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Review Article

Smoking and the risk of type 2 diabetes in Japan: A systematic review and meta-analysis

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ABSTRACT

Cigarette smoking is the leading avoidable cause of disease burden. Observational studies have suggested an association between smoking and risk of type 2 diabetes mellitus (T2DM). We conducted a meta-analysis of prospective observational studies to investigate the association of smoking status, smoking intensity, and smoking cessation with the risk of T2DM in Japan, where the prevalence of smoking has been decreasing but remains high. We systematically searched MEDLINE and the Ichushi database to December 2015 and identified 22 eligible articles, representing 343,573 subjects and 16,383 patients with T2DM. We estimated pooled relative risks (RRs) using a random-effects model and conducted subgroup analyses by participant and study characteristics. Compared with nonsmoking, the pooled RR of T2DM was 1.38 (95% confidence interval [CI], 1.28–1.49) for current smoking (19 studies) and 1.19 (95% CI, 1.09–1.31) for former smoking (15 studies). These associations persisted in all subgroup and sensitivity analyses. We found a linear dose-response relationship between cigarette consumption and T2DM risk; the risk of T2DM increased by 16% for each increment of 10 cigarettes smoked per day. The risk of T2DM remained high among those who quit during the preceding 5 years but decreased steadily with increasing duration of cessation, reaching a risk level comparable to that of never smokers after 10 years of smoking cessation. We estimated that 18.8% of T2DM cases in men and 5.4% of T2DM cases in women were attributable to smoking. The present findings suggest that cigarette smoking is associated with an increased risk of T2DM, so tobacco control programs to reduce smoking could have a substantial effect to decrease the burden of T2DM in Japan.

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Introduction

The United States Surgeon General's report recently documented a 40% increase in the risk of type 2 diabetes mellitus (T2DM) among cigarette smokers compared with nonsmokers, based on a systematic review and meta-analysis of 46 prospective studies, and concluded that cigarette smoking is a cause of T2DM.¹ This conclusion has been supported by a more recent and vigorous systematic review and meta-analysis of 88 prospective studies.² Although it remains debatable whether a causal relationship between smoking and T2DM has been established,^{2–4} eliminating smoking may considerably reduce the burden of T2DM. For example, Pan et al estimated that 11.7% of diabetes cases among

men and 2.4% of diabetes cases among women would be attributable to active smoking if smoking is causally related to diabetes.² Because there are substantial differences in the prevalence of smoking among countries,⁵ the burden of diabetes that is attributable to smoking likely varies across countries. Quantification of the country-specific burden of diabetes associated with smoking would help guide country-specific evidence-based policies.

In Japan, the prevalence of diabetes has been steadily increasing and is expected to increase 10% by 2030.⁶ Obesity is not common in Japan,⁷ so preventative strategies that target weight loss may not be as effective in Japan as in Western populations.⁸ Given the high prevalence of smoking, especially among young men (approximately 32%),⁷ tobacco control may have a substantial importance in managing diabetes in Japan. However, there has been no systematic evaluation of the association between smoking and the risk or burden of diabetes in Japan. Recent systematic reviews^{1,2} of worldwide studies did not include two Japanese studies.^{9,10} Furthermore, increasing evidence from epidemiological studies also suggests that

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passive smoking is associated with an increased risk of diabetes.^{2,11–13} Therefore, the present study was performed to provide 1) a quantitative summary of the association between smoking status (current smoking, former smoking, smoking cessation years, and passive smoking) and the risk of T2DM in Japan and 2) the population attributable fraction (PAF) of diabetes due to smoking in Japan.

Methods

Search strategy

We conducted a systematic search of MEDLINE for the literature published through December 2015 of studies addressing the association between tobacco smoking and T2DM. The Ichushi (Japan Centra Revuo Medicina) database was also searched to identify studies written in Japanese. We used the following texts and keywords in combination with both MeSH terms and text words: diabetes mellitus, type 2 or diabetes mellitus, prediabetic state, smoking, smoking cessation, passive smoking, tobacco, smokeless tobacco use, cigarette, incidence, cohort studies, follow-up studies, survival analysis, Japan, and Japanese. We also searched the reference lists of publications included in the meta-analysis and relevant reviews.

Selection criteria and data extraction

We identified articles eligible for further review by performing an initial screen of identified abstracts or titles. The second screening was based on the full-text review. Two investigators (SA and AG) independently assessed the full text for eligibility; discrepancies were resolved via consensus or determined by a third investigator (TM). Only prospective cohort studies of Japanese populations living in Japan were included. We also considered studies for inclusion if the investigators reported data from an original study and the study was conducted among adults without T2DM at baseline. Exclusion criteria were studies that included participants with a specific disease. In case of multiple publications related to the same study, we included the reports with the longest follow-up or the largest number of incident cases of T2DM.

