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HEALTH IMPACT OF TOBACCO SMOKING: CARDIOVASCULAR, RESPIRATORY AND NEOPLASTIC DISEASES

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ABSTRACT

Introduction and purpose: Smoking remains one of the leading preventable causes of premature mortality worldwide. A structured search of PubMed and Google Scholar databases was performed to identify clinical, epidemiological and experimental evidence, including original studies, systematic reviews and meta-analyses, on the impact of active and passive smoking. This narrative review summarises current evidence on the health effects of tobacco smoking and secondhand smoke exposure, with a particular focus on cardiovascular, respiratory and neoplastic diseases.
Description of the state of knowledge: Tobacco smoke contains numerous toxic and carcinogenic compounds that promote endothelial dysfunction, oxidative stress and chronic inflammation, thereby accelerating atherosclerosis and increasing the risk of myocardial infarction, stroke and other vascular events. Long-term exposure damages the airways and lung parenchyma, leading to chronic obstructive pulmonary disease (COPD), worse asthma control and increased susceptibility to respiratory infections. Smoking is also a major driver of cancer, accounting for most lung cancer cases and contributing substantially to malignancies of the upper aerodigestive tract, pancreas and bladder. Secondhand smoke, although involving lower levels of exposure, is associated with higher risks of cardiovascular disease, respiratory illness, type 2 diabetes and adverse outcomes in children and older adults.
Conclusion: The available evidence clearly shows that there is no safe level of exposure to tobacco smoke. Smoking cessation and effective protection from secondhand smoke are essential components of strategies to prevent cardiovascular, respiratory and neoplastic diseases and should remain central priorities in public health policy and clinical practice.

KEYWORDS

Tobacco Smoke Pollution, Smoking, Cardiovascular Diseases, Neoplasms, Respiratory Tract Diseases

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Introduction

Tobacco was introduced to Europe following the geographical discoveries of the late 15th century and soon gained a reputation as a substance with medicinal properties. During the 16th and 17th centuries people treated different medical conditions with tobacco-based remedies through infusion, powder forms and smoking methods which earned tobacco the names "holy herb" and "divine medicine" [1]. The 19th century saw industrial cigarette production emerge and technological progress which led to smoking becoming a common practice. In the first half of the 20th century, the practice was socially accepted and even endorsed by the medical community—numerous advertising campaigns featured physicians, and smoking prevalence among healthcare professionals remained high [2]. Epidemiological studies from the mid-20th century established tobacco smoking as a direct cause of lung cancer and multiple other malignancies, marking a turning point in the perception of this habit [3]. Despite the growing body of scientific evidence, a considerable proportion of the medical community in the 1960s still did not regard this relationship as conclusively proven [4]. The evolution of views on the health effects of tobacco smoking exemplifies a gradual process in which traditional beliefs in its alleged therapeutic properties were replaced by evidence-based scientific knowledge.

Methods

A literature review was conducted using medical databases such as PubMed and Google Scholar. Articles were searched in English using the following keywords and their combinations: “cigarettes,” “tobacco smoke,” “passive smoking,” “cardiovascular risk,” “chronic obstructive pulmonary disease” and “carcinogenesis”.

Impact on Cardiovascular System

Large prospective cohort studies consistently show that current smokers have a substantially higher risk of cardiovascular events than never-smokers. In an analysis encompassing 1.35 million person-years of follow-up, current smoking was associated with a markedly increased incidence of acute myocardial infarction compared with never smoking (HR = 2.45; 95% CI 2.2–2.7), with risk rising in a clear dose–response fashion with the number of cigarettes smoked per day and duration of smoking [5]. Similar patterns have been demonstrated for a wide spectrum of cardiovascular outcomes, including stroke, heart failure, peripheral arterial disease (PAD), pulmonary embolism and aortic aneurysm [5,6,7]. These excess risks are observed not only in middle-aged adults, but also among older populations, where the number of smoking-attributable cardiovascular events is particularly high [7].

