



The association between smoking and metabolic syndrome: a systematic review and meta-analysis

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Abstract

Aim Some studies have shown that there is a relationship between smoking and metabolic syndrome (MetS), while other studies have not found an association. Considering the importance of this issue, the aim of this study was to review all the studies related to smoking and its relationship with MetS and to update the information.

Subject and methods PubMed, Scopus, and Web of Science were searched to identify studies on the association between smoking and MetS published from January 1, 2000, through August 13, 2023. The pooled odds ratio (OR) and relative risk (RR) and the 95% confidence intervals were used to measure the association between smoking and MetS by adopting a random-effects meta-analytic model. Quality appraisal was undertaken using the Newcastle–Ottawa Scale.

Results A total of 78 studies were included. Regarding the association between smoking and MetS, the pooled RR for current smoking among cohort studies was 1.51 [95% CI: 1.15, 1.99] and the pooled OR for current smoking among cross-sectional studies was 1.12 [95% CI: 1.07, 1.17]. For the relationship between smoking dose and MetS, the pooled OR for subgroups with 10 or more cigarettes/day was 1.57 [95% CI: 1.04, 2.38], but no association was found between subgroups with less than 10 cigarettes/day and MetS (1.17 [95% CI: 0.99, 1.38]). For the association between duration of smoking and MetS, the pooled OR for subgroups with 10 or more years of smoking was 1.17 [95% CI: 1, 1.39], while no association was found between subgroups with less than 10 years of smoking and MetS (0.96 [95% CI: 0.80, 1.16]).

Conclusion Our analysis shows a statistically significant relationship for current smoking among cohort and cross-sectional studies and between smoking dose and duration of smoking and MetS, where the likelihood of MetS increases with dose and duration.

Keywords Metabolic syndrome · Smoking · Systematic review · Meta-analysis

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Introduction

Metabolic syndrome (MetS) is a major public health concern around the world. The prevalence of MetS has been increasing consistently in all societies over the past 20 years (Sepandi and Taghdir 2020). MetS, also known as insulin resistance syndrome, comprises a set of metabolic disorders including central obesity, blood lipid disorders, high fasting blood sugar, insulin resistance, and high blood pressure (Mohammadi et al. 2019). It is a dangerous syndrome that increases the risk of cardiovascular diseases, diabetes, dyslipidemia, stroke, some cancers, and mortality (Rashidi et al. 2010). The global prevalence of this disorder is between 14% and 32% (Farmanfarma et al. 2019).

Until 2017, the prevalence of this syndrome ranged from 2.2% to 44% in Turkey, 16–41% in Saudi Arabia, 14–63% in Pakistan, 26–33% in Qatar, and 6–42% in Iran. Also, the combined prevalence of MetS in the Middle East has been reported at 25%; it is a significant cause of stroke, coronary heart disease, and cardiovascular disease, and it is associated with a sevenfold increase in the risk of type 2 diabetes (Ansarimoghaddam et al. 2018; Cheng et al. 2019). Obesity, physical activity, high alcohol consumption, smoking, and various dietary factors are known as important modifiable risk factors for MetS (Erem et al. 2008).

Previous studies have shown that overall tobacco use is associated with an increased risk of MetS due to its effects on waist circumference, blood lipids, and blood pressure, and as a result, smoking significantly increases the risk of MetS (Jia 2013; Slagter et al. 2013; Al-khalifa et al. 2017). Although smokers have a lower body mass index (BMI) than nonsmokers, the results obtained from recent studies show that these individuals have a larger waist circumference and waist-to-hip ratio, which are risk factors for cardiovascular disorders (Berlin 2008). Thus, smoking is linked to MetS and its individual components. Smokers are more at risk of cardiovascular diseases than nonsmokers, and the prevalence of MetS is higher in this population (Chen et al. 2008). Evidence has shown that short-term smoking has no statistically significant effect on the incidence of MetS (Ishizaka et al. 2005; Liu et al. 2017). A cohort study conducted by Kim et al. (2017) showed that long-term smoking reduces MetS, but this relationship was not statistically significant. Also, two cohort studies have shown that a low dose of smoking has reduced MetS, but this relationship was not statistically significant (Kim et al. 2017; Goodman et al. 2013). These contradictory results show the necessity of conducting a meta-analysis on the dose and duration of smoking and the incidence of MetS.

Considering the importance of this issue, the aim of this study is to review all the studies related to smoking

and its relationship with MetS and to provide an extensive update of the information. Also, the relationship between the dose and duration of smoking and its effect on MetS is investigated.

