

ORIGINAL ARTICLE

A meta-analysis study on the association between smoking and male pattern hair loss

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Abstract

Background: Smoking—which often refers to recreational consumption of the nicotine-containing tobacco—is deemed a risk factor for both the development of and worsening of androgenetic alopecia (AGA). However, there is no published meta-analysis study on the effect of smoking on AGA; so, we quantitatively synthesized the evidence base pertaining to the recreational activity and this form of hair loss in men.

Methods: We systematically searched PubMed and Scopus to identify published studies with suitable data, and pairwise meta-analyses were conducted.

Results: Our search identified eight studies—and the data thereof were used across four meta-analyses. We found that ever smokers are significantly ($p < 0.05$) more likely, than never smokers, to develop AGA (pooled odds ratio (OR) = 1.82, 95% confidence interval (CI): 1.55–2.14). Our results showed that the odds of developing AGA are significantly ($p < 0.05$) higher in men who smoke at least 10 cigarettes per day, than in their counterparts who smoke up to 10 cigarettes per day (pooled OR = 1.96, 95% CI: 1.17–3.29). For men with AGA, the odds of disease progression are significantly ($p < 0.05$) higher among ever smokers than in never smokers (pooled OR = 1.27, 95% CI: 1.01–1.60). We found no significant ($p \geq 0.05$) association between smoking intensity and disease progression.

Conclusions: Findings from the current study—which is the first meta-analysis to our knowledge reviewing the association between AGA and the extent of smoking, can guide further research and update clinical practice guidelines.

1 | INTRODUCTION

Androgenetic alopecia (AGA), the most common form of hair loss, is a cosmetic concern for many people worldwide.^{1,2} Furthermore, this clinically benign condition—which is also referred to as pattern baldness or pattern hair loss—can negatively impact psychosocial well-being.³ Pattern hair loss manifests differently between men and women⁴—a reason for which numerous AGA trials, and meta-analyses,

are often sex-specific. There are various risk factors for both the development and worsening of AGA; one such factor is smoking,⁵ albeit, the evidence thereof insofar as pattern hair loss is inconsistent—as pointed out by Babadjouni et al.⁶ and Kavadya et al.⁷ For instance, Salem et al.⁸ found an association between the development of AGA in males and smoking; however, Severi et al.⁹ reported negative results.

Smoking very often refers the burning of tobacco, a nicotine-containing plant—where nicotine is a highly addictive substance with

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various harmful effects. This harmful substance can be consumed recreationally through various “vehicles” such as cigar, cigarettes, and pipes, to name a few.¹⁰

Unraveling the link between smoking and AGA is worthwhile because, for one, over a billion individuals partake in this recreational activity and, for another, it is a completely preventable risk factor.¹⁰

No meta-analyses pertaining to pattern hair loss (AGA) and smoking has been published hitherto. The aim of the current study was to quantitatively synthesize the evidence base regarding smoking and male AGA.

2 | METHODS

To unravel the relationship between smoking and male pattern hair loss, we first obtained data from studies identified through a systematic search of the literature along with reference mining.

2.1 | Search

On August 4, 2023, we searched PubMed (search term: (“hair loss”[Title/Abstract] OR “alopecia”[Title/Abstract] OR bald*[Title/Abstract]) OR (androgenetic alopecia[MeSH Terms])) AND (“tobacco”[Title/Abstract] OR “nicotine”[Title/Abstract] OR (Tobacco Smoking[MeSH Terms])) and Scopus (search term: (“Smoking” OR “tobacco” OR “nicotine”) AND (“Hair Loss” OR “Alopecia”)). Deduplication was aided by Systematic Review Accelerator (SRA)-Deduplicator¹¹; the screening of both titles/abstracts and full texts were conducted with the Rayyan software.¹² Extracted data were organized into spreadsheets. The two stages of screening and data extraction were conducted by two authors (AKG and MAB); any discrepancy was resolved through discussion with a third author (MT).