Sex-specific analyses indicate that women may be especially vulnerable to the cardiovascular effects of tobacco. Female smokers have higher cardiovascular mortality than male smokers, and for a given level of tobacco exposure, women appear to have roughly a 25% higher risk of developing coronary heart disease than men. Experimental work suggests that this enhanced susceptibility may be related, at least in part, to differences in the expression of genes involved in thrombin signalling and other pro-thrombotic pathways, which could amplify smoking-induced platelet activation and coagulation [8]. These findings underline the need for particular attention to smoking prevention and cessation in women, especially in regions where smoking prevalence remains high.

The pathophysiological mechanisms linking cigarette smoking with cardiovascular disease are multifactorial and mutually reinforcing. Tobacco smoke contains thousands of chemicals, including oxidants, particulate matter (so called TAR – Total Aerosol Residue) and gaseous components such as carbon monoxide (CO). Carbon monoxide has an affinity for haemoglobin approximately 250 times greater than that of oxygen, leading to the formation of carboxyhaemoglobin and a reduction in blood oxygen-carrying capacity. This state of functional anaemia contributes to tissue hypoxia and may precipitate myocardial ischaemia or stroke in the presence of pre-existing atherosclerotic lesions [6,9].

Nicotine activates the sympathetic nervous system, leading to tachycardia, vasoconstriction and increased blood pressure, which raise myocardial oxygen demand while simultaneously reducing coronary blood flow [6]. Chronic exposure to tobacco smoke promotes endothelial dysfunction, impairs vasodilation mediated by nitric oxide (NO), increases oxidative stress and enhances low-density lipoprotein (LDL) oxidation, all of which accelerate the development and progression of atherosclerotic plaques. Smoking also alters haemostatic balance by increasing fibrinogen levels, promoting platelet aggregation and raising blood viscosity, thus predisposing to thrombus formation and vascular occlusion [6,8]. The main mechanisms are summarised in Table 1.

Table 1. Pathophysiological mechanisms linking cigarette smoking with cardiovascular diseases [6-9]

Pathophysiological mechanism	Description of action	Cardiovascular consequence
Increased blood pressure and heart rate	Nicotine-induced sympathetic activation leads to vasoconstriction, tachycardia and elevated blood pressure.	Chronic hypertension and increased myocardial oxygen demand.
Endothelial dysfunction	Oxidative stress and direct toxic effects on the endothelium impair nitric-oxide-mediated vasodilation and promote inflammation.	Reduced arterial compliance and facilitation of atherogenesis.
Atherogenic lipid profile	Smoking increases LDL, lowers HDL levels and promotes LDL oxidation.	Accelerated plaque formation and progression of atherosclerosis.
Enhanced blood coagulability	Elevated fibrynogen level, increased platelet activation and higher blood viscosity favour thrombus formation.	Occlusion of coronary and cerebral arteries; acute ischaemic events.
Carboxyhaemoglobin formation	Carbon monoxide binds haemoglobin with high affinity, reducing oxygen delivery to tissues.	Tissue hypoxia, aggravation of myocardial and cerebral ischaemia.

Recent data highlight that new nicotine delivery systems do not eliminate cardiovascular risk. Acute exposure to electronic cigarettes has been shown to transiently increase heart rate, blood pressure and to stiffen the arteries, indicating rapid adverse effects on vascular function and autonomic balance [6]. Although the

long-term cardiovascular consequences of exclusive e-cigarette use are still being evaluated, current evidence suggests that they cannot be considered harmless substitutes, particularly for individuals with established cardiovascular disease [6,10].

Beyond its direct impact on the coronary and cerebral circulation, smoking contributes to a broader pattern of cardiometabolic morbidity. Smokers more often have additional risk factors such as dyslipidemia, hypertension and diabetes, and the combination of these exposures produces more than additive increases in absolute cardiovascular risk [5,7]. In population-based cohorts, a substantial proportion of smoking-attributable deaths in middle and older age is due to cardiovascular causes, with coronary heart disease and stroke predominating [5]. Among patients with established cardiovascular disease, continued smoking is consistently associated with higher rates of recurrent events and mortality, whereas cessation leads to clinically meaningful improvements in prognosis [6-8].