Methods

Literature search strategy

To identify observational studies on the association between smoking and MetS, a comprehensive search was performed of several electronic databases including PubMed, Scopus, and Web of Science from January 1, 2000, through August 13, 2023. The search terms comprised the following keywords: “cigarette smoking,” “cigar smoking,” “smoking,” “tobacco smoking,” “metabolic syndromes,” and “metabolic syndrome.” We also investigated references of all the articles to identify studies that were not included during the initial search. The following inclusion criteria were selected for meta-analysis: the study comprised a cross-sectional or cohort study design, the primary outcome was a MetS, the relative risk (RR) or odds ratio (OR) or hazard ratio (HR) and the corresponding 95% confidence interval (CI) of MetS associated with smoking were presented, and the study was published in English. Exclusion criteria comprised intervention studies, letters to the editor, reports, case reports, reviews, and meta-analyses.

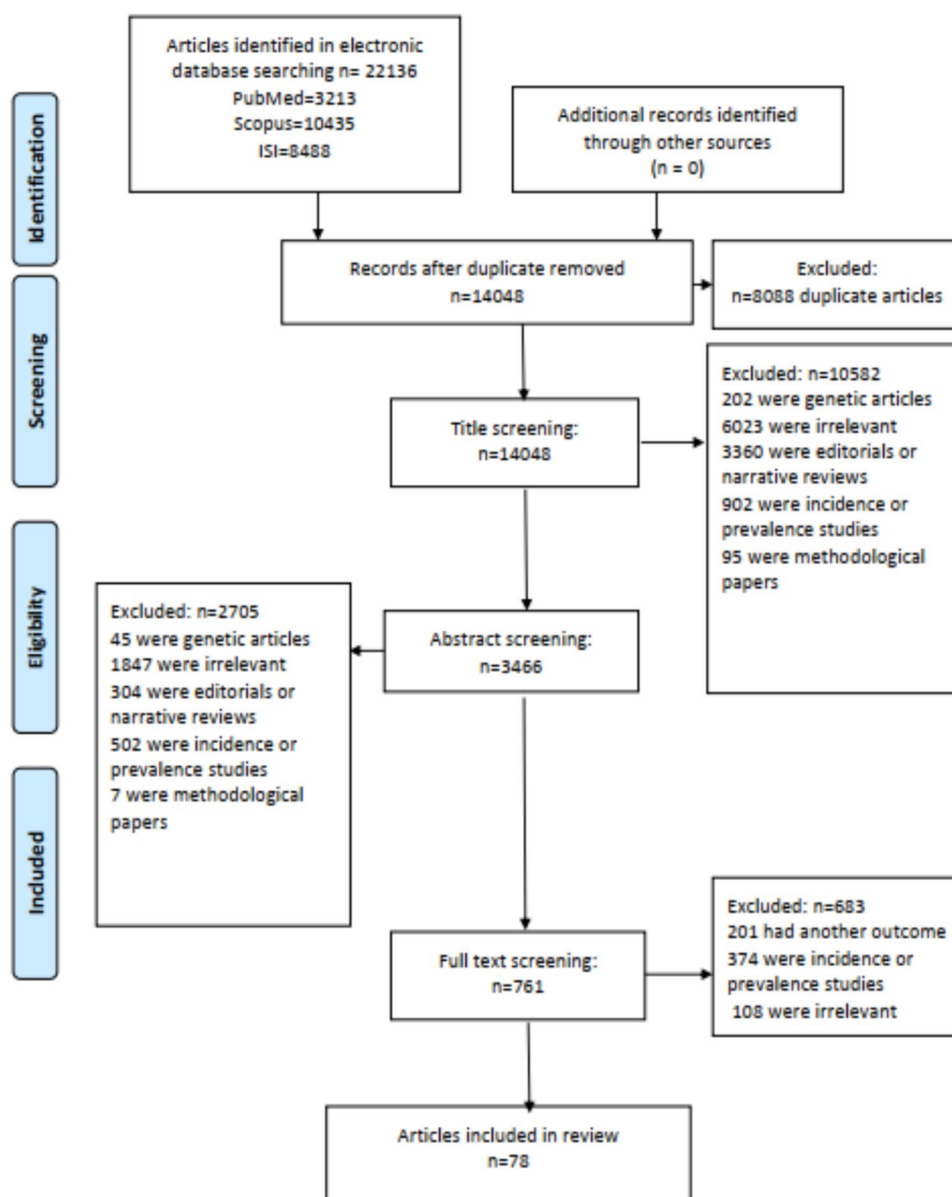
Study selection

Initially, we screened the titles and abstracts of all studies to identify those that met the inclusion criteria. For those that were difficult to determine with titles and abstracts only, full-text assessment was conducted. Two authors (MA and FKH) screened the full text final, and the decision was made for each study after reading the full text of all potentially eligible articles. In cases of disagreement, a third review author was consulted or it was resolved by discussion. In total, 22,136 articles were retrieved, of which 78 articles remained after the review process shown in Fig. 1.

