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The Interplay Of Cardiovascular Health, Lung Function, And Cancer Risk: A Population-Based Study

Dr. B Shyam Prasad Shetty[1*], Dr Sandesh KS[2], Dr Sunil Kumar S[3]

[1*]Assistant Professor, Dept of Cardiothoracic and Vascular Surgery, JSS Medical College, Mysuru
[2]Head Department of PG Studies, Nehru Memorial College Sullia
[3]Assistant Professor of Cardiology, JSS Medical College, Mysuru

Abstract
Cardiovascular diseases (CVD), lung function disorders, and cancer are leading global health issues, contributing significantly to morbidity, mortality, and healthcare burdens worldwide. This review explores the intricate interplay between these conditions, focusing on shared risk factors, biological mechanisms, and their compounded effects on cancer susceptibility. CVD, lung dysfunction, and cancer often share common risk factors such as smoking, obesity, sedentary lifestyle, and poor diet, which exacerbate their progression and contribute to mutual exacerbation. Chronic inflammation, oxidative stress, and metabolic dysfunction are key pathophysiological mechanisms linking these diseases, leading to increased cancer risk. Epidemiological and cohort studies have shown that individuals with impaired lung function and cardiovascular disease are at significantly higher risk for developing various cancers, including lung, colorectal, and liver cancers. Despite growing evidence, there remains a lack of understanding regarding the molecular, genetic, and environmental interactions that drive the synergistic relationship between these conditions. Future research should focus on longitudinal cohort studies and experimental investigations to clarify the causal pathways linking cardiovascular health, lung function, and cancer. Furthermore, integrated healthcare strategies that simultaneously address CVD, lung disease, and cancer are crucial in preventing and managing these interconnected diseases. Public health policies targeting early screening, lifestyle interventions, and environmental health are essential to reducing the burden of these diseases. A comprehensive approach, combining prevention, early detection, and interdisciplinary care, is vital to improving global health outcomes.

Keywords: Cardiovascular diseases, Lung function, Cancer risk, Inflammation, Oxidative stress, Shared risk factors, Epidemiological studies.

1. Introduction

Cardiovascular diseases (CVD), lung function disorders, and cancer are the leading global health issues, and together they are responsible for a significant part of the disease burden, disability, and mortality of the world. CVD stands as the number one cause of death in the world, and the WHO estimates that 17.8 million people die from CVD annually [1]. At the same time, millions of deaths occur annually due to respiratory diseases, such as Chronic Obstructive Pulmonary Disease (COPD), asthma, and several other incapacitating respiratory disorders, which fail to achieve their projected goals in terms of both quality and quantity of lives of an individual affected. As mentioned, lung cancer is the most common and deadliest form of cancer worldwide, with a leading cause of morbidity and mortality, along with other cancers, a diverse group of diseases involving uncontrolled cell growth [2]. These conditions have a clear consequence for public health, beyond their high individual mortality rates, as they share risk factors and compounding effects on patients' overall well-being.

Research on the relationship between cardiovascular health, lung function, and cancer risk is increasing interest and concern. Although diseases have been studied individually in terms of their risk, no work has been done to explore the complex relationships between them [3]. CVD and lung disorders are so common that they often have many risk factors in common, including smoking,

obesity, sedentary life, and poor diet [4]. For instance, people with poor lung function are more prone to have the outcome of cardiac disease. In addition, it has been noted that the occurrence of one disease increases the risk of developing the other, especially in the case of chronic diseases such as COPD and hypertension [5]. In addition, patients with cardiovascular as well as respiratory diseases are more likely to develop cancer, especially lung cancer, and other cancers.

