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Oxidative Stress, Inflammation and Diseases that Induced by Cigarette Smoke Toxicity

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Abstract: Smoking is a global public health crisis driven by highly addictive nicotine. These chemical hijacks the brain's reward system, fueling intense cravings and making cessation extremely difficult. The outcome is devastating: tobacco kills up to half its regular users, causing over 7 million annual deaths, which tragically includes 1.6 million non-smokers exposed to second-hand smoke. The toxicity stems from the smoke's staggering chemical complexity, featuring over 7,000 constituents. Crucially, sidestream smoke from the burning tip comprises 85% of the mixture and carries a higher concentration of toxic gases than inhaled mainstream smoke. The core mechanism of damage is a destructive cycle of oxidative stress and chronic inflammation. Smoking triggers an excessive production of free radicals that overwhelm the body's defenses, severely damaging cellular molecules like DNA and proteins. This pathological state immediately induces proinflammatory effects, leading to chronic, destructive inflammation. This cumulative damage is the foundation for smoking's broad disease spectrum. Tobacco use is the primary cause of lung cancer and is definitively linked to 25% of all global cancer deaths. It is also a major risk factor for heart disease, stroke, COPD, and Type 2 Diabetes. This addictive practice significantly drives global mortality and imposes massive economic and healthcare burdens worldwide. This review aim to elucidate the pathological mechanisms by which cigarette smoke toxicity is mediated through oxidative stress and chronic inflammation, and to discuss the resultant range of associated diseases and their public health impact.

Keywords: Cigarette Smoking, Oxidative Stress, Chronic Inflammation, Nicotine Addiction, Sidestream Smoke, Smoking Prevalence.

REVIEW PAPER

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INTRODUCTION

Smoking is the leading cause of death, and the detrimental effects of smoking on health, such as bronchitis, asthma, and cancer are well known. In addition, economic loss linked to smoking-related health issues among adults accounted for 8% of personal health care expenditures in 1998 [1]. Tobacco smoke is a known carcinogen, but the magnitude of smoking-related cancer risk depends on country-specific, generational smoking patterns [2]. The public health impact in countries like Iraq is evident, where the overall adult smoking prevalence was 18.7% in 2022, marked by a severe gender disparity with 36% prevalence among males versus 1.6% among females [3].

Chemical Composition and Toxicity of Tobacco Smoke

Most cigarettes consist of basic components: tobacco, chemical additives, a filter, and paper wrapping. The process involves burning the tobacco and inhaling the resulting smoke [4]. Tobacco smoke contains a staggering number of compounds; over 7000 chemical constituents have been identified, of which approximately 400 are routinely detected in both mainstream and sidestream smoke. Tobacco combustion produces a mixture of organic substances. Predominant gaseous phase constituents include carbon monoxide (CO), acetaldehyde, formaldehyde, acrolein, other carbonyls, nicotine, and tobacco-specific nitrosamines. Tobacco smoke is categorized into two forms: mainstream smoke, which is drawn through the tobacco into the mouth, and sidestream smoke, which emanates from the burning end of a cigarette [5]. The complete mixture of tobacco smoke is a combination of sidestream smoke (85%) and exhaled mainstream smoke (15%). Critically, sidestream smoke contains a higher concentration of toxic gaseous components than mainstream smoke. Besides cigarettes, tobacco can also be consumed through cigarillos, cigars, pipes, or water

pipes. Furthermore, 'smokeless' tobacco, used for chewing, sniffing, or placing in the mouth, is popular globally and carries significant health risks similar to smoking [6].

Classification of Smoking Patterns and Their Physio logical Mechanisms

Any product created or obtained from tobacco by burning is considered a smoked tobacco product. and the product that contains sliced, ground, powdered, or leaf tobacco and is meant to be ingested through the mouth or nose is considered smokeless tobacco [7].

There are two forms of tobacco smoke: mainstream smoke, which is absorbed directly by the smoker's mouth, and side stream smoke, often known as second-hand smoke or passive smoking, which is produced when the air around a cigarette burns. Environmental tobacco smoke is another name for secondhand smoke. It is the smoke from the end of a burning cigarette combined with the smoke exhaled by the smoker. It's commonly known as passive smoking when someone inhales this smoke [8].

In contrast to a traditional cigarette, which burns at 600°C, heated tobacco products, often referred to as heat-not-burn tobacco products, are battery-powered devices that heat tobacco to a maximum of 350°C. Tobacco producers now market these products by saying that they are less dangerous than traditional tobacco cigarettes since the tobacco leaves are simply heated rather than burnt. However, the same toxic ingredients found in traditional cigarette smoke acrolein, formaldehyde, benzaldehyde, acenaphthylene, nicotine, carbon monoxide, and particulates are also present in the common aerosol of heated tobacco. There is presently no proof that heated tobacco products are less dangerous than regular tobacco products [9].