Data extraction

A structured data extraction form was used to extract data from the papers. The extracted data included the last name of the first author, publication year, country, study population, sample size, age, and confounders. The extraction of data was performed independently by the same two review authors (MA and FKH) who conducted the study selection.

Fig. 1 Flow chart depicting the study selection process (screening)



Evaluating the quality of articles

The quality of studies was assessed using the Newcastle-Ottawa Scale (NOS) adapted for observational studies (Peterson et al. 2011). The NOS is based on three domains: selection of study groups, comparability of groups, and description of exposure and outcome. This scale, including eight items and star scores, assesses the quality of each study in each domain. All items except the comparability domain have one star (the maximum score based on stars for the comparability domain is 2). The total earned stars are calculated as the total quality score for each study. Based on these criteria, study quality was rated on a scale from one star, very poor, to 10 stars, high quality. Studies are rated as high (7–10), medium (5–6), or low quality (<4). Two review

authors (MA and FKH) independently completed the quality assessment. In cases of disagreement or items that remained unclear, a third review author was consulted.

Statistical analysis

The pooled OR and RR and the 95% confidence intervals were used to measure the association between smoking and MetS by adopting a random-effects meta-analytic model. We used adjusted estimates. Statistical heterogeneity was evaluated using Cochran's Q-test and I^2 statistic. Subgroup analysis was carried out according to the dose (cigarettes/day), duration of smoking, and time using cigarettes (current and former). Leave-one-out sensitivity analysis was performed to identify influential studies

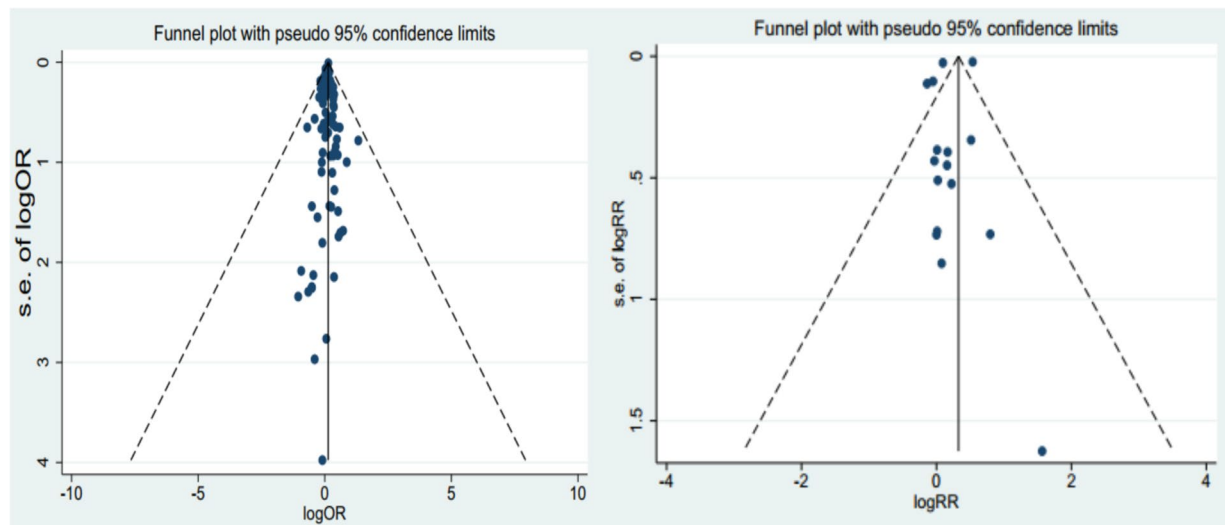


Fig. 2 Funnel plot for publication bias

in the meta-analysis. Publication bias was determined by funnel plots and Begg's and Egger's tests (Fig. 2). A P -value of < 0.05 was considered statistically significant. The analyses were performed using Stata software version 14.

Results

Study characteristics

The search strategy and the algorithm for study selection are shown in Fig. 1. A search by keywords, Medical Subject Headings (MeSH) terms, and Emtree terms identified a total of 22,136 studies. Subsequently, after identifying relevant studies and removing duplicates and considering the inclusion and exclusion criteria, 10,582, 2705, and 683 studies were excluded after reviewing their titles, abstracts, and full texts, respectively. Finally, 78 related studies in quality analysis were evaluated and met inclusion criteria. Of these, 14 studies were conducted in China, 10 studies in Korea, six studies in the United States, four studies in Japan, and four studies in Iran, with other studies in other parts of the world. A cutoff score of 7 or higher was considered as indicative of studies with high quality, and 5–6 was considered as studies with moderate quality. Seventy-one studies were scored 7 or higher, indicating high levels of quality. Five studies were in the range of 5–6, with moderate levels of quality. Supplementary Table 1 summarizes the characteristics of the selected studies.