2.2 | Eligible studies

In context of the population, intervention, comparator, and outcome (i.e., PICO) framework, our analyses used studies that provided data on effect of smoking status (e.g., smoking vs. no smoking) and intensity (e.g., less smoking vs. more smoking) (i.e., the interventions/comparators(I/C)) on developing AGA and worsening thereof (i.e., the outcomes(O)) in men (i.e., population (P)). Studies published in non-English language were excluded from our analyses.

2.3 | Statistical analyses

We conducted pairwise meta-analyses under a common effect model as we assumed one true effect; significance level was set

to 5%. We used the *meta* package in RStudio,¹³ and pooled estimates were presented as forest plots.

3 | RESULTS

Our search identified eight studies which contained data that were suitable for our meta-analyses^{8,9,14–19}; the schematic for our search process is summarized in Figure 1. Table 1 provides a brief description of the eight studies. All the studies were of observational design as they were either case-control or cross-sectional studies. The data we obtained enabled us to conduct pairwise meta-analyses for (1) impact of smoking status on developing male AGA, (2) effect of less smoking versus more smoking on developing male AGA, (3) impact of smoking status on progressing from milder to more severe stages of male AGA, and (4) effect of less smoking versus more smoking on progressing from milder to more severe male AGA. For our analyses, ever-smokers referred to current and former smokers; pooled effects were quantified as odds ratios (ORs)—and their corresponding 95% confidence interval (CI) were reported.

3.1 | Analysis 1: Effect of ever smoking versus never smoking on risk of developing male pattern baldness

Data from six of the eight studies^{8,9,14,16,18,19} were pooled to determine the effect of ever smoking—relative to never smoking—on development of pattern hair loss in men. The forest plot in Figure 2 shows that ever smoking—compared to never smoking—is significantly ($p < 0.05$) associated with developing male AGA (pooled OR = 1.84, 95% CI: 1.55–2.14).

3.2 | Analysis 2: Effect of less smoking (i.e., up to 10 cigarettes/day) versus more smoking (i.e., at least 10 cigarettes/day) on risk of developing male pattern baldness

We analyzed data from two of the eight studies^{8,19} and found that smoking up to 10 cigarettes daily—relative to smoking at least 10 cigarettes per day—is associated with higher likelihood of developing male pattern baldness (pooled OR = 1.96, 95% CI: 1.17–3.29) (Figure 3).

3.3 | Analysis 3: Effect of ever smoking versus never smoking on progressing from milder (Stages I–III) to more severe (Stages IV–VII) male pattern baldness

After pooling data from four of the eight studies^{8,14,15,17} we found that ever smoking—relative to never smoking—is significantly ($p < 0.05$) associated with a greater odd of progressing from milder

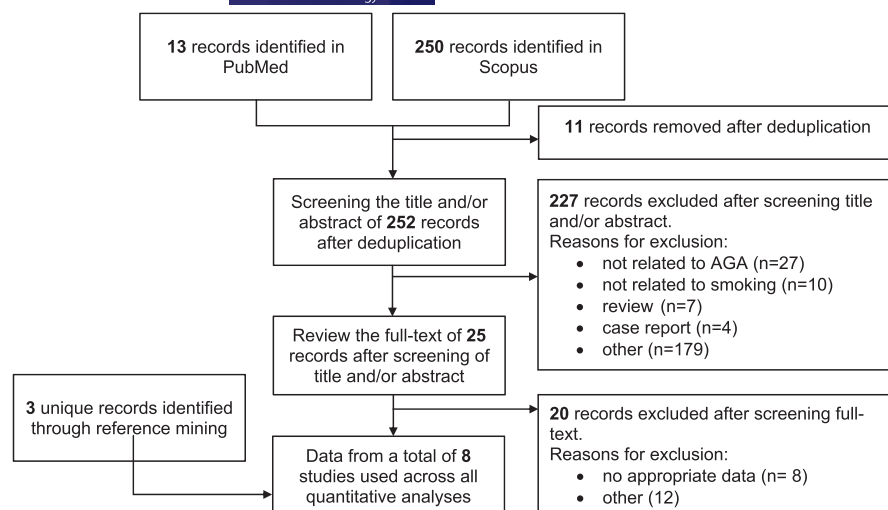


FIGURE 1 Identification of studies used for meta-analyses. To obtain suitable data for all meta-analyses, we electronically searched for relevant published studies through PubMed and Scopus; in addition, reference mining was also conducted. All searches were conducted on August 4, 2023.

