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THE ANALYSIS STUDY OF ASSOCIATION PERSISTENT SMOKING AND LONG TERM OUTCOMES IN PATIENTS WITH ATRIAL FIBRILATION: A COMPREHENSIVE SYSTEMATIC REVIEW

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ABSTRACT

Background: Atrial fibrillation (AF) is the most prevalent arrhythmia worldwide, with an estimated 5 million new cases reported in 2010 and a prevalence of 33 million by 2015. Over the past two decades, numerous prospective epidemiological studies have investigated the link between smoking and AF. This systematic review aims to evaluate the association persistent smoking and long term outcomes in patients with atrial fibrilation by analyzing available studies of the last 10 years.

Methods: The study adhered to PRISMA 2020 standards, examining English literature from 2014 to 2024. It excluded editorials, reviews from the same journal, and submissions without a DOI. PubMed, SagePub, SpringerLink, and Google Scholar were utilized as literature sources.

Result: Initially retrieving 360 articles from online databases (PubMed, SagePub, SpringerLink and Google Scholar) eight relevant papers were selected after three rounds of screening for full-text analysis.

Conclusion: Smoking is generally associated with an increased risk of AF. The extent of this risk is influenced by various factors, including age, gender, and other cardiovascular risk factors. These findings underline the importance of a nuanced approach to AF prevention and treatment, particularly concerning smoking habits.

Keyword: Smoking, risk, atrial fibrillation

INTRODUCTION

Atrial fibrillation (AF) is the most prevalent arrhythmia worldwide, with an estimated 5 million new cases reported in 2010 and a prevalence of 33 million by 2015.[1,2] This prevalence is expected to increase 2.5-fold over the next 50 years due to an aging population and a rising incidence of AF. The condition significantly raises the risk of ischemic heart disease, heart failure, stroke, and mortality, leading to increased healthcare costs and a reduced quality of life. Furthermore, AF exacerbates outcomes in patients with coronary artery disease, elevating the risk of myocardial infarction and other cardiovascular events.[3–5]

Over the past two decades, numerous prospective epidemiological studies have investigated the link between smoking and AF. While three key studies—two population-based and one hospital-based—found that current smokers had a significantly higher risk of developing AF than former or never smokers, other studies did not consistently observe this association.[6,7] This discrepancy has led to smoking being excluded as a predictor in some risk models, such as the Framingham Heart Study, though it was included in others, like the ARIC Study. These varying results are often attributed to differences in study design and data analysis methods.[8,9]

AF is influenced by several well-established risk factors, including age, heart failure, coronary artery disease, hypertension, diabetes, obesity, and elevated resting heart rate. Smoking, a modifiable risk factor, has been widely studied in relation to AF, yet the findings remain inconsistent. The inconsistency in results may be due to differences in study design, sample size, and the degree of adjustment for confounding factors, complicating efforts to draw definitive conclusions.[10,11]

Despite these inconsistencies, some evidence suggests that smokers are indeed at a higher risk of developing AF compared to non-smokers. However, the relationship between smoking and AF, particularly in terms of dose-response, smoking intensity, and duration, remains unclear. This ambiguity underscores the need for an updated systematic review to better understand these associations and to assess the impact of smoking on AF risk more thoroughly.[12–14]

This systematic review aims to evaluate the association persistent smoking and long term outcomes in patients with atrial fibrilation by analyzing available studies of the last 10 years.

METHODS

PROTOCOL

The author carefully followed the rules laid out in the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020. This was done to make sure the study met all its standards. The selection of this methodological approach was specifically aimed at ensuring the precision and reliability of the conclusions drawn from the investigation.

CRITERIA FOR ELIGIBILITY

This systematic to evaluate association persistent smoking and long term outcomes in patients with atrial fibrilation based on literatures of the last 10 years. This study meticulously analyzed data on literatures to provide insights and enhance patient treatment strategies. The primary objective of this paper is to highlight the collective significance of the identified key points.

Inclusion criteria for this study entail: 1) Papers must be in English, and 2) Papers must have been published between 2014 and 2024. Exclusion criteria comprise: 1) Editorials; 2) Submissions without a DOI; 3) Previously published review articles; and 4) Duplicate entries in journals.

