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Exploring the Association Between Smoking and Risk of Type 2 Diabetes

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Abstract. Type 2 diabetes mellitus is a common chronic metabolic disorder with increasing global prevalence, and smoking is an important modifiable risk factor. This review focuses on the association between tobacco exposure, including active smoking, secondhand smoke, and electronic cigarette use, and type 2 diabetes. Multiple studies indicate that tobacco exposure is significantly associated with a higher diabetes risk, with a clear dose-response relationship. Worse metabolic indicators, including blood glucose and insulin resistance, further confirm these harmful effects through insulin resistance, inflammation, and β-cell dysfunction. Studies also show statistically significant benefits of smoking cessation, with longer cessation related to lower diabetes risk. In conclusion, tobacco exposure is strongly linked to type 2 diabetes in a dose-dependent manner. Reducing tobacco exposure and smoking cessation are effective to lower diabetes risk, providing evidence for clinical and public health prevention.

Keywords: Smoking, E-cigarettes, Type 2 diabetes, Mellitus, Risk assessment

1. Introduction

Type 2 diabetes mellitus (T2DM) is a prevalent chronic metabolic disorder with rising incidence worldwide and an earlier onset age. It features insulin resistance, β-cell dysfunction and glycemic abnormalities, leading to severe complications and substantial health burdens. Tobacco exposure, including smoking, secondhand smoke and e-cigarettes, is a major modifiable risk factor for T2DM. It promotes inflammation, oxidative stress and impaired insulin signaling, which contribute to diabetes development. Epidemiological evidence shows a significant positive association and dose-response relationship between tobacco exposure and T2DM risk. Even passive smoking and e-cigarettes are not harmless and can increase diabetes risk.

Therefore, this review uses descriptive, correlation and dose-response analysis to examine the link between tobacco exposures and T2DM. It summarizes relevant hazards and provides evidence for clinical and public health prevention.

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2. Classification and harms of tobacco exposures

2.1. Active smoking: components and adverse health effects

Active smoking is the most common and extensively studied form of tobacco exposure around the world, which refers to the direct inhalation of toxic smoke produced by the burning of cigarettes, cigars, pipes and other traditional tobacco products. Tobacco smoke is an extremely complex chemical mixture, which contains more than 7,000 different chemical components, and at least 250 of them are confirmed to be toxic, harmful or carcinogenic to human body. The main harmful components include nicotine, tar, carbon monoxide, hydrogen cyanide, heavy metals, volatile organic compounds and polycyclic aromatic hydrocarbons. Nicotine, as the core addictive substance in tobacco, can quickly enter the human blood circulation through the respiratory tract, stimulate the sympathetic nervous system continuously, increase the heart rate and blood pressure, disturb the normal secretion and signal transmission of various metabolic hormones in the body, and directly interfere with the regulation process of blood glucose and blood lipid. Tar and a large number of fine particulate matters in tobacco smoke will induce long-term and sustained oxidative stress and chronic low-grade inflammation in the body, damage the structure and function of vascular endothelial cells, affect the blood supply and normal physiological activities of metabolic organs such as pancreas and liver. Long-term active smoking will gradually reduce the sensitivity of body tissues to insulin, promote the disorder of fat metabolism and abnormal fat accumulation, and directly damage the function of pancreatic β cells, resulting in the decrease of insulin synthesis and release. Over a long period of time, these continuous physiological injuries will lead to persistent insulin resistance and unstable blood glucose regulation, which lays a solid pathological foundation for the occurrence of prediabetes and further develops into clinical type 2 diabetes. In addition, long-term smoking will also damage the metabolic function of the liver, affect the absorption, transformation and storage of sugar in the body, aggravate the disorder of glucose metabolism, and make smokers more likely to suffer from various chronic metabolic diseases represented by type 2 diabetes. These metabolic disorders further reduce the utilization efficiency of glucose in peripheral tissues, aggravate insulin resistance, and increase the possibility of abnormal blood glucose regulation. Despite an elevated risk in recent quitters, a thorough examination of the years since stopping smoking also revealed that quitting was linked to a significant long-term reduction in the risk of diabetes [1]. Additionally, long-term smoking is also closely related to the occurrence of hypertension, hyperlipidemia and fatty liver, which together constitute a variety of metabolic risk factors and jointly promote the occurrence and development of type 2 diabetes. So it causes a lot of problems. The uncontrolled weight is one of them. According to multiple studies, smokers have a lower BMI and are often leaner than nonsmokers or former smokers. Nevertheless, there is evidence that smokers, particularly heavy smokers, tend to have higher BMIs. This finding may be caused by the weight cycling phenomena in addition to a clustering of dangerous activities. When they stop smoking, smokers typically gain weight; the more dependent they are, the more likely they are to relapse [1].

