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A SYSTEMATIC REVIEW OF THE RELATIONSHIP BETWEEN QUITTING SMOKING AND THE RISK OF TYPE 2 DIABETES

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Abstract:

Background: *Tobacco misuse has not been universally acknowledged as a modifiable risk factor in diabetes preventive or screening methods, despite mounting data revealing substantial epidemiological and mechanistic connections between smoking, hyperglycemia, and the development of type 2 diabetes.*

Purpose: *A growing number of research on the effect of smoking cessation on the development of type 2 diabetes mellitus. The goal of this systematic review was to search the available evidence on the impact of smoking cessation and risk for type 2 diabetes mellitus.*

Methods: *We searched PubMed, Embase, and Scopus (January 1995--October 2022) for prospective observational studies that evaluated the effects smoking and smoking cessation as risk factors for type 2 diabetes mellitus.*

Results: *Type 2 diabetes risk was increased by a factor of 1.37 (95% CI: 1.33 to 1.42) for current smokers, 1.14 (1.10 to 1.18) for ex-smokers, and 1.22 (1.10 to 1.35) for passive smokers, compared to those who had never smoked (7 studies). The associations persisted across all subgroups, and a dose-response relation was discovered between current smoking and diabetes risk, with the RRs (95% CIs) for light, moderate, and heavy smokers, compared with never smokers, being 1.21 (1.10-1.33), 1.34 (1.27-1.41), and 1.57 (1.47-1.66), respectively. If there is a causal relationship between smoking and diabetes, we calculated that 10% in men and 2% in women of all instances of type 2 diabetes globally (about 25 million) may be attributed to smoking. The pooled RR (95% CI) from 10 studies showed that recent ex-smokers (within 5 years) had a lower risk of relapse than never smokers, while long-term ex-smokers (within 10 years) had a lower risk of relapse than current smokers.*

Conclusion: *Both active and passive smoking are strongly connected with higher risks of type 2 diabetes. The risk of diabetes is higher among recent ex-smokers, although it lowers significantly with time.*

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INTRODUCTION:

Cigarette smoking, despite international efforts to curb the epidemic, is still responsible for nearly 6 million deaths annually [1]. Smoking is widely recognized as a contributor to the development of cancer, respiratory diseases, and cardiovascular disease [2], but the association between smoking and the development of diabetes is less well-known. Twenty-five prospective studies published between 1980 and 2007 were pooled in a previous meta-analysis [3], which summarized their findings on the correlation between cigarette smoking and the development of diabetes. For the first time, a section on smoking and diabetes risk was included in the 2014 US Surgeon General's Report, along with an argument for the causal relation between the two [2]. However, the report did not address the connection between passive smoking and diabetes risk or the benefits of quitting smoking.

Nearly 422 million people around the world are living with diabetes mellitus, making it the direct cause of 1.5 million deaths and an indirect cause of an additional 17.5 million deaths annually [4]. The vast majority of people who develop diabetes have type 2 diabetes (T2D), which is characterized by insulin resistance and the gradual decline in cell function and mass over time. Targeting known risk factors through early lifestyle modification is still the most effective strategy for reducing T2D prevalence and mortality [4], as this disease is strongly linked to environmental, nutritional, and lifestyle determinants. According to the Surgeon General's Report in 2014, tobacco use raises the risk of type 2 diabetes by 30–40% among current smokers compared to nonsmokers, suggesting that encouraging people to quit smoking is a crucial public health strategy in the fight against the global diabetes epidemic [5]. Smoking is a preventable risk factor for T2D, and the World Health Organization recommends that people avoid or quit smoking as part of a healthy lifestyle [6]. Smoking is not currently recognized as a modifiable risk factor for the development of diabetes or as a factor that should prompt diabetes screening by the American Diabetes Association or the International Diabetes Foundation [7, 8].

Additionally, there is mounting evidence from epidemiological studies linking passive smoking to an increased risk of type 2 diabetes [9-11]. Concern about increased diabetes risk with smoking cessation has also been raised by a number of studies [12-21], which found that the risk of diabetes was higher in people who had recently stopped smoking. As a result, direct evidence for clinical practice and public health policy can be gained by quantifying the association between time since smoking cessation and diabetes risk. The current review aimed to summarize the available evidence on the effect of smoking cessation as a risk for type 2 diabetes mellitus (T2DM).

