Smoking cessation is a cheap and effective way to reduce cardiovascular risk

Smoking, smoking cessation and cardiovascular risk

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Summary

Smoking is a widely-recognised risk factor for several diseases, including cardiovascular disease, pulmonary disease and cancer at several sites, and is a primary cause of premature morbidity and mortality.

As early as 1964, the first report of the US Surgeon General pointed out that cigarette smoking has detrimental health effects, summarising studies from the United Kingdom and the United States.

Following those initial efforts, a vast body of research grew across all fields of medicine, surgery and public health, focusing on the role of tobacco as cardiovascular risk factor, the danger of active and passive smoking, and the health effects of smoking cessation.

This review aims to summarise the available evidence on the effect of tobacco on cardiovascular morbidity and mortality and the effect of smoking cessation on cardiovascular risk and disease.

Key words: tobacco; smoking; smoking cessation; cardiovascular risk

Introduction

Smoking is a widely-recognised risk factor for several diseases, including cardiovascular disease, pulmonary disease and cancer at several sites [1], and as such is a major cause of premature morbidity and mortality worldwide [2].

The first report of the US Surgeon General in the 1960s described the detrimental health effect of cigarette smoking [3]; it marked a turning point in the public recognition of the harms and hazards of smoking, as well as of cigarette consumption per capita in the US [1].

Following these initial efforts, mainly based on studies conducted in England and USA, considerable research has expanded the concept from cigarettes to the effects of smoked and smokeless tobacco on health [4, 5], and the role of active and passive smoking as cardiovascular risk factors [6–8].

Moreover, observational and interventional trials have assessed the health effects of smoking cessation achieved with behavioural and pharmacological approaches [9–15].

Tobacco as cardiovascular risk factor

Data reported in the 1950s by Doll and Peto [16] showed that smoking increased death rates due to coronary heart disease (CHD) in British physicians and underlined that smokers had a highly elevated relative risk for dying from CHD. This effect was significant at all ages and was particularly strong in adults younger than 45 years of age.

Further studies have consistently shown that the number of cigarettes smoked on a regular basis strongly correlates with the risk of myocardial infarction in both sexes [17]. Epidemiological data [18] and randomised studies have been summarised in systematical reviews [19, 20]. Pipe and cigar smoking were also clearly shown to increase death rates from several forms of cancer, especially in the oropharynx, and the CHD risk [21–24].

Passive or involuntary smoking occurs when nonsmokers breathe "side-stream" smoke produced by burning tobacco, or "main-stream" smoke exhaled by smokers. In the Nord-Trøndelag Health Survey 2 (HUNT2), which was conducted in 1995–1997 and included 65229 participants aged 20 years or older, exposure to passive smoking increased lung cancer risk approximately 1.50 times [25]; prospective study results suggest that the relative risk of CHD is also raised appreciably [26–28].

More recently, the INTERHEART Study [6] confirmed that no tobacco product may be considered as "safe" for the cardiovascular system. Data from patients recruited in 52 different countries showed that smoked as well as smokeless tobacco was associated with an increased risk of myocardial infarction.

Tobacco affects the cardiovascular system directly and indirectly through unfavourable effects on a large variety of CHD risk factors. Although average smokers are leaner that nonsmokers, oxidation of lipids [29, 30] and lower high-density lipoprotein cholesterol were found more often in smokers than nonsmokers [31, 32]. Regular tobacco users have higher fasting blood glucose due to reduced insulin sensitivity [33–36] and typically have higher blood pressures and heart rate than nonsmokers [33, 37–39].

Other mechanisms by which smoking exerts its negative cardiovascular effects include a hypercoagulable [40, 41] and proinflammatory [42–44] state, characterised by increased fibrinogen levels, greater thromboxane A2, increased platelet activation, increased isoprostanes, and increased C-reactive protein. Smoking increases carbon monoxide in the body, and reduces oxygen delivery to vital tissues [45–47], activating the sympathetic nervous system and causing vasoconstriction beyond the direct vascular effects of nicotine (fig. 1) [48–52].