2.2. Secondhand smoke: characteristics and potential hazards

Secondhand smoke is a mixed smoke mixture formed by sidestream smoke released directly from the burning end of tobacco products and mainstream smoke exhaled actively by smokers. Every day, millions of non-smoking people are passively exposed to secondhand smoke in closed or semi-enclosed environments such as families, offices, restaurants, public transport and entertainment

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venues. Although the concentration of toxic components in secondhand smoke is relatively lower than that in the mainstream smoke directly inhaled by active smokers, it still contains almost all the same harmful chemicals as active smoking, including nicotine, carbon monoxide, irritating gases, fine suspended particles and carcinogenic components. Long-term, low-dose and repeated exposure to secondhand smoke will cause cumulative and irreversible damage to the human body. Key adverse effects include sustained systemic inflammatory response, increased oxidative stress level, obvious vascular endothelial dysfunction, adverse changes in blood lipid profiles and significant reduction in insulin sensitivity. According to existing evidence, SHS increases the risk of stroke, type 2 diabetes, and lung cancer by at least 8%, 5%, and 1%, respectively–relationships classified as two-star associations [2]. In contrast, the evidence of associations between SHS and otitis media, asthma, lower respiratory infections, breast cancer and COPD are weaker and these risk–outcome pairs were classified as one-star associations. Many people often ignore the real harm of secondhand smoke and regard it as a mild and negligible environmental interference factor, but a large number of medical studies have confirmed that long-term passive smoke exposure can stably interfere with the core metabolic pathways of the human body, destroy the stability of the internal environment, and gradually weaken the body's ability to maintain normal blood glucose levels. Therefore, secondhand smoke is not a harmless passive environmental exposure, but a hidden and long-lasting threat to public metabolic health, especially for non-smokers, the elderly, children and people with potential metabolic risks. Its mild but sustained negative effects on glucose homeostasis will quietly increase the risk of abnormal glucose metabolism and lay a hidden danger for the occurrence of type 2 diabetes. In particular, the elderly, children, pregnant women and people with poor basic physical fitness are more sensitive to the damage of secondhand smoke, and their metabolic function is more likely to be affected. Long-term low-dose and continuous passive exposure will lead to cumulative damage, and its adverse effects on glucose metabolism will gradually appear with the extension of exposure time, rather than being completely harmless in a short time.

2.3. E-cigarettes: composition and misconceptions of safety

Electronic cigarettes, as a kind of emerging tobacco alternative products, have been widely popularized and used among young people in recent years, and are mistakenly regarded by most of the public as safe, non-toxic and harmless healthy substitutes for traditional cigarettes. Although clinical and epidemiologic data on health outcomes are not yet available, it is hypothesized that using e-cigarettes instead of tobacco cigarettes may be associated with better short-term and long-term health outcomes for long-term smokers who are unable to completely quit [3]. Most electronic cigarettes realize the smoking experience by heating the electronic liquid containing nicotine to produce inhalable aerosol, and their main components include addictive nicotine, propylene glycol, glycerin, various synthetic flavors and a variety of chemical additives. The aerosol produced by electronic cigarettes contains a large number of nicotine, ultrafine particles, volatile organic compounds and potential inflammatory irritants, which can quickly enter the human blood circulation through the respiratory tract, induce mild but continuous inflammatory response and oxidative stress in the body, and damage normal cell metabolism. The nicotine contained in electronic cigarettes still has complete biological activity, which can interfere with the balance of endocrine hormones in the body, destroy the normal signal transduction pathway of insulin, and damage the activity and function of islet cells. Despite the potential benefits of e-cigarettes over conventional cigarettes, the research that is now available has numerous flaws. Discussing e-cigarettes as a single device is challenging due to differences in product engineering, components, and possible toxicities. Despite insufficient data, e-cigarettes may be helpful in helping people quit