The causes of the interaction of cardiovascular health, lung function, and cancer risk are complex and multifactorial. These diseases are linked by one of the major biological processes and chronic inflammation. Inflammation is a crucial component in the development and progression of both cardiovascular and lung diseases, as well as cancer metastasis [6]. Endothelial dysfunction, a marker in heart disease, also plays a role in lung injury and the propagation of respiratory diseases. It may also result in an increased risk of cancer, using altered immune responses and promotion of tumor growth [7]. Similarly, in both cardiovascular and lung diseases, oxidative stress is also increased and may contribute to DNA damage, mutations, and increase the risk for cancer development. The metabolic disturbances commonly seen with CVD, like insulin resistance and dyslipidemia, can contribute to such an environment that helps with cancer cell growth [8]. The fact that there are shared biological pathways suggests we should look at the collective influence of cardiovascular health on lung function and lung function on the risk of developing cancer. These shared biological pathways underscore the importance of examining the collective impact of cardiovascular health and lung function on cancer risk.

Environmental elements play a role in developing both eating disorders and depression, along with anxiety disorders. An example of a major risk factor for lung disease, cardiovascular disease, and cancer, especially lung cancer, is smoking. Another environmental factor that has been linked to negatively affecting both lung and cardiovascular health, as well as raising the risk of cancer, is air pollution [9]. The prevalence of these risk factors is so high in both developed and developing countries, which makes it extremely difficult to control these diseases. Such population demographics (high incidence of smoking, sedentary behavior, and poor dietary habits) have resulted in a complex network of risk factors for cardiovascular health, lung function, and cancer susceptibility [10]. Cumulative and synergistic effects among these environmental factors on public health constitute a major obstacle to developing effective prevention and intervention strategies.

Despite growing evidence of the individual and collective risks of these diseases, there is a clear lack of literature regarding how these diseases are caused to affect cardiovascular health, lung function, and cancer. There have been many studies on the risk of these diseases, but there is much less understanding of how they interact. How cardiovascular treatments affect cancer risk is not known since some cardiovascular drugs might protect against cancer while others might increase cancer progression [11]. Lung diseases like COPD are known to be risk factors for CVD, but lung function in cancer development is not as well defined. In addition, few large-scale, population studies examine how all these conditions interact over time and whether one condition influences each other's progression. The molecular and genetic bases of these interactions have not been fully characterized, and the role of common inflammatory pathways and immune dysfunction has not been defined [12].

This review aims to provide a comprehensive analysis of the interaction between cardiovascular health, lung function, and cancer risk. Its objective is to identify common biological mechanisms and risk factors shared between these diseases, understand the range of environmental risk factors including smoking and air pollution about both diseases, and assess how the two diseases synergistically predispose people.

2. The Interconnection Between Cardiovascular Health and Cancer Risk

2.1 Mechanisms Linking Cardiovascular Diseases (CVD) to Cancer

Several biological mechanisms common to CVD and cancer include inflammation, oxidative stress, and metabolic derangement. CVD often has chronic inflammation that enhances angiogenesis and immune evasion [13]. In both conditions, oxidative stress leads to DNA damage and mutations and

thereby increases cancer susceptibility. It also has a major role in cancer as a pathogenic facet of CVD, both contributing to the rampant metastasis and advancement of cancer tumors by the formation of a tumorigenic microenvironment [14]. It is also known that atherosclerosis, which is plaque formation in arteries, can promote vascular inflammation and oxidative stress that might accelerate tumor development. One of the reasons for this is the fact that atherosclerotic changes increase the risk of cancers by inducing both upregulation of pro-inflammatory cytokines and growth factors [15]. These shared pathways describe the links between cardiovascular disease and cancer and thus represent an interface where the cure of one disease may affect the other. The role of oxidative stress in cardiovascular failure is illustrated in Figure 1.