1- Cigarette smoking:

Is the most common type of tobacco use [10]. When filter ventilation was first introduced in the middle of the 1960s, it was thought to make cigarettes safer. In contrast to other kinds of lung cancer, the rates of lung adenocarcinoma have paradoxically increased since then. Smokers can inhale more smoke to sustain their nicotine intake because filter ventilation changes the way tobacco burns, raising smoke toxicants; it also creates a false sense of lesser health risks from "lighter" smoke. Human exposure biomarker studies, which do not assess exposure in the lungs or use established biomarkers of injury, seem to be incompatible with a causal association because they show no decrease in exposure. Smoke may become accessible to lung cells that are susceptible to adenocarcinomas due to altered puffing and inhalation. Filter ventilation has likely played a role in the increase in lung adenocarcinomas among smokers, according to the report. Therefore, the FDA ought to think about controlling its use, even to the point of outright banning it [11].

2- **Roll-your-own (RYO)**:

While previously having a ‘downmarket old man image’, RYO is now widely used by younger people and females. This is partly driven by the introduction of new brands, variants and blends, pack and filter innovation, and the rolling papers available. Papers now come in different materials (e.g. rice, bamboo) and myriad colours, shapes (e.g. pre-rolled cones), flavours (e.g. peaches and cream, cognac), weights and sizes [12].

3- Shisha:

Commonly referred to as hookah or waterpipe, is a smoking apparatus designed to deliver a smooth and flavorful experience. It involves heating specially prepared tobacco, often infused with sweeteners and aromatic flavors, and passing the smoke through water before inhalation. This process not only cools the smoke but also enhances the overall enjoyment, making it a centerpiece for gatherings and relaxation [13].

Shisha smoking was classified as light (1 head per sitting), moderate (2 heads per sitting) or heavy (≥3 heads per sitting) [14]. Waterpipe tobacco smoking (WTS) is rising globally, particularly among young people. WTS is not a safe alternative to cigarettes. Its health effects are similar to or worse than those of cigarettes [15].

The global rate of smoking hookah is increasing due to different factors such as misconceptions about its lack of health risks. This might also be due to its social acceptance, availability of different tastes, and it's relatively low cost. The previous studies suggested that different viewpoints regarding lower negative effects of hookah compared to cigarette, easy access, and low cost influenced the prevalence of hookah smoking [16].

A typical hookah session of about 45 minutes can result in exposure equivalent to smoking approximately 15 cigarettes. This level of exposure makes it possible to develop nicotine dependence after only a few sessions, compared to cigarette smoking, hookah use delivers higher amounts of toxic substances. Research indicates that during one session, a hookah smoker may inhale up to eight times more carbon monoxide and 36 times more tar than a cigarette smoker. These harmful substances increase the risk of developing serious health problems, including cancer, cardiovascular diseases, and chronic respiratory conditions. Hookah use also poses additional health risks. Sharing a waterpipe can contribute to the spread of infectious diseases such as colds, influenza, and herpes. Moreover, regular users are more likely to experience oral health problems such as gum disease and tooth loss. For pregnant women, hookah smoking can increase the risk of delivering babies with low birth weight, which may lead to long-term health complications. In both the short and long term, hookah smoking has harmful effects. Acutely, it causes increased heart rate, elevated blood pressure, reduced lung function, and potential

carbon monoxide intoxication. Prolonged use is associated with chronic bronchitis, emphysema, coronary artery disease, and increased risks of lung, stomach, and esophageal cancers. Overall, hookah smoking is a serious threat to both oral and general health [17].

4- Electronic (e)-cigarettes:

Invented in their current form by Chinese pharmacist Hon Lik in the early 2000s. The US patent application describes the e-cigarette device as "an electronic atomization cigarette that functions as substitutes for quitting smoking and cigarette substitutes [18].

E-cigarettes are relatively new product on the market, is an electronic device of variable design that heats up and aerosolizes e-liquids that most often contain nicotine, propylene glycol, glycerin and various flavorant additives. These devices are widely popular, with approximately 9% of the total U.S. population vaping and 16-28% of teenagers and young adult's vaping. The chemical profiles of e-cigarette aerosols are almost entirely different from cigarette smoke, with nicotine being one of the only chemicals consistently found in both inhalants. E-cigarette aerosols generally contain fewer toxic chemicals than traditional cigarette smoke, but have been found to contain harmful substances including heavy met als, volatile organic compounds and cancer-causing chemicals. The short-term (weeks to months), effects of e-cigarette use, such as e-cigarette or vaping product associated lung injury (EVALI), are becoming more apparent as use of these devices with both nicotine and tetrahydro cannabinoids (THCs) escalates worldwide. However, the long-term (decades) effects of e-cigarette use are still unknown [19].