Despite the magnitude of risk, smoking cessation yields substantial and rapid benefits for the cardiovascular system. Large cohort studies and meta-analyses show that the risk of myocardial infarction and stroke begins to fall soon after quitting; within about one year the risk of coronary events is reduced by roughly half compared with continued smoking, and after 10–15 years it may approach that of never-smokers, depending on prior smoking intensity and the presence of other risk factors [6,7]. Even among older adults and in patients with established cardiovascular disease, cessation is associated with meaningful reductions in recurrent events and mortality [7]. International organisations therefore emphasise that identification and treatment of tobacco dependence, using behavioural support and, when appropriate, pharmacotherapy, should be a routine component of cardiovascular care and secondary prevention programmes [6,11].

Smoking and Cancer

Tobacco use is recognised as one of the leading preventable causes of cancer worldwide. It is estimated to be responsible for almost 30% of all cancer deaths [12] and is consistently identified as the single most important modifiable risk factor for cancer incidence and mortality at the population level [13,14]. This substantial burden reflects both the widespread nature of exposure and the fact that smoking increases the risk of multiple tumour types, including cancers of the lung, head and neck, bladder and pancreas (Table 2).

Table 2. Selected tobacco-dependent cancers [13-21]

Cancer type	Mechanisms and clinical features	Additional data on tobacco-related burden
Lung cancer	Direct exposure of bronchial epithelium to multiple inhaled carcinogens; strong association with squamous-cell and small-cell carcinoma.	Around 85% of lung cancer cases are attributable to active smoking.
Oral cavity, pharyngeal and laryngeal cancers	Chronic irritation and DNA damage in upper aerodigestive mucosa; synergy with alcohol; strong causal association, especially in HPV-negative tumours.	About 75% of cases are linked to tobacco use, often in combination with alcohol.
Oesophageal cancer	Direct contact of smoke constituents with oesophageal mucosa; synergy with alcohol and gastro-oesophageal reflux; substantial excess risk among smokers.	Accounts for approximately 50% of cases, particularly when combined with alcohol.
Pancreatic cancer	Systemic delivery of carcinogens to the pancreas; pro-inflammatory and metabolic effects of smoking.	Roughly 20–25% of cases are attributable to smoking.
Bladder cancer	Urinary excretion of carcinogens with prolonged contact with urothelium; high relative risks in current smokers.	Smoking accounts for 50–65% of bladder cancers in men and 20–30% in women.

Tobacco smoke is a complex aerosol composed of thousands of chemicals. Contemporary analytical work indicates that tobacco and tobacco smoke together contain more than 9,500 chemical compounds, and 83 of these have been classified as carcinogens by the International Agency for Research on Cancer (IARC). These include aromatic amines, polycyclic aromatic hydrocarbons, tobacco-specific N-nitrosamines, volatile organic compounds such as benzene and formaldehyde, and a range of heavy metals and radioactive elements [15]. Zagà et al. reported measurable activity of the alpha-emitter polonium-210 in bronchial lavage samples from patients with suspected lung cancer, with detectable levels in all current and former smokers, highlighting the presence of radioactive carcinogens within the bronchial tree [22]. Many of these compounds are converted

in the body into reactive forms that attach to DNA, leading to the formation of adducts and characteristic patterns of mutations. At the same time, cigarette smoke promotes oxidative stress, chronic inflammation, impairment of epithelial barrier function and dysregulation of reparative processes in the airways, collectively creating a environment that favours malignant transformation [15,23].