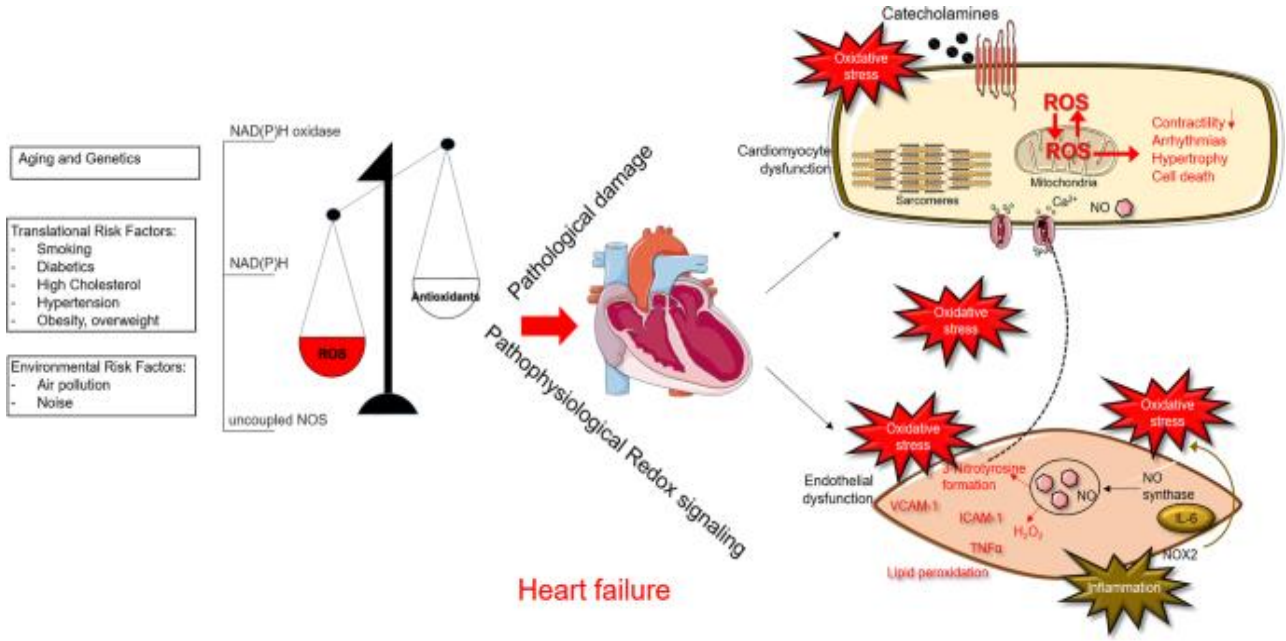

Figure 1: The Role of Oxidative Stress and Redox Signaling in Heart Failure Pathophysiology
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2.2 Epidemiological Evidence

Research from epidemiological investigations reveals that CVD enhances the susceptibility to multiple cancer types. Previous studies involving a broad patient cohort demonstrated that cardiovascular disease patients face increased cancer risks, especially for lung, colorectal, and breast cancers, as these conditions share chronic inflammation and oxidative stress pathophysiological elements [16]. A meta-analysis confirmed that patients with cardiovascular disease experience substantial malignancy risk, which rises based on their cardiovascular condition severity [17].

The relationship between CVD and cancer becomes stronger through common risk elements that include hypertension, alongside obesity and diabetes. The condition of hypertension has demonstrated its ability to damage endothelial cells while supporting tumor angiogenesis. The development of cancer becomes faster, and CVD worsens due to obesity-induced inflammatory conditions [18]. People with diabetes who have cardiovascular conditions risk developing various cancers, such as liver and pancreatic cancers, due to metabolic dysfunction and insulin resistance [19].

2.3 Impact of Cardiovascular Treatments on Cancer Risk

Statins, together with beta-blockers and Angiotensin-Converting Enzyme (ACE) inhibitors, demonstrate conflicting effects on cancer development while affecting treatment results. The inhibition of cholesterol synthesis, together with reduced inflammation, makes statins a promising agent for cancer risk reduction in breast, prostate, and colorectal cancer cases [20]. Beta-blockers

demonstrate a protective effect against cancer development in prostate and breast cancers because of their anti-inflammatory properties [21]. ACE inhibitors, together with angiotensin II receptor blockers (ARBs), demonstrate their cancer prevention ability by managing blood pressure regulation and inflammation pathways [22]. The relationship between calcium channel blockers, along with diuretics, and cancer development remains uncertain, while these drugs remain necessary for blood pressure control and fluid management. Research indicates that aspirin demonstrates anti-inflammatory and antiplatelet abilities, which lead to reduced cancer risks, particularly with colorectal and gastric cancers [23]. These medications show benefits, but they present possible side effects, which include muscle discomfort and gastrointestinal bleeding, and kidney function modifications that practitioners need to evaluate. The impact of Cardiovascular Treatments on Cancer risk and outcomes is mentioned in Table 1.