METHODOLOGY:

Review Question

This review seeks to summarize the available evidence on smoking cessation as a risk for T2DM. The specific review questions to be addressed are:

(1) What is the effect of smoking cessation on the development of T2DM?

(2) Is smoking cessation a risk factor for T2DM?

Study design

This is a comprehensive systematic review of the literature about smoking cessation as a risk for T2DM. This study relied on reviewing the literature using systematic approach. The literature search, study selection based on title and abstract, and data extraction was carried out according to the study objectives and eligibility criteria as demonstrated later in this section. In case of duplicate articles, only one was considered.

Search strategy

The studies were identified through research in the PubMed, Embase, Scopus, Web of Science and google scholar databases, performed in January 2023. Grey

literature was also searched. Search was done and selecting only articled published in English language. Moreover, selected articles were chosen from peer-reviewed journals. In addition, the bibliographies of any qualified articles recognized were checked for extra literature and reference search was done for all included references utilizing ISI Web of Science. The selected period during the search was (1995 – present). Published articles were considered to be compositions that showed up in peer-reviewed journals. Articles present in grey literature were excluded from our review unless they meet eligibility criteria and free of bias. A grey literature search was also conducted by searching libraries and websites of smoking and T2DM reports.

Search Keywords

For each database searched, all keywords were used during the searching process and literature identification. Keywords used for the searching process were (smoking OR cessation OR risk OR Type 2 diabetes mellitus OR systematic review). The search was done by using "AND" and "OR". In some instances, one keyword was used and other instances two or more keywords were used. All searching process was performed during the searching period (1995 – present).

Eligibility criteria

The inclusion criteria are as follows: studies published in English only, published on the date of searching period (1995 – present), evaluating latest updates on smoking cessation as a risk for T2DM.

Selection of the studies

For titles and abstracts that are potentially suitable for the review and met the selection criteria, full text was obtained for further evaluation. Full-text was assessed against the list of eligibility criteria for inclusion. A process of discussion with supervisors resolved any uncertainty of selection. However, in some cases, a third reviewer was consulted.

Outcomes

Primary outcome

To summarize the available evidence on smoking cessation as a risk factor for T2DM.

Secondary outcome

None.

Data extraction and management

After assessing the title, abstract, and full text of the studies according to the eligibility criteria, the data of interest was collected using a standard form. The following information was collected:

- Authors,
- Date,
- Setting,
- Smoking status,
- Diagnosis of T2DM,
- Implication of the study

Information extraction, (choice and coding)

Information was extracted from the included articles utilizing an electronic information extraction structure on Microsoft Access programming. Two reviewers freely extracted information, utilizing a standard information extraction structure which was created by the survey creators with the end goal of the review. The extraction structure incorporated the accompanying data:

1- Publication subtleties: title, authors, journal name, year and place of study, of distribution, country in which the review was led, sort of distribution, and wellspring of financing.
2- Study subtleties: concentrate on plan (cross-sectional, cohort, case-control), settings (clinical or population based), concentrate on transience (planned or review), patients' enlistment techniques (successive or non-continuous), the geographical area, year of information assortment and reaction rate, qualification (consideration and avoidance rules), name of appraisal tool(s), approval of evaluation tool(s).
3- Study members' subtleties: number of people reviewed/examined, population qualities including mean age (SD), and gender distribution, relationship status, demographic data.

Statistical Analysis

All extracted data was coded and entered to Statistical Package of Social Sciences (SPSS) and frequency analysis was performed to determine themes of included data. Custom tabs were done to differentiate between similar data included among selected articles. A descriptive statistics was employed and relevant data was extracted from eligible studies and presented in tables. Then, a narrative synthesis was provided of the summary of the relevant data and information from the selected articles. Forest plots were drawn for suitable variables.

RESULTS:

A total of 12,598 studies were identified in the search, all of them were assessed for eligibility, and 18 articles were included in this review (Figure 1).