Lung cancer remains the malignancy most strongly associated with tobacco use. The risk increases with both the amount smoked and the duration of smoking. On a population level, around 85% of lung cancer cases are attributable to active smoking [13,21]. In the Norwegian Women and Cancer (NOWAC) cohort, more than eight in ten lung cancer cases were estimated to be attributable to active smoking, and the excess risk among former smokers declined only gradually with increasing time since cessation, approaching that of never-smokers after many years of abstinence [21]. Histologically, smoking is strongly associated with squamous cell carcinoma and small-cell lung carcinoma, but also contributes substantially to adenocarcinoma of the lung [13]. The accumulation of multiple carcinogens, including polonium-210, within the bronchial epithelium provides a biological explanation for these strong epidemiological observations [22].

Cancers of the oral cavity, pharynx and larynx are also closely linked to tobacco use. Harmful substances in inhaled smoke directly injure the mucosa of the upper aerodigestive tract, leading over time to dysplasia and malignant transformation [13,15]. Genetic studies based on Mendelian randomisation provide further support a causal relationship between tobacco consumption and head and neck squamous cell carcinoma, especially in tumours that are negative for human papillomavirus (HPV) [20]. Studies in patients with oral and oropharyngeal cancer show that a diagnosis of malignancy does not always lead to smoking cessation, and continued smoking in this setting is associated with poorer treatment tolerance and outcomes [16].

Beyond the lung and upper aerodigestive tract, cigarette smoking contributes to the development of several other solid tumours. Pancreatic cancer is one of the deadliest malignancies, and in many populations the number of new cases is almost the same as the number of deaths. Epidemiological studies consistently identify smoking as a major modifiable risk factor for pancreatic cancer, with heavy smokers having roughly double the risk of never-smokers. It is estimated that approximately 20-25% of pancreatic cancer cases may be attributable to tobacco use, making smoking one of the most important targets for primary prevention of this disease [17]. Proposed mechanisms include direct exposure of pancreatic tissue to carcinogens transported via the bloodstream, the diabetogenic and pro-inflammatory effects of smoking, and interactions with other metabolic risk factors such as obesity and alcohol use [15,17].

Bladder cancer represents another malignancy for which tobacco plays a dominant role. Many of the carcinogens in cigarette smoke are excreted in the urine, exposing the urothelial lining of the bladder to high local concentrations of aromatic amines and other reactive compounds [15]. Large prospective cohort data show that current smokers have approximately three times higher bladder cancer risk than never-smokers, and that risk declines gradually after quitting but remains elevated for many years [18]. Population-level analyses suggest that smoking accounts for roughly 50–65% of bladder cancer cases in men and 20–30% in women, underscoring its central role in the pathogenesis of this tumour [18]. A more recent dose–response meta-analysis confirmed that both smoking intensity and duration are strongly associated with bladder cancer risk, with no evidence of a safe threshold of exposure [19].

For several other cancers, smoking acts as an important co-factor rather than the predominant cause. In cervical cancer, persistent infection with high-risk HPV types is the necessary cause, but cigarette smoking independently increases risk and shows a clear dose-response relationship. The mechanisms are thought to involve local immunosuppression, alterations in cervical mucus and epithelium, and facilitation of viral persistence and progression from pre-invasive lesions to invasive cancer [24].

Importantly, the elevated cancer risk associated with smoking is at least partly reversible. Comprehensive reviews emphasise that smoking cessation reduces the incidence of many smoking-related cancers, most notably lung cancer, although the time for the risk to approach that of never-smokers may extend to two decades or more [13,17,21]. In patients already diagnosed with malignancy, continued smoking is linked to inferior response to oncological treatment, higher rates of treatment-related toxicity, increased risk of second primary tumours and worse overall survival. Conversely, cancer patients who stop smoking experience meaningful improvements in treatment outcomes, quality of life and long-term prognosis [12,13].

Respiratory Diseases

Tobacco smoking is a major cause of chronic respiratory morbidity and mortality worldwide and remains the leading risk factor for the development and progression of chronic obstructive pulmonary disease (COPD). It also worsens the clinical course of asthma and other chronic airway disorders [25,26]. According to recent summaries of evidence from the World Health Organization (WHO), most COPD cases in high-income countries can be attributed to long-term exposure to tobacco smoke, and smoking cessation is the single most effective intervention to slow disease progression [25].