Uncertainties remain regarding their long-term safety. To date, making it impossible to state with certainty that e-cigarettes are safer than traditional cigarettes [20].

Primary evidence indicates that e-cigarettes may exert harmful cardiovascular effects, primarily by inducing sympathetic nerve activation, oxidative stress, endothelial dysfunction, and platelet activation [21].

The respiratory system is also affected. The airway mucosa normally serves as a protective barrier against microbial invasion, but exposure to e-cigarette vapor has been shown to reduce epithelial mucociliary function. Emerging evidence further suggests that vaping may increase susceptibility to respiratory infection by enhancing microbial ability to invade host cells [22].

Oral health risks have also been documented. The vapor produced by e-cigarettes contains a mixture of chemicals and toxins that may contribute to gum inflammation, dry mouth, and a higher risk of dental caries. Additionally, the repetitive act of vaping can irritate the oral mucosa and throat. Nicotine itself, commonly present in e-cigarettes, acts as a vasoconstrictor, restricting blood flow and thereby reducing oxygen and nutrient delivery to oral tissues, which may compromise gum health [23].

The rapid evolution of e-cigarette devices, their patterns of use, and the diversity of available e-liquids complicate research; nevertheless, certain facts are established. Modern devices deliver nicotine at levels comparable to combustible cigarettes and sufficient to elicit strong physiological responses in both humans and experimental models. Nicotine is highly addictive and, along with its metabolites, has carcinogenic potential and adverse effects on adolescent brain development, regardless of its source [24].

Role of Oxidative Stress, Inflammation, and Antioxidant Defense in disease development

The core mechanism of smoking-induced damage is the interplay between oxidative stress (OS) and chronic inflammation. Oxidative stress refers to the excessive production of Reactive Oxygen Species (ROS) or free radicals in cells and tissues that overwhelms the body's endogenous antioxidant defenses [25]. Free radicals are oxygen-containing molecules with an uneven number of electrons, allowing them to react easily with other molecules, initiating large chain chemical reactions known as oxidation [26]. An imbalance in this protective mechanism can result in the damage of crucial cellular molecules, including Deoxyribonucleic Acid (DNA), proteins, and lipids, potentially leading to various health issues [25]. The dual nature of free radicals means that while their physiological production is vital for normal cellular functions like immune response and cell signaling [27], the imbalance caused by factors like smoking and environmental toxins results in OS [28]. The body utilizes endogenous antioxidants, but it relies on external (exogenous) sources, primarily the diet (fruits, vegetables, grains), to obtain the remaining necessary antioxidants [29]. Compounding this effect, cigarette smoke has marked proinflammatory effects that immediately trigger an inflammatory response both locally in the lungs and systemically throughout the body. Inflammation is the immune system's protective response to stimuli such as infections, injuries, and burns, involving an influx of white blood cells to fight infection and heal wounds. While acute inflammation is short-term, chronic inflammation is a long-term condition that can inadvertently attack the body or persist unresolved, causing significant secondary damage. The chemicals present in cigarette smoke interact directly with various immune cell types, increasing their accumulation at inflammation sites and altering the levels of cytokines and other biological molecules that regulate inflammation [30].

The respiratory epithelium constitutes the first line of defence against inhaled pollutants and pathogens. Cigarette smoke can directly damage the airway epithelial barrier, including ciliated cells, goblet cells, basal cells, and submucosal secretory glands. Toxic substances in cigarette smoke, as well as nicotine containing e-cigarettes, can impair the continuity of ciliary oscillation, and lead to mucus hypersecretion, delayed mucociliary clearance, which is conducive to the colonization by and the reproduction of pathogens [31].

Smoking has complicated effects on blood pressure (BP). By activating the sympathetic nervous system, smoking a single cigarette causes an immediate and temporary rise in blood pressure. The average brief rise in systolic blood pressure. When smoking persists, blood pressure stays high. It's uncertain how smoking affects blood pressure over the long term. However, it is known that current smokers with normal to high-normal blood pressure (120–139/75–89 mmHg) are more likely to develop cardiovascular disease (CVD) than nonsmokers whose blood pressure was about 20 mmHg. Also, Nicotine causes short-term increases in energy expenditure, smokers tend to weigh less than nonsmokers initially. However, over time, smoking increases insulin resistance and is associated with central fat accumulation, leading to the development of metabolic syndrome (MetS) and diabetes mellitus (DM) [9].

Smoking disrupts the ovulation process. The toxic ingredients in cigarettes reduce the levels of the female hormone's progesterone and estrogens. As a result, fewer fully developed eggs are accessible for fertilization. Additionally, it can reduce the number of eggs in the ovaries and kill them. Even if smoking does not destroy the eggs, it can reduce their quality and cause erratic development after fertilization. Furthermore, smoking can damage eggs' genetic content, increasing the risk of miscarriages and birth defects [32].