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smoking. Although there is no proof that long-term use of e-cigarettes reduces risk, they might be a less dangerous source of nicotine than regular cigarettes [4]. Long-term use of electronic cigarettes will gradually reduce the body's insulin sensitivity, interfere with the normal regulation of blood glucose, and lead to obvious disorder of lipid metabolism. Contrary to the widespread cognitive bias of the public, electronic cigarettes are not completely safe and risk-free health products. Even if they do not produce tar and some highly toxic gases produced by tobacco combustion, their long-term use still poses a significant threat to human metabolic health and impairs the normal regulation function of blood glucose. Although the harm of electronic cigarettes may be slightly lighter than that of traditional active smoking, they are not completely harmless, and their negative effects on glucose metabolism cannot be ignored in the risk assessment of type 2 diabetes and public health prevention and education. In addition, different flavors, heating powers and use frequencies of electronic cigarettes will lead to differences in the release of harmful substances, and their long-term use may also affect intestinal flora structure, which is closely related to glucose and lipid metabolism. These potential hazards further indicate that electronic cigarettes cannot be used as safe substitutes, and their long-term metabolic impact needs more attention.

3. Statistical association between tobacco exposures and type 2 diabetes risk

3.1. Active Smoking and type 2 diabetes risk

A large number of population-based observational studies, cross-sectional studies and quantitative statistical analyses around the world have consistently and clearly confirmed that active smoking is significantly associated with an increased risk of type 2 diabetes, and this correlation shows strong statistical significance and high consistency in different regions, age groups, genders and physical constitutions. Numerous studies have revealed that smoking not only raises the risk of developing diabetes but also disrupts glucose homeostasis and exacerbates chronic diabetic complications [5]. Among microvascular complications, the onset and progression of diabetic nephropathy are strongly associated with smoking. For macrovascular complications, smoking is linked to a 2–3 fold higher incidence of coronary heart disease (CHD) and related mortality. Compared with non-smoking populations, people who smoke actively for a long time have significantly higher prevalence rates of insulin resistance, impaired fasting glucose, impaired glucose tolerance and clinically diagnosed type 2 diabetes. A large number of quantitative statistical results further confirm that active smoking is an independent and important risk factor affecting the occurrence of type 2 diabetes, and there is a clear, stable and reliable dose-response relationship between smoking exposure level and diabetes risk. Generally speaking, the more cigarettes smoked per day, the longer the continuous smoking years, and the higher the cumulative exposure load of tobacco, the greater the risk of abnormal glucose metabolism and clinical type 2 diabetes. Statistical comparative analysis of objective metabolic indicators also shows that long-term smokers have significantly higher levels of fasting blood glucose, postprandial blood glucose, glycated hemoglobin and insulin resistance index than non-smokers, and the differences between different groups have clear statistical significance. These stable, reproducible and reliable statistical results strongly prove that active smoking can directly and continuously interfere with the normal glucose metabolism process of the human body, and steadily increase the risk of type 2 diabetes in an obvious dose-dependent manner, which provides solid and reliable data support for tobacco control work in the prevention and management of type 2 diabetes. Much evidence indicates a positive association between smoking and the incidence of type 2 diabetes, wherein insulin resistance, beta-cell impairment, and chronic inflammation are putative mediating pathways. In patients with established diabetes, smoking further elevates susceptibility to

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complications, most notably those affecting the microvasculature and macrovascular system. While diabetics face a higher risk of these events, the magnitude of the smoking-related relative risk (RR) increment does not differ significantly from that observed in non-diabetic individuals [6].

3.2. Secondhand smoke exposure and type 2 diabetes risk

Most prior studies have focused on the relationship between type 2 diabetes risk and active smoking. However, mounting data indicates that secondhand smoking could also be problematic [7].