Second hand smoking and cardiovascular risk

The negative effects of tobacco on the cardiovascular system are not limited to active users. People who are not using tobacco but are exposed to other's tobacco smoke have been shown to have an increased risk of CHD.

Second-hand smoke, involuntary or passive smoking are defined as passive inhalation of a smoke mixture comprising 15% mainstream smoke (inhaled and exhaled by the smoker) and 85% side-stream or tertiary smoke (which is released from the burning tip of a cigarette between puffs). Tertiary smoke results from burning tobacco at a much higher temperature and with less available oxygen, and so toxic substances such as carbon monoxide, methane, cyanides and carcinogenic benzene are found in greater amounts than in mainstream smoke (nicotine 2:1, benzene 10:1, nitrosamines 100:1) [53].

A meta-analysis of 10 prospective cohort studies and 8 case-control studies [54] showed that second-hand smoke increases the relative risk of cardiovascular morbidity and mortality by 25% among nonsmokers. Passive smoking was consistently associated with an increased relative risk of coronary heart disease in men and in women, as well as in those exposed to smoking at home or in the workplace. The meta-analysis by He and colleagues [54] identified a significant dose-response relationship, with relative risks of 1.23 and 1.31 for nonsmokers who were exposed to the smoke of 1 to 19 cigarettes per day and 20 or more cigarettes per day, respectively, as compared with nonsmokers not exposed to smoke (p = 0.006 for linear trend).

