Wei Liang:** Writing – review & editing, Methodology. **Jiaoyang Li:** Writing – review & editing, Project administration, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e38568.

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