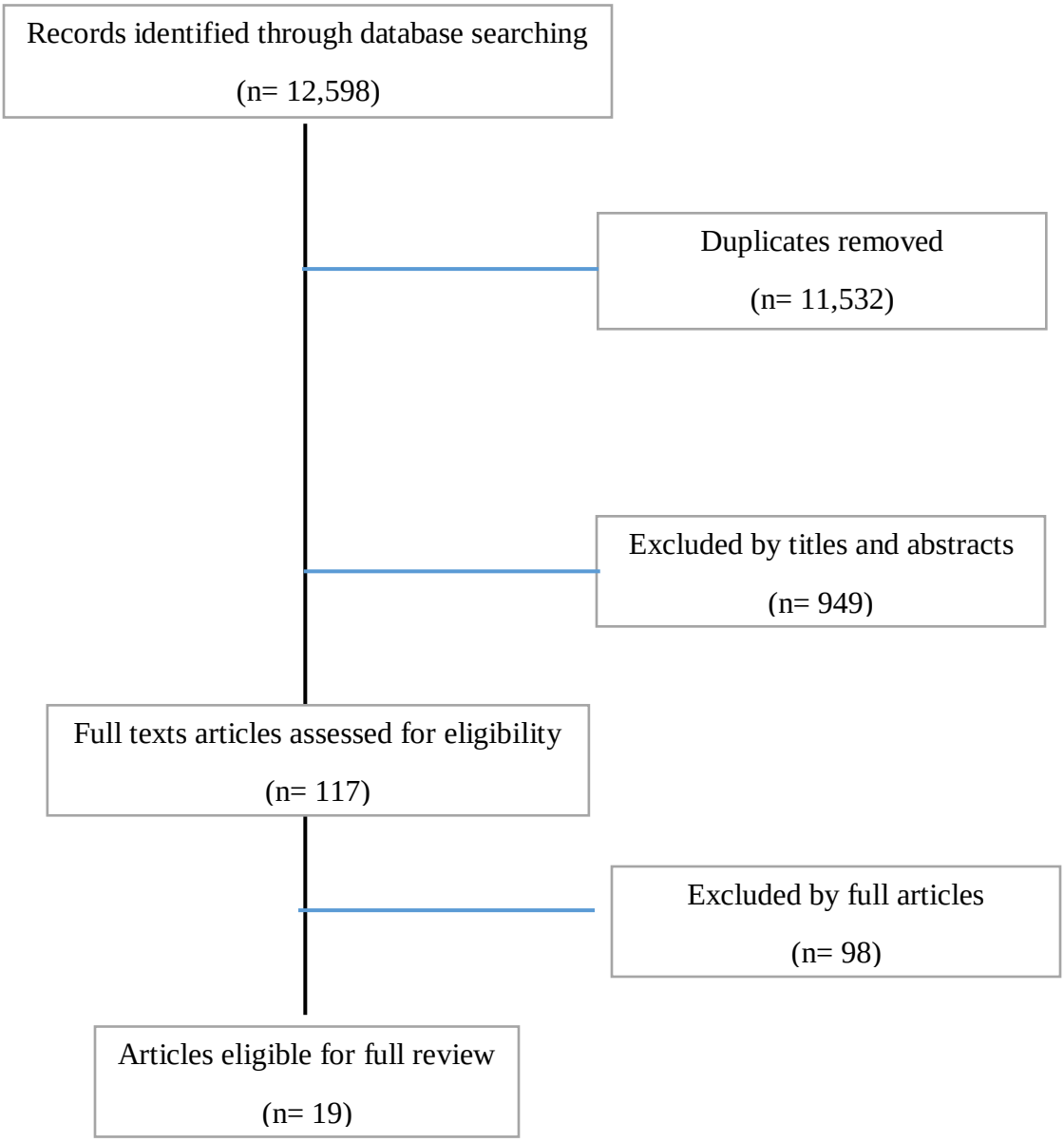

Figure 1: Study-flow diagram showing the number of studies screened, assessed for eligibility and included in the review