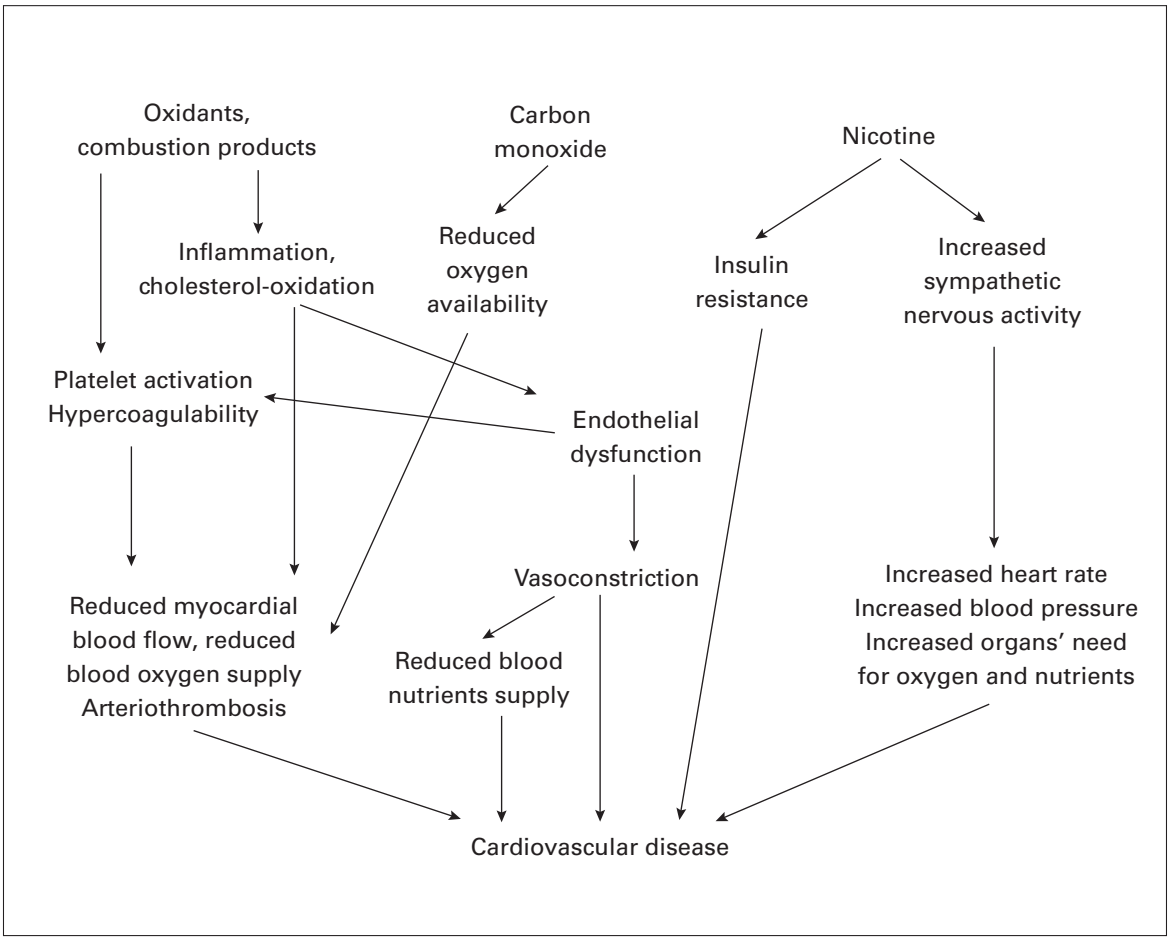

Figure 1: Known mechanisms linking tobacco smoking and cardiovascular diseases.

The Auckland stroke study [55] pointed out that the risk of acute stroke was also increased. Exposure to environmental tobacco smoke was associated with an increased risk for stroke in men (odds ratio [OR] 2.06; 95% confidence interval [CI] 1.34–3.17) and women (OR 1.50; 95% CI 1.01–2.21). After adjustment for age and sex, exposure to environmental tobacco smoke was associated with an overall increased risk of stroke (OR 1.74; 95% CI 1.31–2.32).

The CARDIO2000 case-control study [56] investigated the association between environmental tobacco smoke exposure (at least 30 minutes a day) and the risk of developing acute coronary syndromes. The results of this study showed that, in a follow-up of 20 years, the risk of ischaemic heart disease in people without preexisting cardiovascular damage was increased by 1.61 in the group with high exposure (cotinine level 2.8–14 ng/ml).

The INTERHEART study showed that in subjects who had never smoked cigarettes, passive smoking accounted for 10.8 and 18.6% of all myocardial infarctions in women and men, respectively [6].

More recently, an analysis of 24 968 men and women followed up for 11 years in the Tromsø Study [57] showed that the risk of myocardial infarction increased linearly for both active and passive smoking in both sexes, but rather consistently more strongly for women than for men. Restricting the analysis to never-smokers' passive smoking was found to be an independent risk factor for myocardial infarction in never-smoking women.

The cardiovascular risk associated with environmental tobacco smoke is rapidly reversible, as multiple studies performed all around the world have documented a rapid decline in hospital admissions for myocardial infarction after the adoption of smoking bans [58–63].

Smoking cessation and cardiovascular risk and diseases

Ceasing to smoke is not an easy challenge: people who try to stop smoking without additional aids face a high relapse risk. Individuals may have moderately high success in the short term with this approach, but the results at 12 months are generally not very encouraging (1–5% are still nonsmokers) [64].

The smoking cessation issue is complex and depends on individual factors, as well as factors that are prevalent in the society where the smoker lives.

Determinants of smoking cessation have been studied in the 1999–2001 follow-up of the European Community Respiratory Health Survey (ECRHS) [65]. Data from 9053 adults from 14 countries were collected. Overall, 28.8% of the participants stopped smoking during the follow-up; older age, and higher education level and social status were positively associated with a higher cessation rate.