TABLE 1 Brief description of studies included in meta-analyses.

Reference	Year of publication	Objective ^a	Study design
Severi et al. ⁹	2003	The authors attempted to investigate the association between AGA and smoking as a primary objective	Case-control
Su et al. ¹⁵	2007	To determine prevalence of AGA—and the different types thereof—in Taiwanese men	Cross-sectional
Arias-Santiago et al. ¹⁶	2010	To analyze the presence of carotid atheromas and risk factors of cardiovascular disease in persons with AGA	Case-control
Lai et al. ¹⁷	2013	To estimate the prevalence of AGA—and the different severity types thereof—in Taiwanese men	Cross-sectional
Fortes et al. (2018) ¹⁹	2018	To determine whether there is an association between Mediterranean diet and AGA	Case-control
Vora et al. ¹⁸	2019	To study the association between coronary artery disease and AGA	Cross-sectional
Danesh-Shakiba et al. ¹⁴	2020	To determine the association between AGA and anthropometric indices, lifestyle habits and blood pressure	Case-control
Salem et al. ⁸	2021	To characterize the clinical and demographic features of early-onset AGA in smokers and nonsmokers, and to evaluate whether the smoking affects the prevalence of AGA	Cross-sectional

Abbreviation: AGA, androgenetic alopecia.

^aThe objective presented in this table refers to the respective studies' primary objective. So, for studies whose primary objective did not directly pertain to AGA and smoking, data from such studies were still used as long as raw data on AGA and smoking were provided. For example, Su et al. (2007) did not primarily investigate smoking and AGA, nonetheless their paper contained data on smoking and male pattern hair loss.

to more severe pattern hair loss in men (i.e., from any stages within Norwood-Hamilton I–III to any stages within Norwood-Hamilton V–VII) as the pooled odds ratio for the comparison was 1.27 (95% CI: 1.01–1.50) (Figure 4).

3.4 | Analysis 4: Effect of less smoking (i.e., up to 20 cigarettes/day) versus more smoking (i.e., at least 20 cigarettes/day) on progressing from milder (Stages I–III) to more severe (Stages IV–VII) male pattern baldness

Pooled analysis of data from two of the eight studies^{8,15} showed no significant ($p \geq 0.05$) association between smoking intensity and worsening of AGA in men (pooled OR=1.01, 95% CI: 0.69–1.50) (Figure 5).

No studies of randomized design were available and a major reason for this is likely ethical; as per the "Declaration of Helsinki,"²⁰ research participants cannot be randomized to harm. So, any empirical data on smoking and AGA would be observational at best. However, comparing the effect of smoking cessation on pattern hair loss is ethical and can be done using a randomized study design. Randomized evidence from such trials can somewhat verify observational results—including those of the current study; for example, the inverse (or converse) estimates for effect of smoking cessation on reducing severity of AGA can be close to the "true effect" of smoking versus quitting smoking on worsening of AGA.

Our analyses have some limitations. Because of the cross-sectional and case-control designs, temporality could not be established and therefore the estimates could be subject to time-varying confounding. Another limitation is that exact nicotine levels could not be ascertained from the given data. So, for our analyses that