Some studies [22-25] employed less rigorous criteria for diabetes and included patients with impaired fasting glucose (61 mmol/L or 110 mg/dL), however all studies reported outcomes for type 2 diabetes. Except for the research by Houston et al [22], which confirmed self-reported smoking status against blood cotinine level, all investigations relied on self-reported smoking status. There were a total of 5853952 people included in the study, with 294081 developing diabetes as an occurrence. Of the 84 research, the most majority were carried out in European nations (n=32), the United States (n=19), and Japan (n=17), with the remaining studies being undertaken in Korea (n=5), mainland China and Taiwan (n=4), Australia (n=3), Israel (n=2), Turkey (n=1), and Iran (n=1). There were mixed-gender samples in most research, but there

were also 27 male-only studies and 5 female-only studies. The median number of years of follow-up was 9 (IQR = 5.9–12.9). Contradictory results were found for the prevalence of current smoking across different research. There were 96 comparisons based on 84 research, 12 of which presented their findings separately by sex.

From the random-effects model, the relative risk (RR) (95% CI) between current smokers and never-smokers was 137 (133-142), while between the two groups, the RR was 129 (128-132) from the fixed-effects model (data not shown). The findings were the same whether comparing current smokers to nonsmokers or former smokers to never smokers (RR 1.40; 95% CI 1.33-1.47; 1.35; 1.27-1.42; 1.35; 116-1.57). Compared to never smokers, the pooled RRs for light, moderate, and heavy smokers were 1.21 (95% CI 1.10-1.33), 1.34 (95% CI 1.27-1.41), and 1.57 (95% CI 1.47-1.66), respectively. For both current smoking status and cumulative smoking exposures, similar dose-response patterns were seen whether measured in cigarettes smoked per day or pack-years (Figure 2).

There was a significant amount of heterogeneity found (I2=68.6%; p.001), therefore the RR should be seen as a median estimate of the many correlations between cigarette smoking and the incidence of diabetes. Consequently, the range of probabilities within which we are 95% certain is 1-in-110. In order to dig further into the diversity, we used stratified analysis, and we discovered that the elevated risk was present across all strata, even though the diversity was still rather high. When cigarette smoking was the primary risk factor and diabetes was diagnosed by monitoring of blood glucose levels, a higher correlation was seen in men than in women. All meta-regression p-values were over.010, however, indicating that there were no statistically significant differences across the various strata.