Smoking and metabolic syndrome

Figure 3 presents the results of the random-effects meta-analysis and the pooled adjusted RR among cohort studies for the association between smoking and MetS stratified by current and former smoking. Based on the results, the pooled RR was 1.24 [95% CI: 1.05, 1.46], which represents a 24% increase in MetS. The pooled RR for current smoking was 1.51 [95% CI: 1.15, 1.99], which represents a 51% increase in MetS, but no association was found between former smoking and MetS (1.02 [95% CI: 0.90, 1.15]). Figure 4 presents the pooled adjusted OR among cross-sectional studies for the association between smoking and MetS stratified by current and former smoking. Based on results, the pooled OR was 1.11 [95% CI: 1.07, 1.15], which represents an 11% increase in MetS. The pooled OR for current smoking was 1.12 [95% CI: 1.07, 1.17], which represents a 12% increase in MetS, and the pooled OR for former smoking was 1.09 [95% CI: 1.02, 1.15], which represents a 9% increase in MetS. However, there is evidence of significant heterogeneity among cohort studies (current smoking: $I^2 = 98.4\%$; $P = 0.000$ and former smoking: $I^2 = 88.8\%$; $P = 0.000$) and cross-sectional studies (current smoking: $I^2 = 84.3\%$; $P = 0.000$ and former smoking: $I^2 = 55.6\%$; $P = 0.001$). Sensitivity analysis in the cohort studies showed that the studies by Park et al. (2021) and Sakboonyarat et al. (2022) were the source of observed heterogeneity. Moreover, sensitivity analysis showed that there was no single study as a potential source of heterogeneity in cross-sectional studies. We determined the possibility of publication bias using funnel plots (Fig. 2) and Begg's and Egger's tests in