SEARCH STRATEGY

The keywords used for this research smoking, risk, atrial fibrillation. The Boolean MeSH keywords inputted on databases for this research are: *("smoke"[MeSH Terms] OR "smoke"[All Fields] OR "smoke s"[All Fields] OR "smoked"[All Fields] OR "smokes"[All Fields] OR "smoking"[MeSH Terms] OR "smoking"[All Fields] OR "smokings"[All Fields] OR "smoking s"[All Fields]) AND ("risk"[MeSH Terms] OR "risk"[All Fields]) AND ("atrial fibrillation"[MeSH Terms] OR ("atrial"[All Fields] AND "fibrillation"[All Fields]) OR "atrial fibrillation"[All Fields]).*

DATA RETRIEVAL

The authors assessed the studies by reviewing their abstracts and titles to determine their eligibility, selecting relevant ones based on their adherence to the inclusion criteria, which aligned with the article's objectives. A consistent trend observed across multiple studies led to a conclusive result. The chosen submissions had to meet the eligibility criteria of being in English and a full-text.

This systematic review exclusively incorporated literature that met all predefined inclusion criteria and directly pertained to the investigated topic. Studies failing to meet these criteria were systematically excluded, and their findings were not

considered. Subsequent analysis examined various details uncovered during the research process, including titles, authors, publication dates, locations, study methodologies, and parameters.

QUALITY ASSESSMENT AND DATA SYNTHESIS

Each author independently evaluated the research presented in the title and abstract of the publication to determine which ones merited further exploration. The subsequent stage involved assessing all articles that met the predefined criteria for inclusion in the review. Decisions on including articles in the review were based on the findings uncovered during this evaluation process.

Table 1. Article Search Strategy

Database	**Strategi Pencarian**	***Hits***
Pubmed	*("smoke"[MeSH Terms] OR "smoke"[All Fields] OR "smoke s"[All Fields] OR "smoked"[All Fields] OR "smokes"[All Fields] OR "smoking"[MeSH Terms] OR "smoking"[All Fields] OR "smokings"[All Fields] OR "smoking s"[All Fields]) AND ("risk"[MeSH Terms] OR "risk"[All Fields]) AND ("atrial fibrillation"[MeSH Terms] OR ("atrial"[All Fields] AND "fibrillation"[All Fields]) OR "atrial fibrillation"[All Fields])*	100
Science Direct	*((smoking) AND (risk)) AND (atrial fibrillation)*	150
Sagepub	*((smoking) AND (risk)) AND (atrial fibrillation)*	50
Google Scholar	*((smoking) AND (risk)) AND (atrial fibrillation)*	160

Table 2. JBI *Critical appraisal of Study*

Parameters	Lubitz (2015)	Albertsen (2014)	Suzuki (2015)	Zuo (2018)	Ahmad (2018)	Lee (2021)	Kwon (2016)	Wu (2024)
1. Bias related to temporal precedence Is it clear in the study what is the "cause" and what is the "effect" (ie, there is no confusion about which variable comes first)?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. Bias related to selection and allocation Was there a control group?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
3. Bias related to confounding factors Were participants included in any comparisons similar?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
4. Bias related to administration of intervention/exposure Were the participants included in any comparisons receiving similar treatment/care, other than the exposure or intervention of interest?	No.	No.	No.	No.	No.	No.	No.	No.
5. Bias related to assessment, detection, and measurement of the outcome Were there multiple measurements of the outcome, both pre and post the intervention/exposure?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Were the outcomes of participants included in any comparisons measured in the same way?	No.	No.	No.	No.	No.	No.	No.	No.
Were outcomes measured in a reliable way?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
6. Bias related to participant retention Was follow-up complete and, if not, were differences between groups in terms of their follow-up adequately described and analyzed?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
7. Statistical conclusion validity Was appropriate statistical analysis used?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