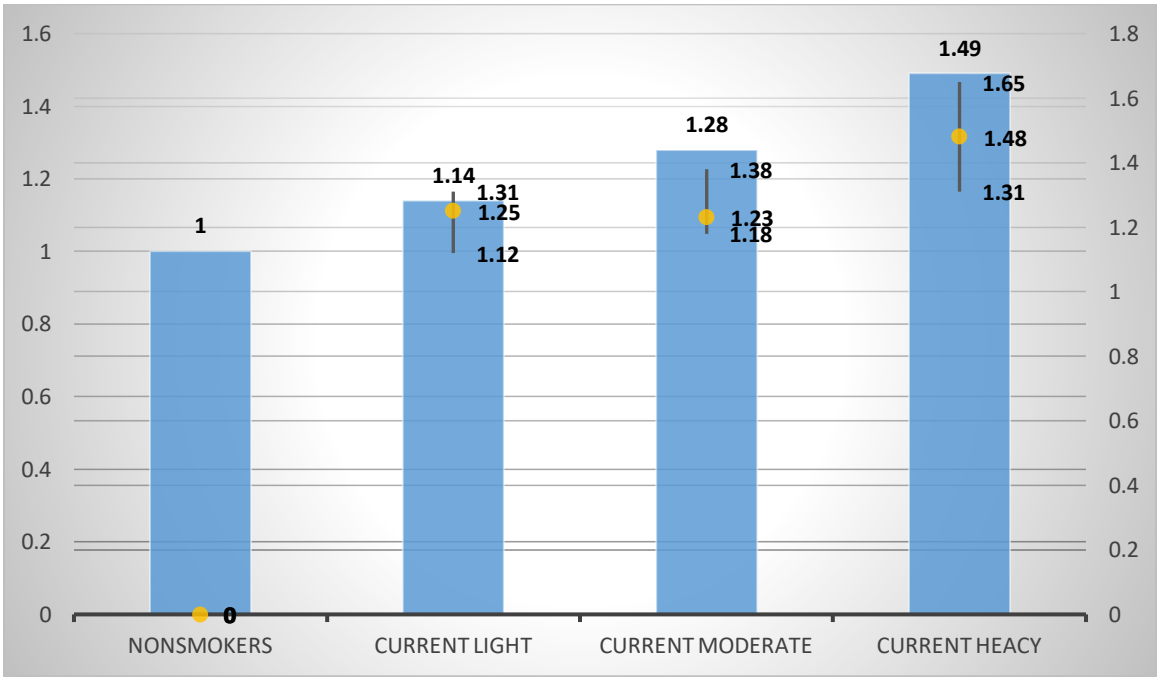

Figure 2: Relation of Smoking Intensity with Risk of Incident Type 2 Diabetes

Only three (17), twenty-two (26), and four (27, 30) of the seven studies demonstrating a link between passive smoking and diabetes risk were included in the active smoking and diabetes analysis. Two studies [17, 30] included only women, whereas five [22, 26-29] included both sexes. There were 156439 total participants and 7843 incident incidences of diabetes. There were three studies conducted on people in the United States [17, 22, 28], two on people in Europe [27, 30], and two on people in Asia [26,29]. The pooled RR for type 2 diabetes in passive smokers was 1.22% (95% CI 1.010 to 1.35), with minimal heterogeneity (I2=32.6 %; p=0.18) compared to never smokers with no passive smoke exposure.

Ten research on ex-smokers provided extensive data on the number of years after stopping. The United States had five studies (12,14,15,17,20), Europe had two (13, 21), and Asia had three (15,18,19). (Korean and Japanese). Only males were included in four

studies (12,13,15,19), women in two research (17,20), and both sexes in four investigations (14,16,19,21). (while three [14,19,21] of them reported results separately for men and women). There was a pooled RR (95% CI) of 154 (136-174) in recent ex-smokers (5 years), 1118 (107-129) in intermediate ex-smokers (5-9 years), and 111 (102-120) in long-term ex-smokers (10 years) compared to never smokers (Figure 3). When comparing recent ex-smokers with never-smokers, the RRs were consistently higher in all 10 investigations, demonstrating that the risk of developing diabetes remained elevated for at least some time after stopping smoking. Among new quitters, the risk was highest among Asians (RR 204; 95% CI 150-277) and lowest among Caucasians (RR 143; 95% CI 126-162; p value from meta-regression = 0045). The combined RR for the 10 trials comparing ever smokers to those who had never smoked was 1.47 (95% CI 1.34-1.62).