Only 15–20 % of heavy smokers acquire lung cancer, despite smoking being the leading risk factor for cancer mortality [33]. By boosting angiogenesis, preventing apoptosis, and increasing the likelihood of mutations, chronic inflammation raises the risk of lung cancer. After acute and subacute stages, inflammation brought on by irritants or infections typically resolves. But occasionally, this resolution stage is missed, leaving the lungs inflamed for a long period, which might cause cancer. several cell activation mechanisms that cause cell proliferation and malignant transformation involve several proteins [34].

Smokers are twice as likely to get TB disease, and those who have TB are twice as likely to die while receiving treatment [35]. Human innate and adaptive immunity are both impacted by smoking, which reduces defensive immunity. Smokers appear to be more susceptible to extra-pulmonary tuberculosis because of this. However, within six weeks of quitting smoking, the detrimental effects of cigarette smoking on the immune system go away. Research shows that smoking is very common among people with tuberculosis. Smoking cigarettes raises the chance of contracting Mycobacterium tuberculosis and the likelihood that an infected person would acquire tuberculosis illness. In a similar vein, secondhand smoke exposure, or passive smoking, increases the risk of contracting mycobacterium tuberculosis and getting TB illness [36].

The prevalence of smoking-related asthma seems to be comparable to that of asthma in the general population. Active smokers, however, typically have more severe and challenging-to-manage asthma. Numerous investigations have shown a rapid deterioration in lung function and a poor response to inhaled corticosteroids (ICS). Lastly, people with asthma appear to have a higher prevalence of nicotine dependence, making quitting smoking more challenging. Compared to asthma patients who do not smoke those who actively smoke have a higher risk of developing an obstructive ventilatory condition. A cluster of smokers with asthma is caused by nicotine addiction [37].

It is not at all surprising that the combined harmful effects of elevated blood glucose with cigarette smoking accelerates vascular damage in people with diabetes who smoke. It is widely accepted that cigarette smoking substantially increases the risk of micro and macrovascular complications in patients with type 2 DM (T2DM). This risk is significantly decreased by quitting smoking. Reducing cigarette smoke exposure is crucial for public health, but most clinical guidelines emphasize that it is even more important for individuals with diabetes mellitus [38].

Factors that Affect the Severity of Smoking Effects

The risk and severity of smoking-related illnesses and fatalities are strongly correlated with key behavioral variables, notably the number of cigarettes smoked daily and the duration of smoking (smoking years). Research indicates that smoking intensity (the amount consumed) is itself modulated by several characteristics, including age, sex, and smoking type. Quantitatively, a substantial sex difference was observed: male smokers were found to consume 1.196 times as many cigarettes per day as female smokers. Furthermore, intensity shows a positive correlation with age, with a 5% rise in cigarette consumption noted for every decade of life. Beyond these demographic and behavioral variables, other crucial factors such as genetic elements, socioeconomic status, and the individual's Health-Related Quality of Life (HRQoL) also play a substantial role in determining the overall severity of smoking's health impact [39-41].

Laboratory Markers for Assessing the Biological Im pact of Smoking

1- Cotinine Test:

The best measure of exposure to tobacco smoke is cotinine. Cotinine levels in blood, saliva, and urine can be measured using a variety of techniques, including gas chromatography, colorimetric assay, high performance liquid chromatography, and the NicAlert saliva test. Urine has four to six times the otinine concentrations of blood or saliva. This increases the validity and dependability of quantitative techniques that detect urine cotinine, such as gas chromatography/mass spectrometry, high performance liquid chromatography, colorimetric tests, and immunoassays. Recently developed semi-quantitative solutions have addressed the drawbacks of increased cost and time consumption [42].

Analyzing the levels of a smoke component in an exposed person's bodily fluids (a biologic marker or biomarker) would be the best way to determine their exposure to tobacco smoke. The main proximate metabolite of nicotine, cotinine, has been used extensively as a biomarker of tobacco exposure. The concentrations of otinine in nonsmokers' plasma, urine, and saliva have been used to gauge population exposure to ETS in order to calculate the risk of lung cancer associated with ETS exposure [43]. The amount of nicotine in the urine peaks due to its fast absorption through the lungs. However, very little nicotine is excreted unaltered, instead, the majority of nicotine is excreted as its primary metabolite, cotinine. There was a high correlation between the number of cigarettes smoked and the urine cotinine, however the peak excretion of cotinine occurs approximately two hours after smoking and its clearance is delayed. ratio of creatinine [44].