In a longitudinal study of 2564 middle-aged smokers, nearly 40% quit smoking during an approximate 10-year study period [66]. The authors found an association between smoking cessation and fewer smoking years, as well as a high level of education. Neither presence of airway disease nor the known cardiovascular risk factors hypertension and diabetes seemed to predict smoking cessation. This was true also for those who developed airway disease, diabetes or hypertension during the study period. Known heart disease at baseline did not predict smoking cessation, but an acute episode of ischaemic heart disease during the study period was associated with a four-fold increase in the chance of smoking cessation. Other studies have shown that the presence of tobacco-related disease was a positive predictor for smoking cessation [67].

When smokers are able to stop, and remain nonsmokers, the rates of new cardiovascular events decrease significantly a few years after smoking cessation. In the Multiple Risk Factor Intervention Trial [68], the men who stopped smoking during the first 3 years of the study experienced a relative risk for CHD of 0.38 compared with those who had continued to smoke during the 10.5 years of follow-up. Moreover, Critchley and Capewell [69] showed that the mortality risk in patients with coronary heart disease was significantly reduced by smoking cessation.

Weight gain after smoking cessation, even though with wide variation, has been well documented [70] and remains an issue because it may deter smokers from quit attempts and women are probably less likely to respond to nicotine replacement therapy [71]. In combination with other cardiovascular risk factors, weight gain may subtract slightly from the health benefits of cessation [72] and ought to be counteracted by dietary and exercise counselling in the outpatient setting or during rehabilitation.

All smoking cessation interventions, such as personal advice from medical personnel [73–75] or via media/Internet [76, 77] and the use of nicotine replacement therapy [78], bupropion [79, 80] or and varenicline [79, 81] increase the rate of smoking cessation [82], are very cost-effective [83] and do not have any negative effect on cardiovascular risk [84, 85]. Swiss data [86] demonstrate the effectiveness of a counselling intervention in the setting of hospitalisation for an acute coronary syndrome hospitalisation in inducing patients to take up further counselling. Public policy interventions are beyond the scope of this review, but multinational evidence seems

clearly in favour of public smoking bans as a means to reduce cardiovascular and respiratory disease burden [60, 87, 88].

However, smoking cessation receives generally less attention than counselling and pharmaceutical therapy for other classic cardiovascular risk factors such as hypertension, possibly because of physicians' concerns that the intervention may take an excessive amount of time with limited reimbursement. It has been shown [89] that approximately 3 minutes of a general practitioner's consultation time is enough to effectively initiate and even maintain cessation effects by following the 5A mnemonic: "(1) ask the patient if he or she uses tobacco, (2) advise him or her to quit, (3) assess willingness to make a quit attempt, (4) assist those who are willing to make a quit attempt, and (5) arrange for follow-up contact to prevent relapse" [89].

Vaporisers and heat-not-burn devices either heat a liquid with or without nicotine or heat an amalgam of tobacco to a lower temperature than a burning cigarette does. In 2016, an updated Cochrane review on whether these "e-cigarettes" have a place as aides in smoking cessation was published [90]; it summarised 24 studies of which only 2 were eligible randomised controlled trials, including 662 patients. The sparse data from randomised controlled trials is the most pressing problem as it has been and persists to be in other areas of research on harm by tobacco. Aside from the question of the safety of such devices in comparison with tobacco or in their own right, we fully endorse the conclusion 17–10 from the 2018 National Academies of Sciences consensus report: "Overall, there is limited evidence that e-cigarettes may be effective aids to promote smoking cessation" [91].

Conclusion

Smoking cessation is a cheap way to prevent cardiovascular disease and reduces the associated morbidity and mortality. Informing people who use tobacco regularly that quiting smoking may rapidly reduce the risk of future cardiovascular events may be highly motivating. Despite unequivocal evidence, tobacco is often a forgotten cardiac risk factor, receiving less attention than treating hypertension, hyperlipidaemia or diabetes.

Routine identification of smoking status, advising cessation and referring to counselling resources should be standard practice. Moreover, it is important that communication to people using tobacco and to the general population should include information about second-hand smoke as a risk factor for cardiovascular disease.

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