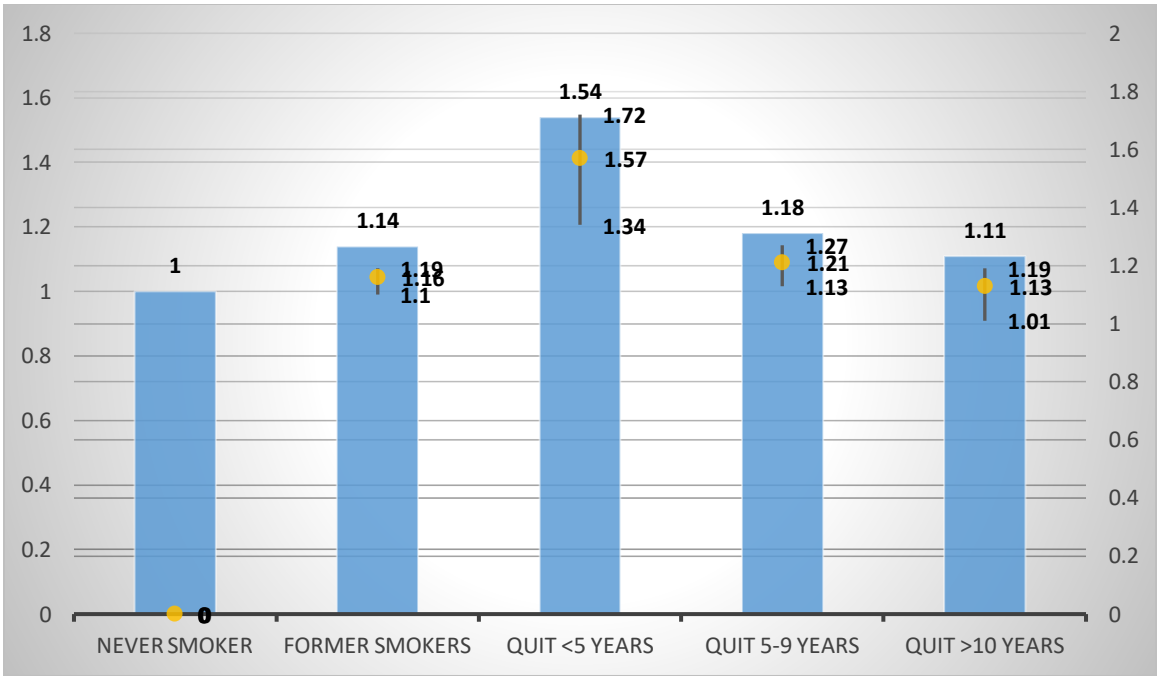

Figure 3: Relation of Smoking Cessation with Risk of Incident Type 2 Diabetes

DISCUSSION:

According to the results of the present systematic review, both active and passive smoking were related with a higher risk of incident type 2 diabetes (RR=1.37 and 1.22) among the general population. In addition, the relationships remained across all subgroups stratified by different research and participant's characteristics. Furthermore, while former smokers still had a greater risk compared to never smokers, the elevated risk (RR=1·14) was substantially lower compared to that in present smokers (RR=1·37), confirming the advantages of smoking cessation to decrease diabetes risk. A comprehensive examination of years following quitting smoking also demonstrated that smoking cessation greatly cut diabetes risk in the long-term despite heightened risk in the new quitters.

Two earlier meta-analyses of smoking and diabetes risk [2, 3] are consistent with our findings. The current US Surgeon General's Report comprised 46 studies published before 2010, and generated a pooled RR of 1·37 (95% CI 1·31–1·44) [2]. Many further research have been published since then. Our latest meta-analysis expanded earlier results and included a total of 84 research on active smoking, thus offering the most precise and complete estimations of smoking and diabetes risk. The research by Willi et al. [3] revealed that the correlation differed considerably in certain strata, including baseline age, BMI, and follow-up years. Nonetheless, there were just 25 papers included in that meta-analysis, thus the results were not very conclusive. Our present meta-analysis did not indicate large variations between study and participant's characteristics; nonetheless, significant heterogeneity persisted in most subgroups.

Most prior research have focused on active smoking and risk of type 2 diabetes, and mounting data has shown that secondhand smoking might potentially be an issue. While many recent meta-analyses [9-11] have provided a summary of the available evidence on this issue, they have all included just a small sample of cohort studies, with one of these studies even being a cross-sectional study [9]. A recent investigation [31] found that over 30% of worldwide non-smokers were exposed to secondhand smoking and it contributed to roughly 1% of global total mortality and 0·7% of disease burden. It should be noted, however, that diabetes was left out of the aforementioned illness burden study. Taken together, our results further underline the necessity of implementation and enforcement of smoke-free laws to limit the number of persons exposed to secondhand smoking.