2- Nicotine Test:

Cigarette nicotine is quickly absorbed. After first entering the venous circulation and then the arterial absorption circulation, nicotine levels thereafter decrease as it is transferred from the bloodstream to different body tissues. Nicotine levels thereafter decrease, with an elimination half-life of roughly two hours. Measuring and adding up all of the metabolites in the urine is one method of measuring nicotine intake. The total of all metabolites in a 24-hour urine excretion at steady state (where drug intake and metabolism production rates are equal to drug and metabolite removal rates) indicates the daily intake of nicotine. Nicotine consumption from tobacco can be measured using four broad methods: (1) Measure blood nicotine levels in smokers while they smoke cigarettes in a circadian manner. Blood levels while smoking can be converted to an absolute daily dose of nicotine if the clearance of nicotine is also assessed by intravenous infusion. (2) After a person has smoked one or two cigarettes, blood level data can be used in the same way. (3) A common method of estimating nicotine intake is to measure blood cotinine levels during ad libitum cigarette smoking. (4) Lastly, nicotine consumption can be estimated by detecting urine nicotine and metabolites during ad libitum smoking [43].

3- Carboxyhemoglobin (COHb) Test

The incomplete combustion of hydrocarbons produces the poisonous, colorless, and odorless gas known as carbon monoxide (CO). It has a 250-fold higher affinity for hemoglobin than for oxygen. After CO breaks the oxygen–hemoglobin connection, it attaches to this molecule irreversibly to produce carboxyhemoglobin (COHb). Burning fossil fuels, biomass, and tobacco produces 60% of the carbon dioxide (CO) in our atmosphere, with the remaining 40% coming from natural sources. CO inhalation can have serious negative health effects. It results in poisoning, and its clinical signs might range from vague symptoms to death. It is generally agreed that in non-smokers, the percentage of COHb in blood should be lower than 2%, while in smokers it can reach up to 10%.3–7 High levels of COHb may frequently be attributed to tobacco use, but another common cause is inhalation of CO in the home or the workplace [45].

Since the amount of carbon monoxide (CO) in tobacco smoke is constant, blood COHb levels in smokers provide a reliable and objective indicator of how much of the gaseous phase of tobacco smoke enters the lungs. In the majority of the previously mentioned studies, blood COHb levels were used as a proxy for tobacco smoke exposure [46].

4- Complete Blood Count (CBC):

Continuous cigarette smoking raises erythrocyte count, hemoglobin concentration, hematocrit, leukocyte count, mean corpuscular volume, and mean corpuscular hemoglobin concentration. The phenomenon of cell movement from other lymphoid organs in the peripheral blood may be associated with the higher number of leukocytes in the peripheral blood of healthy smokers, or smoking may reduce these cells' ability to adhere to blood vessel endothelium cells, resulting in an overall increase in the number of blood cells [47].

Nicotine's function is to promote hormone release, which raises the leukocyte count. Smoke's irritant effects on the respiratory system cause inflammation and cytokine production, which affect the leukocyte count. Numerous additional hematological parameters, such as hemoglobin concentration (Hb), packed cell volume (PCV), red cell count (RBC), and mean corpuscular volume (MCV), are higher in smokers than in nonsmokers. In healthy smokers, an increase in erythrocyte mass results in an increase in packed cell volume [48].

Quitting smoking

The smoker's motivation is the fundamental prerequisite for any successful attempt to quit. Modern

smoking cessation strategies, which are particularly beneficial for the elderly, achieve the highest abstinence rates when combining behavioral or cognitive counseling with pharmacological aids. Approved medications include Nicotine Replacement Therapy (NRT), often delivered most effectively via transdermal patches, as well as non-nicotine options like the antidepressant bupropion (which can be used alone or with NRT), and selective nicotinic partial agonists. The effectiveness, side effects, and proper use of these treatments have been extensively validated, even among older populations [49].

Strong evidence confirms that systemic inflammation and oxidative stress are present even in seemingly healthy smokers. Fortunately, quitting yields rapid biological benefits: stopping smoking for just two weeks is enough to increase muscular fatigue resistance and return inflammatory status almost to normal levels. These immediate advantages are crucial for preventing the worsening consequences of low-grade systemic inflammation and significantly support ongoing cessation efforts [50].

Smoking cessation requires trying various strategies, including Cold Turkey (abrupt cessation without aid), Nicotine Replacement Therapy (NRT) via products like gum, patches, lozenges, sprays, and inhalers, prescription medications such as bupropion and varenicline, and essential Cognitive-Behavioral Therapy (CBT) counseling strategies [51].

The health benefits observed after quitting smoking may be partially attributed to a rapid, modest increase in bilirubin concentrations, even while remaining within the normal reference range. Longitudinal studies confirm that smoking cessation directly leads to these increases. Since bilirubin acts as a potent endogenous antioxidant, this early rise may contribute significantly to some of the immediate health advantages experienced when a person stops smoking [52].