From full-text articles, we extracted data on the year of publication, study design, number of participants, exposures, the time of the exposures assessment, outcomes, confounders, and the measures of association. The main exposure variable of interest was the presence or absence of tobacco smoking at baseline. The preferred reference group was never smokers. The majority of studies defined a group of former smokers, but a few studies defined smokers and nonsmokers without mentioning whether former smokers were included in the nonsmoking group.

The outcome variable of interest was T2DM. The definitions and diagnostic criteria to define T2DM varied somewhat across studies. The criteria used to define T2DM have changed over time, as is evident by comparing the World Health Organization 1985 criteria¹⁴ (fasting plasma glucose [FPG] ≥ 140 mg/dL) with the World Health Organization 1999 criteria¹⁵ or the American Diabetes Association 1997 criteria¹⁶ (FPG ≥ 126 mg/dL). Some recent studies published after 2010 also used hemoglobin A1c (HbA1c) in defining T2DM based on American Diabetes Association criteria¹⁷ (FPG ≥ 126 mg/dL or HbA1c $\geq 6.5\%$). The diagnosis of diabetes was based on objective measurement (blood tests) except for in one study, which solely based diagnosis on self-reporting by patients. We included information available from publications, but when we did not obtain sufficient information about the outcome, exposure, and study design from the article, we communicated with the authors of the original reports for further details.

Quality assessment of the included studies

Using the Newcastle-Ottawa Scale,¹⁸ we assessed the overall quality of each study by totaling scores of the 9 criteria (0–9 stars): the representativeness of the exposed cohort, the selection of the nonexposed cohort, ascertainment of exposure, and outcome of interest not present at the start of the study (maximum of 4 stars); comparability of the cohorts on the basis of study design and analysis (maximum of 2 stars); and finally, the assessment of the outcome (maximum of 3 stars). Studies with scores of ≥ 6 , 4–5, and 0–3 were defined as a high, moderate, and low quality studies, respectively.

Statistical analysis

Relative risks (RRs) were used as the common measure of association across studies. Hazard ratios and incidence density ratios were directly considered as RRs, and odds ratios were regarded as approximate to RRs in view of the low incidence rates. Pooled risk estimates were performed according to the type of smoking. We used DerSimonian and Laird random-effects models for calculating the summary estimates.¹⁹ We used funnel plots and Egger's regression asymmetry test to assess publication bias.²⁰ Additionally, we performed trim-and-fill procedures to further evaluate possible effects of publication bias.²¹ We also conducted subgroup analyses according to follow-up years (≤ 10 vs. > 10 years), sample size ($\leq 20,000$ vs. $> 20,000$), number of confounding factors (≤ 8 vs. > 8 factors), mean age (≤ 50 vs. > 50 years), and diagnostic criteria of diabetes (FPG ≥ 126 mg/dL only vs. FPG ≥ 126 mg/dL or HbA1c ≥ 6.5). We assessed the difference in association between groups using meta-regression analysis. We undertook sensitivity analyses by excluding studies in which former smokers were included in the nonsmoker group.

In assessing dose-response relationships, we treated the number of cigarettes smoked per day as the explanatory variable. Because most studies reported cigarette consumption as categorical

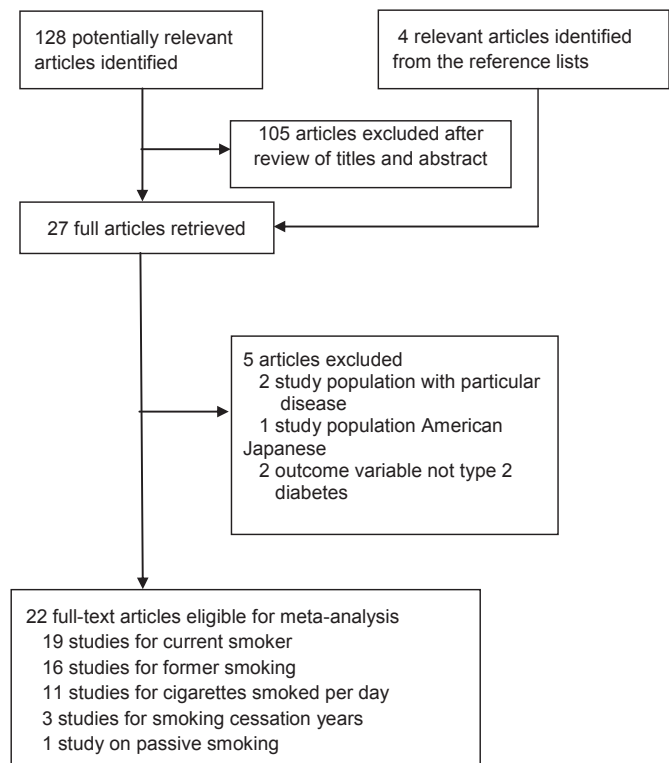


Fig. 1. Flowchart of the selection of studies included in meta-analysis.

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