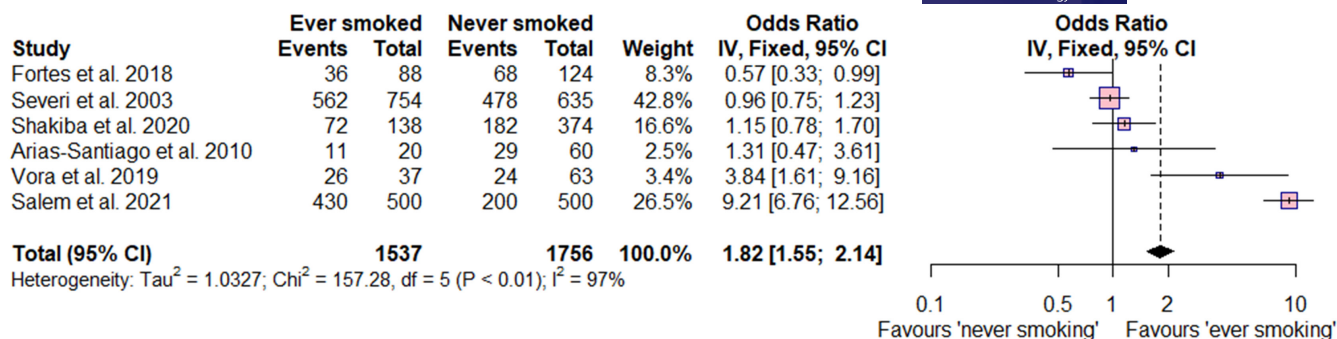


FIGURE 2 Forest plot for effect of smoking status on developing pattern hair loss in men. This meta-analysis shows that men who ever smoke have significantly ($p < 0.05$) higher odds of developing pattern hair loss than their counterparts who never smoke (pooled odds ratio = 1.82, 95 confidence interval: 1.55–2.14).

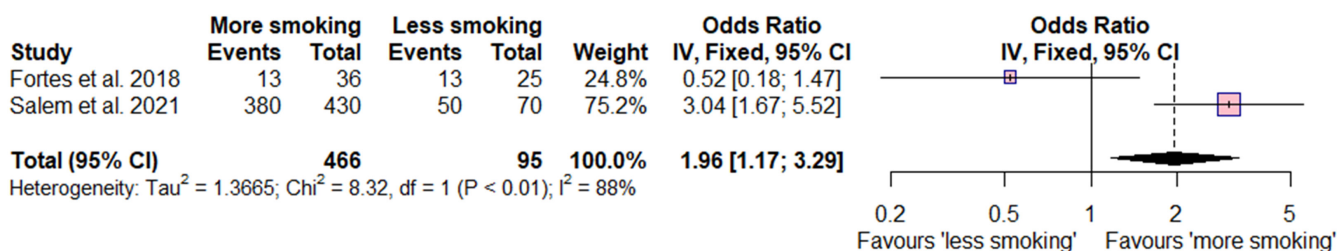


FIGURE 3 Forest plot for effect of smoking intensity on development of pattern hair loss in men. This meta-analysis shows that men who smoke more (i.e., at least 10 cigarettes per day) have significantly ($p < 0.05$) higher odds of developing pattern hair loss than their counterparts who smoke less (i.e., up to 10 cigarettes per day) (pooled odds ratio = 1.96, 95 confidence interval: 1.17–3.29).

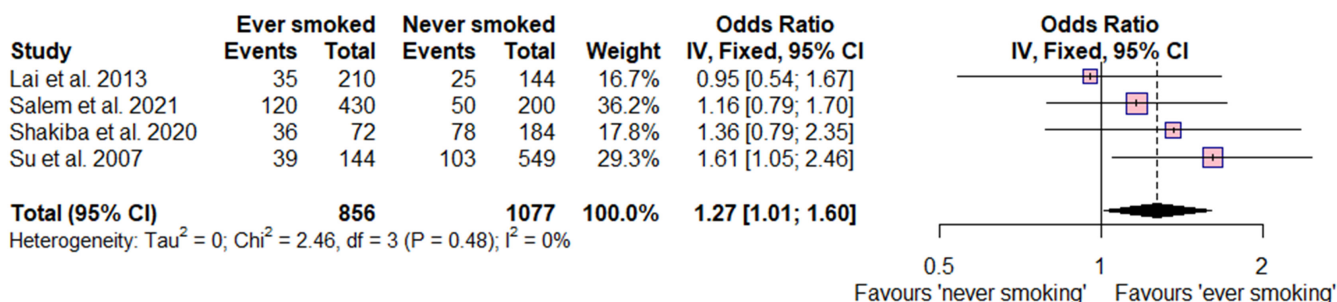


FIGURE 4 Forest plot for the effect of smoking status on progression from milder to more severe pattern hair loss in men. This meta-analysis shows that, across men who have pattern hair loss, the odds of disease progression (i.e., progression from any stage between Norwood–Hamilton I–III, to any stage between Norwood–Hamilton V–VII) is significantly ($p < 0.05$) higher among those who ever smoke than in those who never smoke (pooled odds ratio = 1.27, 95 confidence interval: 1.01–1.60).