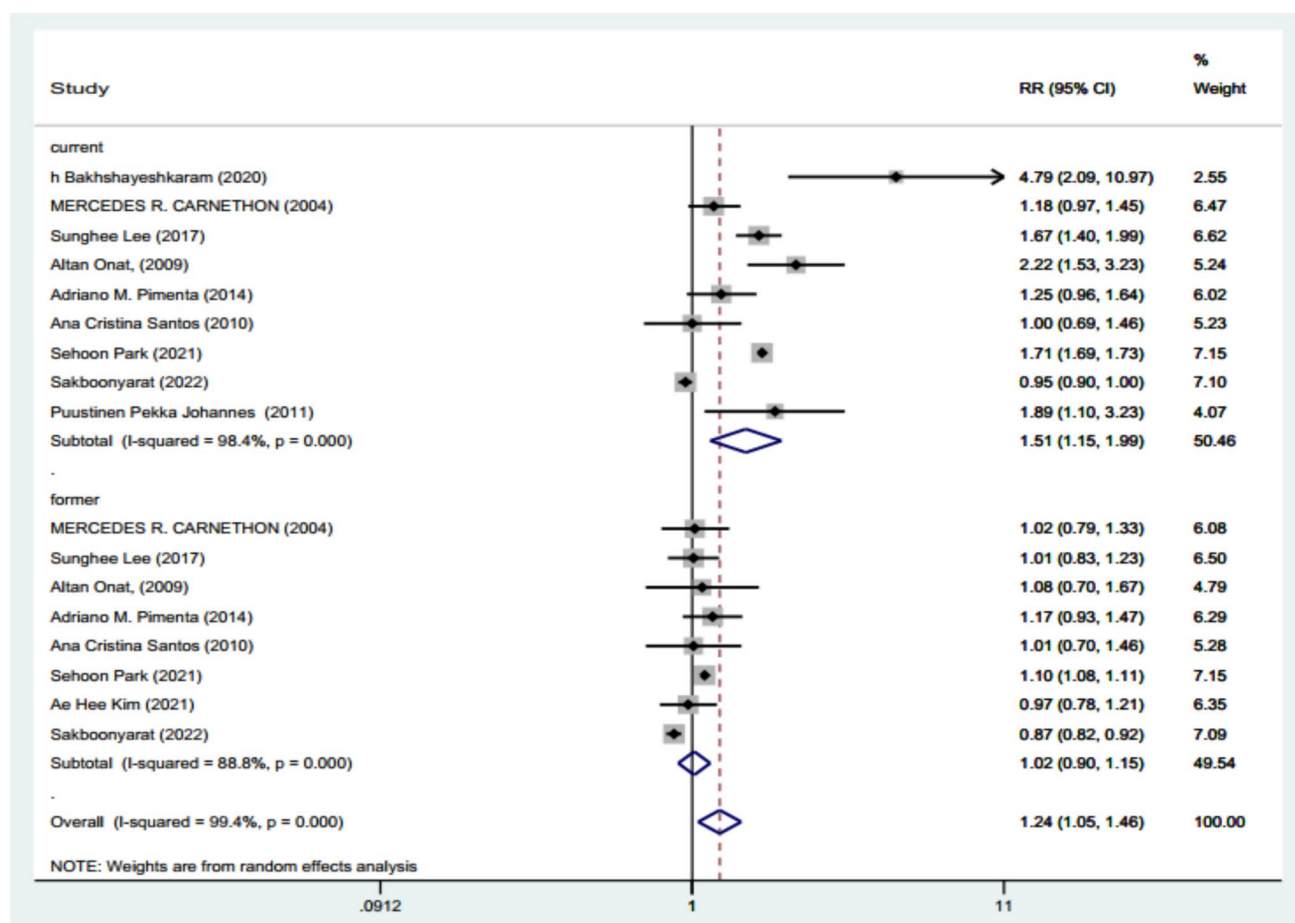


Fig. 3 Forest plot of the association between smoking and metabolic syndrome in cohort studies

cohort and cross-sectional studies. The studies are almost symmetrical, scattered on both sides of the vertical line showing the absence of publication bias. Based on Begg's ($P=0.166$) and Egger's ($P=0.448$) tests in cohort studies and Begg's ($P=0.533$) and Egger's ($P=0.157$) tests in cross-sectional studies, we found no evidence of publication bias.

Smoking dose and metabolic syndrome

The results of the relationship between smoking and MetS stratified by dose (< 10 cigarettes/day versus ≥ 10 cigarettes/day) are shown in Fig. 5. The pooled OR for the subgroups with less than 10 cigarettes/day was 1.17 [95% CI: 0.99, 1.38], which indicates that no association was found between smoking less than 10 cigarettes/day and MetS. The pooled OR for the subgroups with 10 or more cigarettes/day was 1.57 [95% CI: 1.04, 2.38], representing a 57% increase in MetS. Significant heterogeneity was found among subgroups with 10 or more cigarettes/day ($I^2=94.1\%$; $P=0.000$), but heterogeneity was lower in subgroups with less than 10

cigarettes/day ($I^2=50.2\%$; $P=0.061$). Sensitivity analysis showed that the studies by Kim et al. (2017, 2021) were the source of observed heterogeneity. Begg's ($P=0.669$) and Egger's ($P=0.119$) tests revealed no evidence of publication bias.

Duration of smoking and metabolic syndrome

Figure 6 presents the results of the pooled adjusted OR for the association between smoking and MetS stratified by duration of smoking (< 10 years versus ≥ 10 years of smoking). Based on the results, the pooled OR for the subgroups with less than 10 years of smoking was 0.96 [95% CI: 0.80, 1.16], which indicates that no association was found between subgroups with less than 10 years of smoking and MetS. The pooled OR for subgroups with 10 or more years of smoking was 1.17 [95% CI: 1, 1.39], which represents a 17% increase in MetS. There is no evidence of heterogeneity among subgroups with less than 10 years of smoking ($I^2=0.0\%$; $P=0.818$), but there is significant heterogeneity among subgroups with 10 or more years of smoking ($I^2=87.0\%$;