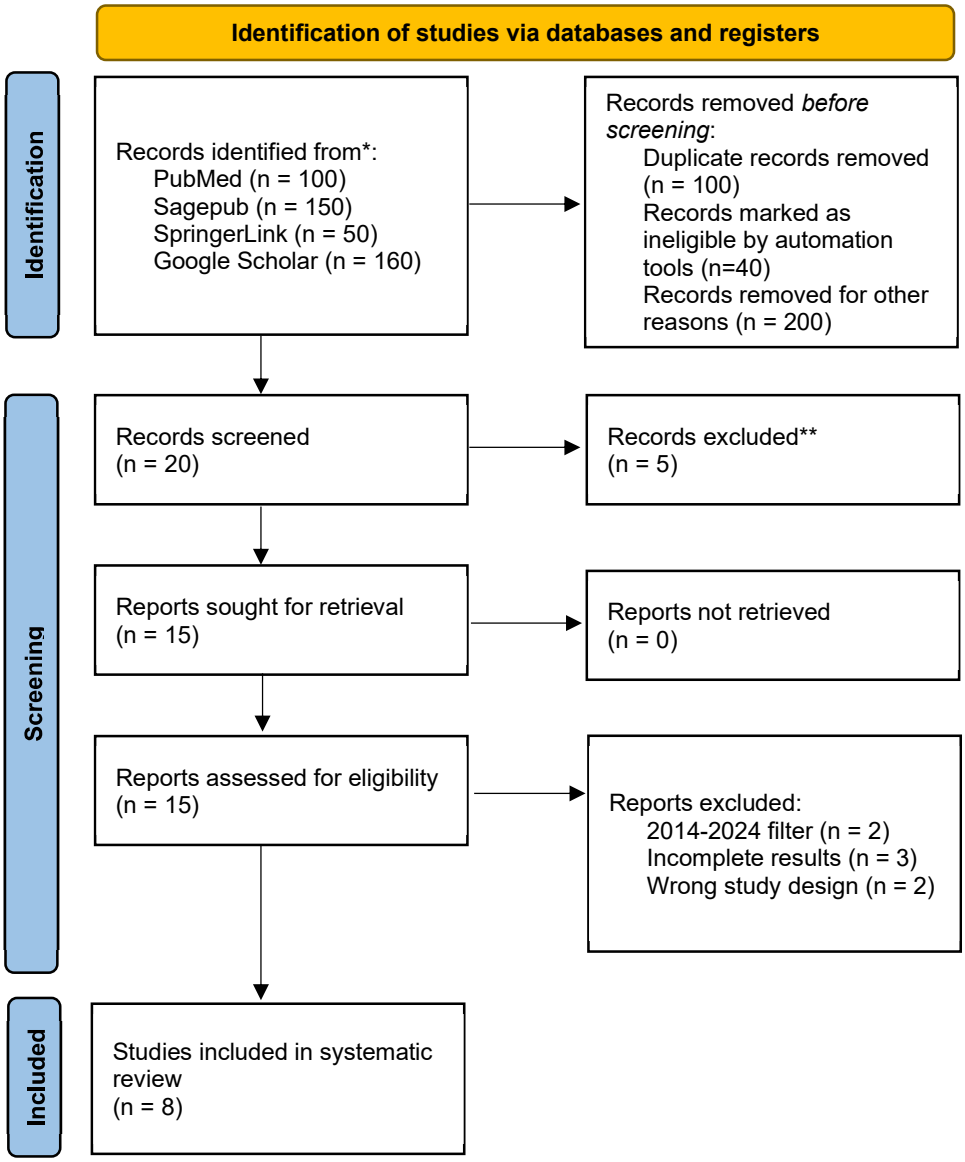

Figure 1. Article search flowchar

RESULT

The initial number of articles retrieved from online databases (PubMed, SagePub, SpringerLink, and Google Scholar) is 360 articles. After conducting three levels of screening, eight articles that directly relate to the current systematic review have been chosen for further assessment through full-text reading and analysis. Table 1 presents the selected literature included in this analysis.

Table 1. The literature included in this study

No.	Author	Origin	Method	Sample	Result
1.	Lubitz, et al.[15] (2015)	USA	Retrospective cohort	439 patients	In a study of 439 patients diagnosed with atrial fibrillation (AF) during secondary precipitant events, cardiothoracic surgery (30%), infection (23%), non-cardiothoracic surgery (20%), and acute myocardial infarction (18%) were the most common triggers. Among 846 individuals without permanent AF, AF recurred in 544 cases, with 5-, 10-,