DISCUSSION

In the discussion section, we will address common misconceptions and key points of contention regarding the toxicity of cigarette smoke or unclear to society including: The result of filter ventilation on smoking behavior and health risks, are e-cigarettes actually less harmful, how dangerous is passive smoking to non-smokers, and the potential effects after quitting smoking?

Filter ventilation was adopted in the mid-1960s and was initially equated with making a cigarette safer. Since then, lung adenocarcinoma rates paradoxically increased relative to other that changing cigarette designs have caused an increase in lung adenocarcinomas, implicating cigarette filter ventilation that lowers smoking machine tar yields, this was published in 2017 by Song and et al [11]. In the other resacrch, Carroll et al in 2021 [53], A convenience sample of 114 cigarette varieties, representing sub-brands of 11 well-known cigarette brands, were bought from retail establishments in Minneapolis, Minnesota, between 2015 and 2016 in order to investigate the connections between cigarette filter ventilation levels, biomarkers of exposure (BOE), and potential harm (BOPH). Three packs of each cigarette flavor were bought from various merchants, one pack per retailer, in order to get representative average filter ventilation values. To get triplicate data for each cigarette kind, one cigarette was randomly removed from each pack and examined independently. It was concluted that Filter ventilation was not associated with biomarkers of exposure (BOE) or potential harm (BOPH), yet smokers of higher ventilated cigarettes perceived their brand as less harmful than other brands compared with smokers of lower ventilated cigarettes.

On the other hand, A study was conducted on adults 25+ years of age who smoked daily, this observational study found no strong evidence of a causal effect of Filter Ventlation on past 30-day smoking at approximately 1 and 3 years follow-up [54]. While the original intent behind introducing filter ventilation was to make cigarettes seem safer, the fundamental danger lies in the presence of tobacco and the thousands of toxic compounds it produces when burned. Even though filters can decrease the quantity of tar and other materials measured by machines, this does not eliminate the risk. The most significant issue is that these filters encourage smokers to inhale more deeply to get their desired nicotine dose. This compensatory behavior delivers the harmful toxins further into the lungs. Furthermore, since tar is cumulative, even small doses can cause significant long-term damage. Therefore, the scientific community has not reached a consensus on the 'safety' of these filters, because their impact is directly tied to a smoker's individual behavior and the duration of their habit, not to any inherent safety feature of the filter itself.

Electronic nicotine dispensing systems (ENDS), also referred to as electronic cigarettes or e-cigarettes, have been widely regarded as a less harmful alternative to traditional cigarette smoking since they first hit the market more than ten years ago. Tobacco companies frequently promoted e-cigarettes as a smoking cessation tool when they first entered the global market in 2004. The results of toxicological analyses indicate that e-cigarettes may be safer than traditional cigarettes. There are commercially available e-liquids with varying amounts of nicotine, the primary addictive ingredient in tobacco, and even e-liquids without nicotine. For this reason, e-cigarettes are frequently seen as a smoking cessation aid. Since e-cigarettes do not burn, and since most of the harmful and well-known effects of tobacco come from this reaction, there is a widespread and widespread belief that using e-cigarettes, or "vaping," is safer than smoking traditional cigarettes. According to a

2021 study by Marquees *et al.,* [55] e-cigarettes appear to be less harmful than smoking tobacco.

While Wang and colleagues (2021) [56], the researchers analyzed the association between adult e-cigarette usage and quitting smoking by examining data from 64 studies, it was found that there is no significant correlation between e-cigarette use as a consumer product and quitting smoking in the overall adult population. If the dangers are equal to the benefits, e-cigarettes would be worth considering as a prescription medication to be used as part of a clinically supervised smoking cessation effort. On the other hand, Glantz and Bareham in 2018 [57] adopted a different strategy, incorporating all 20 available studies with a suitable control group that provided a quantitative estimate of the relationship between e-cigarette use and quitting smoking (2 clinical trials, 15 cohort studies, and 3 cross-sectional studies as of April 2015). Additionally, they provided a comprehensive analysis of all 38 published research, regardless of whether they contained the data required to calculate the impact of e-cigarette use on quitting smoking. Their findings showed that e-cigarettes are contributing to the tobacco epidemic by drawing smokers who are interested in quitting but decreasing the likelihood that those smokers will successfully quit. Those who used e-cigarettes had a 28% lower chance of quitting cigarettes than those who did not. The fact that more cigarettes were consumed in the US in 2015 than in 2014 and that more people were smoking cigarettes for the first time since 1973 may be evidence of this effect.