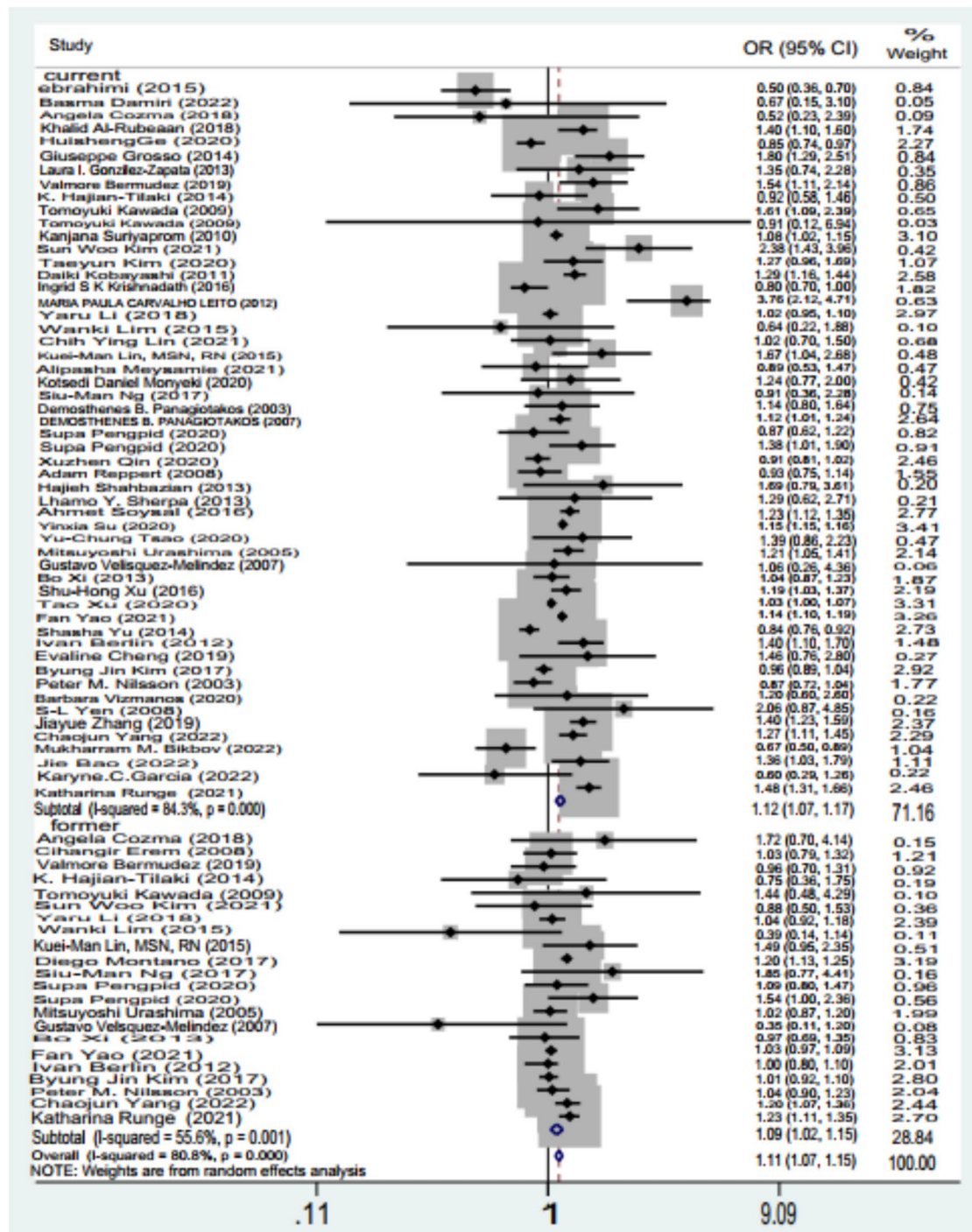


Fig. 4 Forest plot of the association between smoking and metabolic syndrome in cross-sectional studies

$P = 0.000$). Sensitivity analysis showed that the study by Kim et al. (2017) was the source of the observed heterogeneity. Based on Begg's ($P = 0.128$) and Egger's ($P = 0.291$) tests, we found no evidence of publication bias.

Discussion

This meta-analysis found a positive association between smoking and MetS in cohort and cross-sectional studies. However, the results of the subgroup of former smokers in cohort studies were not statistically significant. A positive relationship was shown between 10 or more cigarettes/day