					and 15-year recurrence rates of 42%, 56%, and 62% in those with secondary precipitants compared to 59%, 69%, and 71% in those without. The stroke risk and mortality were similar between groups, with hazard ratios of 1.13 and 1.00, respectively. However, the heart failure risk was reduced in those with secondary precipitants (hazard ratio, 0.74).
2.	Albertsen, et al.[16] (2014)	Denmark	Retrospective cohort	3,161 patients	In patients with atrial fibrillation (AF), 34% were current smokers and 37% were former smokers. After adjusting for vitamin K antagonist treatment, current heavy smoking (>25 g/day) was linked to a significantly higher risk of thromboembolism or death, with hazard ratios (HRs) of 3.13 for women and 2.73 for men. Even after adjusting for established risk factors, the associations remained strong, with HRs of 3.64 for women and 2.17 for men. A sensitivity analysis confirmed that smoking was still strongly associated with thromboembolism or death, even after excluding individuals with a cancer diagnosis during follow-up.
3.	Suzuki et al.,[17] (2015)	Japan	Retrospective cohort	15,221 patients	During a follow-up period of 2.0 ± 2.1 years, new atrial fibrillation (AF) occurred at an incidence rate of 9.0 per 1000 patient-years in smokers, compared to 5.0 per 1000 patient-years in non-smokers. Smokers were independently associated with a higher risk of developing new AF, with a hazard ratio (HR) of 1.47. Current smokers had an even higher risk (HR 1.81), and those with a Brinkman index of 800 or more were also at increased risk (HR 1.69). The risk did not significantly differ based on the Brinkman index in current smokers.
4.	Zuo, et al.[18] (2018)	Norway	Retrospective cohort	538 patients	In a study with a median follow-up of 11 years, 538 participants developed atrial fibrillation (AF). Current smoking was associated with an increased risk of AF (hazard ratio [HR]: 1.41), while former smoking did not show a significant association (HR: 1.03). Plasma cotinine levels also indicated a higher risk of AF, with an HR of 1.40 for participants with cotinine levels ≥85 nmol/L compared to those with lower levels. The risk increased with higher plasma cotinine levels up to 1199 nmol/L (HR: 1.55), after which it plateaued.

5.	Ahmad, et al.[19] (2018)	USA	Retrospective cohort	954 patients	During the follow-up period, 954 new cases of atrial fibrillation (AF) were identified, with 9.5% in smokers and 7.8% in non-smokers ($p < 0.001$). Initially, smoking was associated with a 15% increased risk of AF (odds ratio [OR]: 1.15), but this association became non-significant after adjusting for cardiovascular risk factors (OR: 1.12). Subgroup analyses revealed variations: the risk was higher in younger individuals (OR: 1.31) compared to older individuals (OR: 0.99), and in those with prior cardiovascular disease (OR: 1.18) compared to those without (OR: 1.06). The association was significant in Black participants (OR: 1.51) but not in White participants (OR: 0.99), though the interaction p-value was not statistically significant ($p = 0.65$).
6.	Lee, et al.[20] (2021)	South Korea	Retrospective cohort	97,637 patients	In a study of 97,637 patients with an average age of 61 years and a mean CHA2DS2-VASc score of 2.3, 6.9% stopped smoking after an atrial fibrillation (AF) diagnosis. With a mean follow-up of 3.2 years, quitters had significantly lower risks of ischemic stroke (hazard ratio [HR]: 0.702) and all-cause death (HR: 0.842) compared to current smokers. They also had reduced risks of fatal ischemic stroke (HR: 0.454) and death from cerebrovascular events (HR: 0.664).
7.	Kwon, et al.[21] (2016)	USA	Retrospective cohort	1222 patients	In a study of 1222 participants from the ARIC study (mean age 63.4) and 756 participants from the CHS study (mean age 79.1) with incident atrial fibrillation (AF), with mean follow-ups of 6.9 and 5.7 years respectively, there were 332 and 335 composite events. Current smokers had a higher incidence of the composite endpoint in the ARIC cohort (hazard ratio [HR]: 1.65) but not in the CHS cohort (HR: 1.05). In ARIC, adding current smoking status did not improve risk prediction beyond the CHA2DS2-VASc score. No significant associations were found with alcohol consumption or BMI concerning cardiovascular outcomes in AF patients in either cohort.
8.	Wu, et al.[22] (2024)	China	Retrospective cohort	85 patients	The Mendelian randomization (MR) analysis found that smoking initiation is associated with a higher likelihood of developing atrial fibrillation (odds

					ratio [OR]: 1.11; 95% confidence interval [CI]: 1.02–1.20; p=0.013). The analysis used 85 SNPs as instrumental variables from GWAS pooled data. The MR-Egger method showed no evidence of horizontal pleiotropy (Egger intercept = -0.005; p=0.371), and the 'leave-one-out' sensitivity analysis confirmed the robustness of the results. Reverse MR analysis found no effect of atrial fibrillation on smoking initiation (OR=1.00; 95% CI: 0.99–1.02; p=0.684).