Although electronic cigarettes were presented to the market as a less harmful substitute to conventional cigarettes, argue that they cannot be considered a safe or effective tool for smoking cessation. On the contrary, they may contribute to the beginning of smoking amongst non-smokers, particularly adolescents and young adults, due to the wide variation of appealing flavors that make these products more attractive to individuals with no prior history of tobacco routine. The claim that e-cigarettes are an effective aid for quitting smoking remains controversial; several studies indicate that many users fail to achieve complete nicotine abstinence and instead engage in double use of both conventional and e-cigarettes, which increases rather than reduces health risks. Additionally, nicotine, the main addictive ingredient in tobacco, is delivered via e-cigarettes. which means that switching to them does not free smokers from dependence but instead perpetuates addiction in another form. It is also important to highlight that the long-term consequences of e-cigarette usage on health, specifically on the respiratory and cardiovascular systems, remain insufficiently understood, raising significant concerns about their future safety profile. For these reasons, reliance on e-cigarettes as a public health strategy for smoking stop should be viewed with caution, as their use carries substantial risks and uncertain outcomes.

Indicates short-term exposure to passive smoking reduces oxygen consumption in peripheral tissues and has detrimental effects on Secondhand smoking causes about 1.2 million deaths (SHS). Active smoking is a choice, however passive exposure to SHS happens and can also have an impact on non-smokers. SHS exposure is the third most common preventable cause of mortality worldwide and presents serious health risks to people. Passive smoking is a serious risk factor for numerous health morbidities, just like active smoking. SHS has been linked in a number of studies to dementia, cognitive impairment, and other neurodegenerative illnesses. In addition to causing severe disease, exposure to SHS has major negative social and economic effects. In healthy nonsmokers, burden and icrovascular reactivity after ischemic occlusion This is consistent with the findings of Ünver and colleagues (2023) [58] in their study where they used a stratified two-stage cluster sampling technique based on data from the Turkey Health Survey carried out by TURKSTAT in 2016 and 2019. that covered all rural and urban areas in Türkiye. The study focused on older adults aged 60 years and above, with 3,657 participants in 2016 and 3,595 participants in 2019. Data were collected through face-to-face interviews, and statistical analyses were performed using Stata 15, taking into account the complex sampling design and applying sampling weights to ensure population representativeness. To examine the factors associated with tobacco exposure among older adults, a generalized ordered logistic regression model was employed, on the other hand, Jorge and colleagues (2016) [59] confirmed that the results of their study supported the association between learning difficulties in students exposed to both active and passive maternal smoking. They highlighted that parental smoking was linked to a higher occurrence of children's linguistic deficits, behavioral issues, and learning challenges. In a similar vein, learning disabilities arising from neurobehavioral problems linked to passive smoking were more common in the United States, according to lading the prenatal and postnatal periods for the mother, may have detrimental effects on the child's learning development.

From this, it can be stated that passive smoking causes health problems Like those brought on by active smoking, with the severity of these effects depending on the duration and intensity of exposure, as well as individual susceptibility and coexisting risk factors like age, overall health status, as well as genetic susceptibility. The more time spent with secondhand smoke, the higher the likelihood of developing cardiovascular and respiratory diseases, neurological impairments, and negative effects on the immune system.

When quitting smoking, most people gain weight, however the amount varies widely. In study conducted by Pankova and colleagues clarified 2018 [60] that Excess weight gain during the first few months of

abstinence is a poor indicator of later and excessive growth in persons who have quit smoking for a year, and there don't seem to be any other reliable indicators of excessive weight gain that are assessed at a standard smoking cessation clinic. According to the writers, It's unclear exactly what mechanisms lead to weight increase. It has been proposed that the majority of quitters have PCWG as a result of an imbalance where energy expenditure (such as resting metabolic rate) and energy intake (such as caloric intake) are out of balance. However, the cause of this imbalance is still unknown. Nicotine withdrawal is one explanation. Nicotine lowers food intake and raises basal metabolic rate, despite the fact that it lacks an anorectic effect and does not immediately decrease appetite or eating. while Sahle and colleagues in 2021 [61], more than 16,000 Australian adults were followed between 2006 and 2014. The researchers tracked smoking status, weight changes, and health outcomes, and used Cox regression models to examine the researchers found that quitting smoking was significantly connected with weight and Body Mass Index (BMI) gains when compared to continuing to smoke. They also found a correlation between post-cessation weight gain and the risk of chronic diseases and mortality. Regardless of the amount of weight and BMI gain, persons who quit smoking had a much reduced chance of dying than those who kept smoking. Gaining weight or BMI after quitting smoking was not linked to a higher risk of cancer, type 2 diabetes, cardiovascular disease (CVD), or chronic obstructive pulmonary disease (COPD). According to the results, quitting smoking has more advantages than disadvantages in terms of lowering overall population mortality without raising the chance of developing the main chronic illnesses.