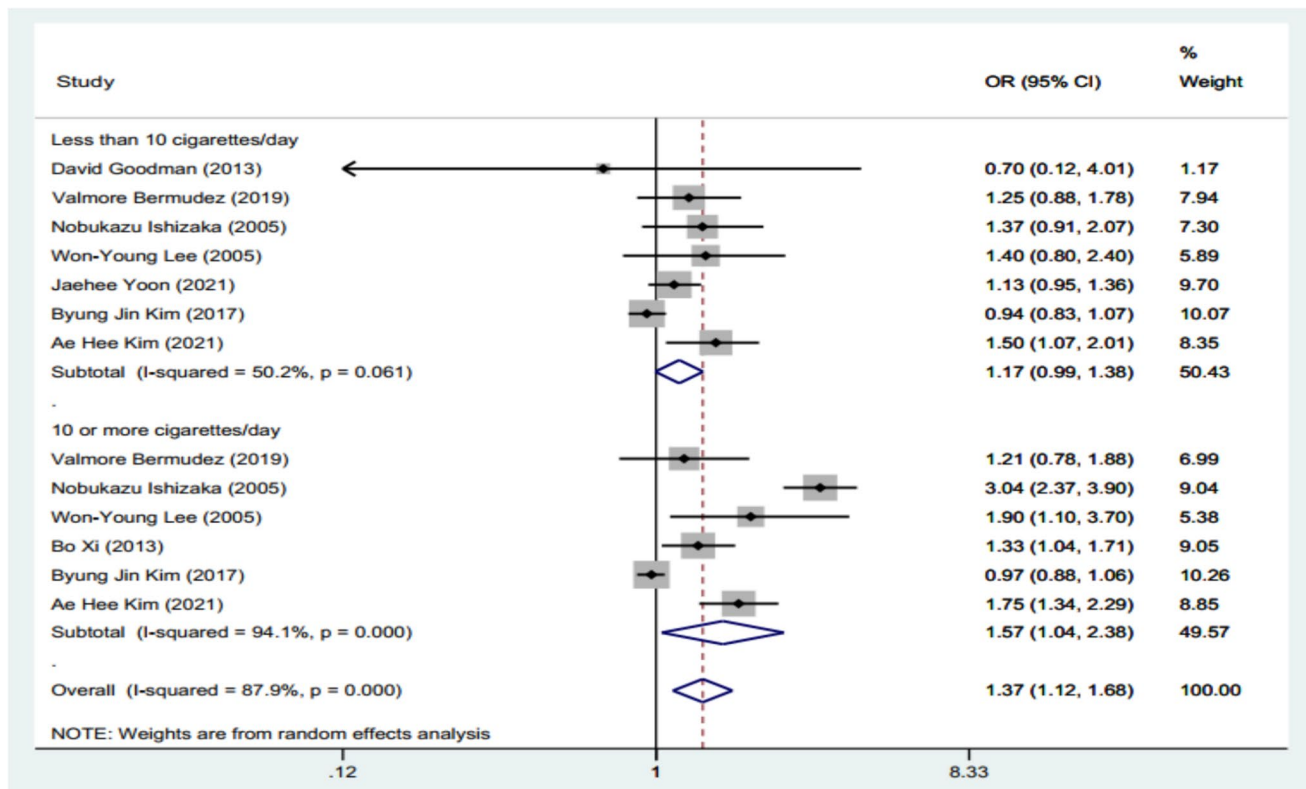


Fig. 5 Forest plot of the relationship between smoking dose and metabolic syndrome