The pathophysiological effects of smoking on the respiratory tract are multifactorial. Tobacco smoke contains thousands of chemicals, including oxidants, free radicals and a wide spectrum of toxic organic and inorganic compounds that induce oxidative stress and chronic inflammation in the airway epithelium [15,25]. Continuous exposure damages epithelial cells, disrupts tight junctions and impairs the integrity of the mucosal barrier, reducing its ability to protect against inhaled irritants and pathogens. This injury is accompanied by increased production of pro-inflammatory mediators, recruitment of neutrophils and macrophages and activation of proteolytic enzymes, all of which contribute to structural remodelling of the bronchial tree [25,27].

Experimental studies have provided additional insight into the mechanisms by which cigarette smoke alters the airway epithelium. In vitro models show that tobacco smoke extract impairs epithelial wound repair and modulates epithelial–mesenchymal transition (EMT) processes, with a shift towards a more mesenchymal phenotype that favours fibrosis and structural distortion of the airways. It has also been shown to interfere with Notch-1 signalling, a key pathway regulating epithelial cell differentiation and proliferation, leading to an imbalance between ciliated and mucus-producing cells [23]. These changes result in the loss of ciliated cells, goblet cell hyperplasia and increased mucus production, which together impair mucociliary clearance and hinder the removal of inhaled particles and microorganisms from the airways [23,27].

Clinical and experimental data show that tobacco smoke profoundly alters the airway surface microenvironment. Observations highlight that smoking disrupts the composition of the airway surface liquid, affects epithelial immune defences and changes the expression of antimicrobial peptides [27]. These alterations make the bronchial epithelium more susceptible to colonisation by pathogens and less capable of providing an effective host defence. Over time, the combination of impaired mucociliary clearance, chronic inflammation and defective epithelial repair establishes a chronic injury state that forms the biological basis for COPD and other smoking-related respiratory diseases (Table 3).

Table 3. Pathophysiological mechanisms linking tobacco smoking with respiratory diseases [23,25,27]

Mechanism	Description	Consequences for the respiratory tract
Damage to the airway epithelium	Toxic components of tobacco smoke damage epithelial cells, disrupt tight junctions and impair barrier integrity.	Reduced protective function of the mucosa, increased vulnerability to irritants and pathogens, and initiation of chronic inflammation.
Impaired epithelial repair and epithelial–mesenchymal transition (EMT)	Cigarette smoke delays epithelial wound repair, modulates EMT and dysregulates Notch-1 signalling.	Structural distortion of the airways, fibrosis, loss of ciliated cells and goblet cell hyperplasia, contributing to airflow limitation.
Mucus hypersecretion and impaired mucociliary clearance	Smoking increases mucus production and alters ciliary function, leading to less effective mucociliary transport.	Accumulation of mucus and pollutants in the airways, favouring infection and chronic bronchitis.
Chronic inflammation and protease–antiprotease imbalance	Persistent inflammatory cell recruitment and release of proteolytic enzymes exceed local antiprotease defences.	Parenchymal destruction and emphysema, reduced lung elasticity and irreversible airflow limitation.
Altered innate immune responses	Cigarette smoke modifies the composition of airway surface liquid and impairs epithelial innate immune signalling.	Increased susceptibility to viral and bacterial infections and more frequent exacerbations of chronic respiratory diseases.

COPD is the main chronic respiratory disease caused by smoking and is characterised by persistent respiratory symptoms and airflow limitation that is not fully reversible. COPD encompasses chronic bronchitis with mucus hypersecretion, small-airway disease and emphysematous destruction of the lung parenchyma, all of which are strongly linked to long-term tobacco exposure [25]. Smokers with COPD typically exhibit an accelerated decline in lung function, measured by forced expiratory volume in one second (FEV1), compared with never-smokers. Recurrent exacerbations further damage the lungs, leading to progressive dyspnoea, exercise intolerance and chronic respiratory failure in advanced stages [25,28]. COPD is a leading cause of disability, long-term oxygen therapy and premature retirement from work, which translates into a substantial socioeconomic burden [29].