Similar to earlier studies that have suggested a causative association between smoking and several disease outcomes (cancer, CVD, and death) [2], our data have indicated that smoking is likely to be a causal risk factor for diabetes. In particular, our meta-analysis offers more evidence of dose-response connection (by both present smoking intensity and cumulative smoking exposure), and more crucially, the large advantages connected to smoking cessation in the long run. Our pooled estimates revealed that new ex-smokers were at a higher risk than never-smokers (RR=154), but that this elevated risk greatly diminished after 5 years to roughly 118 and to 111 after 10 years. This is the first meta-analysis to our knowledge to examine the link between quitting smoking and reduced risk of diabetes. Smokers tend to have a lower body mass index (BMI) than nonsmokers, however quitting smoking often results in weight increase [32]. For motivated smokers, the higher risk of developing diabetes in the near term may be explained by the fact that quitting smoking is linked with a mean rise in body weight of 4-5 kg after 12 months of abstinence [33]. The elevated risk persisted even after accounting for baseline BMI in the models, and in some studies [15, 18, 19] after further accounting for weight growth or using BMI as a time-varying covariate. The higher risk was previously documented by Oba et al. [19], who similarly stratified the study by weight gain status, but only for male new quitters who gained 3 kg weight over the first 5 years of quitting. After accounting for weight growth, the link weakened marginally in a few of trials [12,16]. Therefore, the short-term diabetes risk linked with smoking cessation is not exclusively driven by weight increase. However, the results are not entirely consistent [37-39], and a recent large retrospective cohort study in the UK primary care database even found a deterioration effect in glycemic control that lasted for 3 years after smoking cessation, independent of weight gain in type 2 diabetic patients [40].

People who attempt to stop smoking are more likely to have pre-clinical problems or high cumulative smoking exposures, which may explain the short-term higher risk among recent quitters. There was a lack of information in the studies on why people stopped smoking, so we couldn't determine whether ex-smokers were at an increased risk for diabetes. Several smoking measures, including as age at beginning, number of smoking years, and quantity of cigarettes smoked per day at baseline, were not significantly different between new quitters and continuing smokers [16, 18, 20]. It's also doubtful that the findings were largely influenced by the naturally elevated risk of diabetes among recent ex-smokers, given all the studies have rigorously adjusted for baseline lifestyle characteristics and health status. Last but not least, a number of studies [16, 19, 20] found that the elevated risk among recent ex-smokers was limited to long-term heavy smokers but not light smokers. Therefore, the cumulative smoking exposures before to quitting may determine the higher risk among recent quitters. It is yet unclear what processes underlie the correlation between quitting smoking and a temporary increase in diabetes risk. Smoking cessation, on the other hand, has been shown to minimize the risks of cardiovascular disease [41,42], mortality [43,44], and diabetes [41,42], both in the short and long term. The increased risk of diabetes in the short term would therefore be outweighed by the long-term advantages of giving up smoking on diabetes and other health outcomes.

Despite significant declines in smoking prevalence over the last several decades [45], tobacco use continues to pose a serious hazard to public health worldwide [1]. We calculated that if smoking is a causative risk factor for diabetes, then 2.3 percent of female cases and 10.3 percent of male cases occur due to current smoking. Multiple factors suggest that these estimates may be too low. As a starting point, we relied on the daily smoking prevalence [45], but if we had instead utilized the current smoking prevalence from the WHO database, the PAFs would have been much higher [46]. Second, we did not account for exposure to passive smoking; if people stopped smoking, passive smoking would go down, too, which would have a positive effect on diabetes risk. However, since the risk among current smokers does not revert to 1

with long-term quitting, the PAF may be an overestimate. Our findings are consistent with those from the most recent WHO report [1], which found that smoking was responsible for 16% of male deaths and 7% of female deaths worldwide, and with those from the Global Burden of Disease Study 2010, which found that tobacco use (including passive smoking) was responsible for 8% of male disease burden and 3% of female disease burden [47].

CONCLUSION:

In conclusion, this systematic review adds to the growing body of data showing that cigarette smoking, both active and passive, is a major modifiable risk factor for developing type 2 diabetes. Short-term risk of diabetes remains high even when smoking is stopped, while long-term risk drops dramatically. There is a clear correlation between the rising number of diabetics and the widespread use of tobacco products; as a result, limiting tobacco use should be a top public health priority in an effort to stem the global diabetes pandemic. More action has to be done to implement and enforce the rules of the WHO Framework Convention on Tobacco Control to decrease smoking rates among existing smokers. In addition to helping safeguard nonsmokers, smoke-free laws may boost the number of smokers who are able to successfully quit the habit. Finally, further research is needed into the processes behind the temporarily elevated risk of diabetes in the recent ex-smoker, so that effective pharmacological and lifestyle therapies may be developed to raise the proportion of smokers who are able to stop.

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