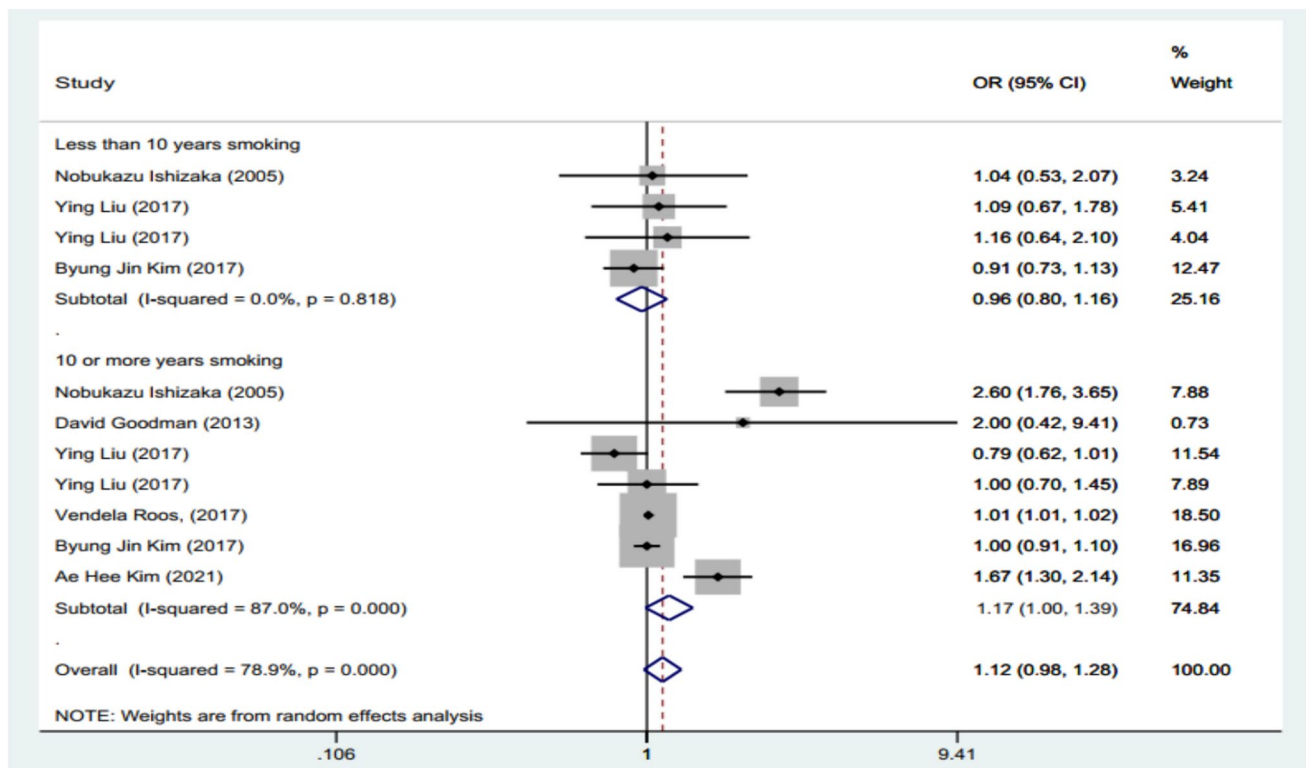


Fig. 6 Forest plot of the association between duration of smoking and MetS

and MetS, and it was found that 10 or more years of smoking increased the risk of MetS.

Many studies have shown a positive relationship between smoking and MetS (Bakhshayeshkaram et al. 2020; Damiri et al. 2021; Lee et al. 2017; Park et al. 2021). Smoking is associated with various diseases. Evidence suggests that smoking causes insulin resistance, which might increase the risk of developing type 2 diabetes, and is also associated with increased triglycerides, blood pressure, and other cardiovascular diseases, which are risk factors for MetS (Kondo et al. 2019; Mukamal 2006; Willi et al. 2007; van der Plas et al. 2023). Biologically, smoking causes the accumulation of visceral fat, which itself is an important factor in the onset of MetS through insulin resistance (Yun et al. 2012). In this study, the relationship between current and former smokers and MetS was investigated in both cross-sectional and cohort study designs, and it was shown that both former and current smoking had a positive relationship with MetS in cross-sectional studies. There are some possible explanations for why smoking cessation did not attenuate this relationship. One, the long-term effects of smoking on insulin resistance might cause vascular changes that negatively affect glucose uptake by skeletal muscle (Facchini et al. 1992). Second, current and former smoking reduces adiponectin levels, which contributes to insulin resistance (Matsuzawa et al. 2004; Miyazaki et al. 2003). However, an important point is that in cross-sectional studies, the main mechanism could not be shown due to the lack of temporality, and this relationship should be investigated by prospective studies. This meta-analysis, which was conducted on cohort studies, showed that only current smoking had a statistically significant relationship with MetS, which shows the importance of quitting smoking.

This study showed a positive relationship between smoking 10 or more cigarettes per day and MetS and also found that 10 or more years of smoking increased the risk of developing MetS, which is consistent with the existing evidence in this field (Ishizaka et al. 2005; Kim et al. 2021). In a previous meta-analysis (Sun et al. 2012), it was noted that the risk of developing MetS was stronger for men who were active smokers than for ex-smokers and also for heavy smokers, and the dose of smoking was examined only in men. In this study, we specifically did not apply a restriction on gender, and we examined the relationship between the dose and smoking in the general population, which showed that as long as smoking continues, its effects on MetS are visible, and a high dose of smoking was an important factor in the incidence of MetS.

It should be noted that one of the strengths of our study is that it provides an extensive and up-to-date review in this field. Unlike a previous study that reported the relationship between dose of smoking and MetS only in men, this study investigated this relationship in the general

population. Also, we included studies in the meta-analysis that considered and adjusted possible confounders such as sex, age, and alcohol use. Finally, the relationship between dose and duration of smoking was also investigated, and the results were in acceptable agreement with each other.

Nevertheless, the study has limitations. For instance, a significant degree of heterogeneity was found among studies, including heterogeneous populations and different settings in these populations, and differences in exposure dose. Also, due to the limited number of studies on the dose and duration of smoking and the risk of MetS, cohort and cross-sectional studies were combined. In addition, we only included studies published in English because we believed that high-quality studies were more likely to be published in English. Also, we only included studies published after 2000.

Conclusion

In this study, the relationship between smoking and MetS was investigated. The results showed that smoking was associated with the development of MetS; in addition, 10 or more years of smoking and 10 or more cigarettes/day increased the risk of MetS. This shows the importance of quitting smoking to prevent MetS.

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s10389-025-02461-w>.

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Authors' contributions MA conducted research. EMY provided essential reagents/provided essential materials. FKH performed the statistical analysis. EMY, MA, FKH, ZB, FSS, EKH, HA, KP, MD, and MK wrote the paper. MA had primary responsibility for the final content; EMY and MA had responsibility for all parts of the manuscript. All authors have approved the final article for publication.

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Declarations

Ethics approval Not applicable.

Informed consent Not applicable.

Consent to publication All the authors approved the publication of this manuscript.

Conflicts of interest The authors declare that they have no conflict of interest.

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