Janzon and colleagues (2004) [62] followed a cohort of women to examine changes in blood pressure and body weight after smoking cessation. Participants' smoking status, blood pressure and body, weight were measured at baseline and during follow-up visits. The researchers analyzed how quitting smoking affected these physiological parameters over time, then they found that Weight gain with quitting smoking is a well-known occurrence. Women who smoke frequently use tobacco to control their weight, thus fear of gaining weight is a big barrier to quitting. Numerous theories have been put up to explain the connection between smoking and a reduced body weight. Nicotine appears to have an impact on the hypothalamus' monoaminergic control of food intake. According to a lengthy follow-up, women who quit smoking acquired about 3 kg more weight than those who never smoked, suggesting that smokers have a greater metabolic rate, which may be a factor in weight gain after quitting.

Based on the above, it can be concluded that post-cessation weight gain is a common phenomenon; however, its extent varies among individuals and is often overestimated. Nevertheless, concerns about such weight gain should not overshadow the well-established health benefits of smoking cessation, especially since According to research, gaining weight after quitting is not linked to a higher risk of dying or developing chronic illnesses. Fear of weight gain represents a psychological barrier that can be controlled, as potential increases in weight can be managed through adopting a healthy diet and engaging in regular physical activity. Therefore, smokers should not allow the possibility of weight gain after quitting preventing them from making the decision to stop, which may deprive them of the significant health benefits of cessation. Although several explanations have been proposed, such as nicotine's influence on metabolic rate, the exact mechanisms underlying post-cessation weight gain remain insufficiently understood and warrant further investigation.

CONCLUSION

This review unequivocally confirms that cigarette smoking remains a global public health crisis, driven by a fundamental and destructive biological pathology: the synergistic interplay between oxidative stress (OS) and chronic inflammation. The chemical complexity of tobacco smoke, which features over 7,000 constituents, including a higher concentration of toxic gases in the sidestream smoke component, overwhelms the body's endogenous antioxidant and immune defenses. This overload results in excessive production of Reactive Oxygen Species (ROS), causing severe damage to vital cellular molecules such as DNA and proteins. This pathological state immediately triggers a systemic proinflammatory response, establishing the foundation for smoking's broad spectrum of associated diseases, including lung cancer, cardiovascular disease (CVD), COPD, and Type 2 Diabetes.

A critical analysis of common public health misconceptions reveals several key challenges:

Filter Ventilation: The introduction of filter ventilation, initially perceived as a safety measure, paradoxically contributed to an increase in lung adenocarcinoma rates. This is primarily due to the filter-induced compensatory smoking behavior—deeper inhalation to maintain nicotine intake—which delivers toxins further into susceptible lung tissue.

Novel Nicotine Products: Despite being marketed as "reduced harm" alternatives or cessation aids, products like electronic cigarettes (e-cigarettes) and heated tobacco products still deliver highly addictive nicotine and contain harmful substances, including heavy metals and volatile organic compounds. The lack of long-term safety data remains a major concern. Furthermore, significant evidence suggests that e-cigarette use as a consumer product may impede successful cessation, leading many users to engage in risky dual use.

Secondhand Smoke (SHS): Passive exposure to SHS is a serious health threat to non-smokers, representing the third most common preventable cause

of mortality globally. SHS exposure is directly linked to a wide range of morbidities, including respiratory illness, cardiovascular damage, and neurological impairments, particularly learning difficulties in children.

Crucially, the decision to quit smoking offers immediate and profound biological benefits. Abstinence for as little as two weeks is sufficient to increase muscle fatigue resistance and return inflammatory status close to normal levels. While post-cessation weight gain is a common phenomenon, it is often overestimated. Research strongly indicates that weight gain after quitting is not associated with an increased risk of all-cause mortality or major chronic diseases (CVD, COPD, T2DM, or cancer). Therefore, the fear of potential weight gain should not be a psychological barrier that prevents smokers from making a life-saving decision.

Recommendations

1- Increase public knowledge of the risks of smoking and the negative impacts it has on people's health and society, including both active and passive smoking.
2- Encourage a healthy lifestyle by reducing oxidative stressors, maintaining a balanced diet, and engaging in frequent physical activity.
3- Support smokers to quit using various medical and behavioral strategies, such as pharmacological treatments, psychological counseling, and group support programs.
4- Integrate health education programs in schools, universities, and local communities to reduce smoking prevalence among youth.
5- Encourage research studies on the connection between smoking and several illnesses.
6- Highlight the short-term and long-term health advantages of quitting smoking to encourage people to do so without worrying about weight gain or other issues.
7- Enact stringent legislation that restricts smoking in public areas and promotes the decrease in tobacco use and manufacturing.

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