Cigarette smoking also has important consequences for asthma. In adults, active smoking is associated with poorer symptom control, more frequent exacerbations and a greater need for rescue medication. Studies show that smokers with asthma often have a reduced response to inhaled corticosteroids and may require higher doses or additional controller therapies to achieve similar levels of disease control as non-smokers [26]. Smoke induces increased mucus production, thickening of the airway wall and low-grade neutrophilic inflammation, which may further contribute to persistent airflow limitation in a subset of patients with features overlapping asthma and COPD. These findings indicate that smoking does not simply add to existing risk but also alters the disease pattern and the response to therapy [25,26].

Beyond chronic airway obstruction, smoking increases susceptibility to respiratory infections. Smokers experience more frequent episodes of acute bronchitis and pneumonia and are at higher risk of hospitalisation for lower respiratory tract infections than never-smokers. In patients with established COPD or asthma, such infections are a major trigger of exacerbations, which are associated with declines in lung function, reduced quality of life and increased short- and long-term mortality [25,26].

Smoking cessation is the cornerstone of respiratory disease prevention and management. Evidence-based reviews indicate that complete cessation is the only intervention that can substantially slow the decline in lung function in COPD and reduce the frequency of exacerbations. Former smokers with COPD have fewer lower respiratory tract infections and exacerbations than current smokers, and their lung function tends to stabilise or decline more slowly after quitting [28]. In asthma, smoking cessation is associated with improved symptom control and a better response to inhaled corticosteroids [26]. From a health-system perspective, integrating structured tobacco-dependence treatment into routine respiratory care, including spirometry-based case finding, motivational interventions and pharmacological support, is essential for reducing the long-term burden of smoking-related respiratory diseases [28].

Secondhand Smoke

Secondhand smoke, also known as passive smoking, is defined as involuntary exposure to smoke exhaled by smokers or released from burning tobacco products. It contains many of the same toxic and carcinogenic compounds as mainstream smoke, although often at lower concentrations, and is recognised as an important cause of preventable morbidity and mortality [15]. Meta-analyses indicate that exposure to secondhand smoke increases the risk of ischaemic heart disease in never-smokers by approximately 25–30% [30]. Global estimates indicate that passive smoking is responsible for roughly 1.3 million deaths each year, with a large share of these deaths occurring in low- and middle-income countries where smoke-free protections are often incomplete [11].

The cardiovascular effects of secondhand smoke are similar to those of active smoking, but on a smaller scale. Even short periods of exposure to ambient tobacco smoke can impair endothelial function, promote platelet activation and increase activity of the sympathetic nervous system [9,30]. Over the long term, recurrent exposure contributes to the development and progression of atherosclerosis and is associated with increased risks of coronary heart disease, stroke and, to a lesser extent, COPD in non-smoking adults [25,30].

Secondhand smoke also has substantial effects on the respiratory system. In adults, chronic exposure is associated with reduced lung function, more frequent respiratory symptoms and a higher incidence of lower respiratory tract infections [25,30]. In individuals with pre-existing COPD or asthma, secondhand smoke acts as an important trigger of exacerbations. Passive exposure may also attenuate the response to inhaled corticosteroids in some patients with asthma, contributing to suboptimal disease control [25,26].

Children are particularly vulnerable to the harmful effects of secondhand smoke because of their higher respiratory rate, ongoing lung development and the fact that they often cannot choose to leave smoke-filled spaces. Systematic reviews show that passive smoking in childhood is associated with an increased risk of lower respiratory tract infections, including bronchitis and pneumonia, as well as a higher likelihood of

developing or worsening asthma. Exposure has also been linked to more frequent wheeze, otitis media and, in infancy, an elevated risk of sudden infant death syndrome (SIDS) [31]. These early-life insults may have lasting consequences, predisposing to reduced lung function and chronic respiratory morbidity in adulthood [25,31].