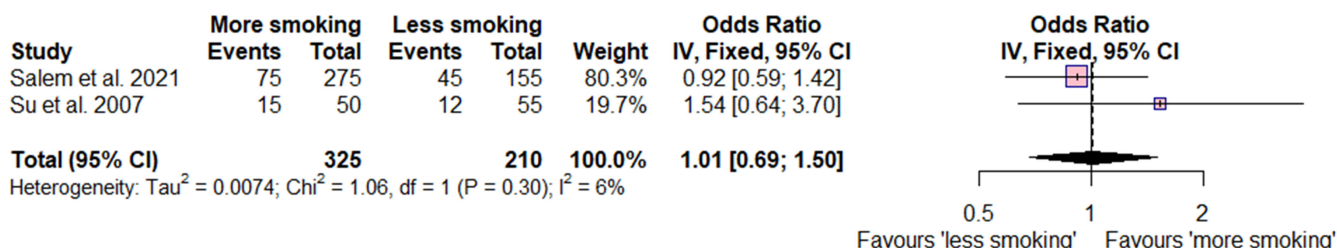


FIGURE 5 Forest plot for effect smoking intensity on progressing from milder to more severe male pattern baldness. This meta-analysis shows that there is no significant ($p \geq 0.05$) association between smoking intensity (i.e., smoking up to 20 cigarettes per day vs. smoking at least 20 cigarettes per day) and worsening of pattern hair loss in men (i.e., progression from any stage between Norwood–Hamilton I–III, to any stage between Norwood–Hamilton V–VII).

pertained to smoking intensity, we assumed that cigarettes across the different studies contained similar nicotine levels.

4 | DISCUSSION

The current study quantitatively synthesized observational evidence pertaining to smoking and male pattern hair loss. Our pairwise meta-analyses used data from a total of eight studies—that were either cross-sectional or case-control design. By pooling the data from these studies, meta-analyses increased the sample size and the precision of the estimates, as well as reduced the effects of random error and bias. Thus, it provides a high level of evidence for the effectiveness of an intervention or the association of an exposure and an outcome.

Our pooled analyses are able to reconcile some of the inconsistencies the systematic review by Kavadya et al.⁷ pointed out: for instance, some studies showed a positive association between smoking status and occurrence of AGA, while others did not. Meta-analyses are advantageous because the pooled analysis will always be far more statistically powered than the analyses in the constituent studies. So, the constituent studies that failed to show an association may have been underpowered.

We found that men who ever smoke have higher odds of developing AGA, compared to their counterparts who never smoke. Our results showed that the likelihood of developing pattern hair loss is greater in men who smoke at least 10 cigarettes per day compared to their counterparts who smoke up to 10 cigarettes daily. Our analyses showed that, across men who have pattern hair loss, smokers have a significantly higher odds of disease progression compared to their counterparts who never smoke. Though our pooled analysis found no significant association between smoking intensity and severity of male AGA, a positive correlation may exist and be detected through an analysis that is statistically more powered (i.e., have a lower Type II (β) error rate).

We found that men who have ever smoked are 1.8 times more likely ($p < 0.05$) to experience male pattern baldness (AGA) compared to individuals who have never smoked. Men who smoke 10 cigarettes per day are two times more likely ($p < 0.05$) to develop AGA than those who smoke up to 10 cigarettes per day. Additionally, the chances of male pattern baldness worsening are 1.3 times more likely ($p < 0.05$) compared to males who have never smoked. Our patients with male pattern baldness need to be educated about the negative effects of smoking, given the fact that this condition can have a profound negative psychological impact on those who suffer from it. Furthermore, there is only a limited number of nonsurgical treatments available to address male-pattern AGA, and some of these treatments may have possible adverse effects that could negatively affect patient compliance.²¹

AUTHOR CONTRIBUTIONS

Conception of the manuscript was done by MT and AKG. The work was drafted by MAB, and substantively revised by AKG and MT.

CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

Authors declare human ethics approval was not needed for this study.

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REFERENCES

- Piraccini BM, Alessandrini A. Androgenetic alopecia. *G Ital Dermatol Venereol*. 2014;149(1):15-24.
- Kligman AM, Freeman B. History of baldness. From magic to medicine. *Clin Dermatol*. 1988;6(4):83-88.
- Cash TF. The psychosocial consequences of androgenetic alopecia: a review of the research literature. *Br J Dermatol*. 1999;141(3):398-405.
- Yi SM, Son SW, Lee KG, et al. Gender-specific association of androgenetic alopecia with metabolic syndrome in a middle-aged Korean population. *Br J Dermatol*. 2012;167(2):306-313.
- Trüeb RM. Association between smoking and hair loss: another opportunity for health education against smoking? *Dermatology*. 2003;206(3):189-191.
- Babadjouni A, Pouldar Foulad D, Hedayati B, Evron E, Mesinkovska N. The effects of smoking on hair health: a systematic review. *Skin Appendage Disord*. 2021;7(4):251-264.
- Kavadya Y, Mysore V. Role of smoking in androgenetic alopecia: a systematic review. *Int J Trichology*. 2022;14(2):41-48.
- Salem AS, Ibrahim HS, Abdelaziz HH, Elsaie ML. Implications of cigarette smoking on early-onset androgenetic alopecia: a cross-sectional study. *J Cosmet Dermatol*. 2021;20(4):1318-1324.
- Severi G, Sinclair R, Hopper JL, et al. Androgenetic alopecia in men aged 40-69 years: prevalence and risk factors. *Br J Dermatol*. 2003;149(6):1207-1213.
- Le Foll B, Piper ME, Fowler CD, et al. Tobacco and nicotine use. *Nat Rev Dis Primers*. 2022;8(1):19.
- Clark J, Glasziou P, Del Mar C, Bannach-Brown A, Stehlik P, Scott AM. A full systematic review was completed in 2 weeks using automation tools: a case study. *J Clin Epidemiol*. 2020;121:81-90.
- Ouzzani M, Hammady H, Fedorowicz Z, Elmagarmid A. Rayyan—a web and mobile app for systematic reviews. *Syst Rev*. 2016;5(1):210.
- Posit Team. RStudio: Integrated Development Environment for R 2022.
- Danesh-Shakiba M, Poorolajal J, Alirezaei P. Androgenetic alopecia: relationship to anthropometric indices, blood pressure and lifestyle habits. *Clin Cosmet Investig Dermatol*. 2020;13:137-143.
- Su LH, Chen TH. Association of androgenetic alopecia with smoking and its prevalence among Asian men: a community-based survey. *Arch Dermatol*. 2007;143(11):1401-1406.
- Arias-Santiago S, Gutiérrez-Salmerón MT, Castellote-Caballero L, Buendía-Eisman A, Naranjo-Sintes R. Androgenetic alopecia and cardiovascular risk factors in men and women: a comparative study. *J Am Acad Dermatol*. 2010;63(3):420-429.
- Lai CH, Chu NF, Chang CW, et al. Androgenic alopecia is associated with less dietary soy, lower [corrected] blood vanadium and

- rs1160312 1 polymorphism in Taiwanese communities. *PLoS ONE*. 2013;8(12):e79789.
18. Vora RV, Kota R, Singhal RR, Anjaneyan G. Clinical profile of androgenic alopecia and its association with cardiovascular risk factors. *Indian J Dermatol*. 2019;64(1):19-22.
19. Fortes C, Mastroeni S, Mannooranparampil T, Abeni D, Panebianco A. Mediterranean diet: fresh herbs and fresh vegetables decrease the risk of androgenetic alopecia in males. *Arch Dermatol Res*. 2018;310(1):71-76.
20. World Medical Association. World medical association declaration of helsinki: ethical principles for medical research involving human subjects. *JAMA*. 2013;310(20):2191-2194.
21. Gupta AK, Venkataraman M, Talukder M, Bamimore M. Finasteride for hair loss: a review. *J Dermatolog Treat*. 2022;33(4):1938-1946.

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