Lubitz, et al.[15] (2015) showed that that AF recurrence is common, regardless of the presence of secondary precipitants, and that stroke and mortality risks remain similar. Future research should focus on whether enhanced arrhythmia surveillance or adherence to general AF management in patients with reversible precipitants could reduce morbidity.

Albertsen, et al.[16] (2014) concludes that smoking is associated with a higher risk of thromboembolism or death in AF patients, with the strongest associations observed among women.

Suzuki et al.,[17] (2015) concludes that smoking is independently associated with the development of new AF, and cessation of smoking is important for prevention. The study's limitations include its single hospital-based design and relatively short observation period.

Zuo, et al.[18] (2018) concludes that current smoking is linked to a higher risk of developing AF, a finding supported by plasma cotinine measurements.

Ahmad, et al.[19] (2018) suggests that the association between smoking and AF may be influenced by cardiovascular risk factors and highlights the variability in this relationship across different subgroups.

Lee, et al.[20] (2021) suggest that quitting smoking can lower the risk and severity of ischemic stroke and reduce the incidence of cerebrovascular events in patients with new-onset AF.

Kwon, et al.[21] (2016) showed that smoking increased the risk of ischemic stroke or cardiovascular death in the ARIC cohort (mainly aged 65–74 years) but not in the CHS cohort (mainly aged ≥75 years). The addition of smoking to the CHA2DS2-VASc score did not enhance risk prediction for these outcomes.

Wu, et al.[22] (2024) concludes that smoking initiation significantly impacts the risk of atrial fibrillation, but atrial fibrillation does not influence smoking initiation, highlighting the genetic link between smoking and atrial fibrillation.

DISCUSSION

The relationship between smoking and atrial fibrillation (AF) is complex and multifaceted, with numerous studies pointing to smoking as a significant risk factor for the development and progression of AF. The evidence gathered from various retrospective cohort studies, such as those by Lubitz et al. (2015) and Suzuki et al. (2015), suggests that smoking may increase the risk of AF, but the strength and consistency of this association vary across different populations and study designs.[15,17,23]

AF recurrence appears to be a common issue among patients, regardless of whether they have secondary precipitating events, such as surgery or infections. While Lubitz et al. (2015) showed that smoking does not significantly impact AF recurrence or related risks like stroke and mortality, the overall influence of smoking on these outcomes remains a topic of ongoing research.[15,24]

In several studies, heavy smoking has been linked to an increased risk of adverse outcomes in AF patients, such as thromboembolism or death, particularly in certain subgroups like women. This was notably observed by Albertsen et al. (2014), highlighting the importance of considering smoking status in the broader context of AF management, especially in populations more vulnerable to cardiovascular complications.[16]

Other research, such as the study by Suzuki et al. (2015), supports the notion that smoking independently raises the risk of developing AF, underscoring the need for effective smoking cessation interventions as a preventive strategy. However, the

varying methodologies and follow-up periods in these studies mean that generalizability can be challenging, and there remains a need for further research to solidify these findings.[17,25]

Some studies, including that by Zuo et al. (2018), have employed biomarkers like plasma cotinine levels to strengthen the association between smoking and AF risk, adding a layer of reliability to the findings. Yet, these studies also highlight the need for more detailed investigations into how smoking intensity and duration influence AF risk.[18,26]

The relationship between smoking and AF also appears to be influenced by other cardiovascular risk factors, with Ahmad et al. (2018) suggesting that the association is stronger in certain subgroups, such as younger individuals or those with preexisting cardiovascular conditions. This variability points to the need for personalized approaches in assessing and managing AF risk in smokers.[19]

Additionally, evidence from Lee et al. (2021) suggests that smoking cessation can significantly lower the risk of severe outcomes, such as ischemic stroke and cardiovascular events, in AF patients. This reinforces the role of smoking cessation as a crucial component of AF management.[20]

Recent genetic studies, like those by Wu et al. (2024), have even provided evidence for a causal link between smoking initiation and AF development, though this relationship does not appear to work in reverse. Such findings emphasize the importance of smoking prevention and cessation in reducing AF incidence.[22]

CONCLUSION

Smoking is generally associated with an increased risk of AF. The extent of this risk is influenced by various factors, including age, gender, and other cardiovascular risk factors. These findings underline the importance of a nuanced approach to AF prevention and treatment, particularly concerning smoking habits.

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