Secondhand smoke is not only harmful to the heart and lungs but is also linked to a wider range of other chronic diseases. Epidemiological evidence indicates that long-term passive smoking increases the risk of type 2 diabetes. Prospective cohort studies demonstrate higher incidence of diabetes among those with regular household or workplace exposure compared with unexposed individuals. Proposed mechanisms include systemic inflammation, endothelial dysfunction and the promotion of insulin resistance, similar to pathways described for active smoking [32]. In older adults, exposure to secondhand tobacco smoke has been associated with worse performance on cognitive tests and an increased prevalence of cognitive impairment, suggesting that chronic passive smoking may contribute to neurodegenerative processes or cerebrovascular injury [33].

Secondhand smoke is also relevant in the context of cancer risk. Although the relative risk from passive exposure is lower than that seen with active smoking, secondhand smoke still increases the likelihood of lung cancer in never-smokers and may also play a role in the development of other malignancies [13,14]. This carcinogenic potential reflects the presence in sidestream and exhaled smoke of many of the same aromatic amines, polycyclic aromatic hydrocarbons and tobacco-specific nitrosamines that are found in mainstream smoke [13,15].

Reducing exposure to secondhand smoke is a central component of comprehensive tobacco control. Smoke-free legislation that prohibits smoking in workplaces, restaurants, bars and on public transport has been associated with substantial declines in population exposure and is considered a key measure to prevent diseases related to passive smoking [11,30]. However, a large proportion of exposure still occurs in private settings, particularly homes and cars, where children and other vulnerable individuals may be unable to avoid inhaling smoke [31]. Clinical guidelines therefore advise clinicians to routinely ask about secondhand smoke exposure in both adults and children, provide advice to promote smoke-free homes and cars, and refer smokers to structured cessation services, as these steps can help reduce the health impact of passive smoking [25].

Conclusions

Tobacco smoking remains one of the most important avoidable causes of premature death and disability worldwide. The evidence summarised in this review shows that active smoking affects multiple organ systems through interrelated toxic, inflammatory and thrombotic mechanisms. Its health effects are not limited to smokers themselves but also extend to people who are involuntarily exposed to secondhand smoke. Cigarette smoke contains a complex combination of carcinogens and toxins that together drive the development of a broad spectrum of non-communicable diseases.

The data reviewed here indicate that there is no safe level of exposure to tobacco smoke. For cardiovascular disease, even relatively low levels of exposure are associated with a measurable increase in risk, while heavier and longer smoking are linked to marked increases in the incidence of acute coronary events, stroke and other vascular complications. In the respiratory system, chronic inhalation of tobacco smoke causes structural and functional changes in the airways and lung parenchyma that underlie COPD, worsen asthma control and increase susceptibility to acute infections. When it comes to cancer, smoking accounts for the majority of lung cancer cases and contributes substantially to cancers of the upper aerodigestive tract, pancreas and bladder, while secondhand smoke adds further to the burden of malignancy in never-smokers.

Taken together, these findings show why smoking cessation should be a priority. Studies demonstrate that quitting smoking leads to substantial reductions in cardiovascular risk within a few years, with continued risk decline over subsequent decades. In respiratory disease, smoking cessation is the only intervention that clearly slows the long-term decline in lung function in COPD, and it is also an important component of improving symptom control in asthma. Among patients with cancer, stopping smoking is associated with better tolerance of oncological treatment, fewer complications and improved survival, underscoring the need to make the treatment of tobacco dependence an integral component of cancer care.

From a public health perspective, these findings reinforce the importance of comprehensive tobacco control strategies that combine strong regulation of tobacco products, smoke-free policies, price and tax measures, and broad access to evidence-based cessation support. Particular attention should be paid to protecting children and other vulnerable groups from secondhand smoke in homes, vehicles and public spaces. We still need further research to track new nicotine delivery systems, to provide more precise estimates of risk for individual diseases and to improve treatments for tobacco dependence. However, the main message is already clear: reducing exposure to tobacco smoke remains essential for preventing cardiovascular, respiratory and neoplastic diseases worldwide.

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