A large number of targeted statistical studies focusing on the correlation between secondhand smoke exposure and type 2 diabetes risk have also formed consistent, stable and highly credible research conclusions. Long-term chronic passive exposure to secondhand smoke can significantly increase the risk of glucose metabolism disorders and clinical type 2 diabetes in non-smoking populations. Meanwhile, secondhand smoke (SHS), which contains more than 7,000 chemicals, has long been recognized as a risk factor for diabetes [8]. Comparative statistical analysis shows that non-smokers with long-term secondhand smoke exposure have higher levels of inflammatory markers, worse blood lipid profiles and higher insulin resistance levels than those without obvious exposure. Their overall risk of suffering from type 2 diabetes is also significantly higher than that of the non-exposed control group. Although the dose-response relationship between secondhand smoke exposure and diabetes risk is relatively weaker than that of active smoking due to the low exposure intensity and small dose, the statistical correlation between them is still stable and credible in multiple independent studies. This means that even low-concentration, long-term and intermittent passive smoke exposure is enough to damage the normal metabolic regulation function of the human body, significantly reduce insulin sensitivity, and increase the body's susceptibility to diabetes. Therefore, secondhand smoke exposure should be officially included in the list of controllable and preventable risk factors for type 2 diabetes, and its potential metabolic harm deserves sufficient attention and key prevention in family health protection and public health management.

3.3. E-cigarette use and type 2 diabetes risk

For the emerging electronic cigarette exposure, most of the current statistical analyses, comparative studies and observational investigations have shown a clear and stable positive correlation between long-term electronic cigarette use and increased risk of type 2 diabetes. It is worth noting that the current relevant research has a relatively short follow-up time and insufficient sample size, so the relevant statistical data are not as sufficient and comprehensive as those of traditional smoking, and there are individual differences in research conclusions in different regions and groups. Although the relevant research evidence is not as abundant and sufficient as that of traditional active smoking, the existing objective test data and group statistical comparison results show that long-term electronic cigarette users have higher insulin resistance index, a higher prevalence of prediabetes and a higher rate of persistent blood glucose abnormality than non-users. Statistical differences in key metabolic indicators clearly indicate that the aerosol and nicotine components produced by electronic cigarettes can interfere with the normal blood glucose regulation mechanism of the human body, damage the activity and repair ability of islet cells, reduce the body's insulin sensitivity, and promote the occurrence and development of lipid metabolism disorders. Compared with traditional active smoking, the overall harmful effect of electronic cigarette use on diabetes risk is relatively mild in the comprehensive statistical data, but it still shows a stable positive correlation trend, rather than no harmful effect at all. It can be fully concluded that the use of electronic cigarettes is not harmless to human metabolic health, and long-term use will still significantly increase the risk of glucose

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metabolism disorders and type 2 diabetes. These important statistical findings provide key evidence support for strengthening the supervision of electronic cigarette products, correcting public cognitive biases, and carrying out targeted metabolic health education for young people and high-risk groups.

Thus, smoking cessation confers clear benefits by reducing or slowing the risk of cardiovascular morbidity and mortality among individuals with diabetes, similar to its effects in the general population [1, 9].

4. Conclusion

This review systematically elaborates on the classification, hazards and statistical correlations of active smoking, secondhand smoke exposure and electronic cigarette use with type 2 diabetes mellitus. Statistical analyses reveal that each tobacco exposure is significantly associated with an increased risk of type 2 diabetes, and a clear dose-response relationship exists between exposure intensity, duration and diabetes risk, suggesting that tobacco exposure is an independent and modifiable risk factor for type 2 diabetes. This paper also integrates relevant evidence to explain the potential internal connection between tobacco exposure and abnormal glucose metabolism, providing a clear theoretical basis for diabetes prevention and tobacco control.

However, this review still has several limitations. First, this study only summarizes existing literature and lacks original data analysis and quantitative calculation, resulting in insufficient accuracy of statistical result verification. Second, this review fails to conduct in-depth comparative analysis of different e-cigarette types, use frequencies and exposure doses, leading to an incomplete understanding of its metabolic hazards.

In the future, the research direction of tobacco exposure and type 2 diabetes will focus on more detailed quantitative research, large-scale prospective population studies, and accurate dose-response effect analysis. In-depth mechanistic studies at the molecular and cellular levels will also be strengthened to clarify the specific pathways of tobacco components affecting glucose metabolism. With the deepening of research, more reliable evidence will be provided for clinical prevention, public health policy formulation and targeted risk warning of type 2 diabetes related to tobacco exposure.

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