Table 1: Impact of Cardiovascular Treatments on Cancer Risk and Outcomes: A Comprehensive Overview

Cardiovascular Treatment	Impact on Cancer Incidence	Mechanism of Action	Cancer Types Affected	Impact on Cancer Outcomes	Potential Side Effects
Statins	Potential reduction in cancer risk	Inhibition of cholesterol biosynthesis, reduction of inflammation	Breast, prostate, colon, liver	Improved survival in some cancers	Muscle pain, liver enzyme elevation
Beta-blockers	Potential cancer risk reduction in specific cancers	Reduction in heart rate and oxygen demand, anti-inflammatory effects	Prostate, breast, lung	Mixed effects on cancer prognosis	Fatigue, low blood pressure
ACE inhibitors	Mixed evidence on cancer risk reduction	Inhibition of ACE, reduction of blood pressure, and anti-inflammatory effects	Breast, lung, colon, prostate	No conclusive effect on cancer outcomes	Cough, high potassium levels
ARBs	Possible reduction in certain cancers	Inhibition of angiotensin II binding, reduction of blood pressure	Breast, colorectal	Potential improvement in survival	Dizziness, kidney function changes
Calcium channel blockers	No clear effect on cancer incidence	Relaxation of blood vessels, lowering of blood pressure	No specific cancer association	No clear impact on cancer outcomes	Swelling, dizziness
Diuretics	No clear effect on cancer incidence	Reduction of fluid volume, lowering of blood pressure	No specific cancer	No clear impact on cancer outcomes	Dehydration, electrolyte imbalance

			association		
Aspirin	Reduced risk of cancer through anti-inflammatory effects	Anti-inflammatory and antiplatelet effects	Colorectal, lung, and gastric	Potential improvement in survival, anti-cancer properties	Gastrointestinal bleeding, ulcers
Warfarin	Possible increase in certain cancers due to anticoagulation	Anticoagulation, inhibition of blood clot formation	Lung, liver, colorectal	No clear impact on cancer outcomes	Bleeding, bruising
Antiplatelet agents	Reduction in certain cancers due to antiplatelet effects	Inhibition of platelet aggregation, anti-inflammatory effects	Colon, breast, lung	Improvement in cancer survival in some cancers	Bleeding, rash

ACE-Angiotensin-Converting Enzyme; ARBs-Angiotensin II Receptor Blockers; CVD-Cardiovascular Disease; COPD-Chronic Obstructive Pulmonary Disease

3. Population-based insights into the Interplay Between Cardiovascular Health, Lung Function, and Cancer Risk

Key cohort and longitudinal investigations have delivered important data that demonstrate the effects on cancer susceptibility from cardiovascular health and the lungs. Results from the Framingham Heart Study demonstrated that people with poor lung function, including those affected by COPD, face higher odds of developing lung cancer alongside colorectal cancer and liver cancer [24]. The American Cancer Society Cancer Prevention Study found that decreased lung capacity in smokers intensifies the combined cardiovascular disease and lung dysfunction cancer risks [25]. The European Prospective Investigation into Cancer and Nutrition (EPIC) study confirmed that poor lung function elevates cancer risks in people exposed to tobacco smoke and air pollution, according to their results [26].

The decline of lung function directly impacts cancer risks, primarily toward lung cancer development, through the actions of chronic inflammation combined with genetic mutations and environmental pollution. Tissue changes toward tumor promotion stem from long-lasting inflammation and oxidative stress, which advance genetic mutations that transform lung and additional tissues into cancer cells [27]. COPD, together with asthma, creates extensive health risks by raising the probabilities of both lung cancer and different cancer types such as pancreatic and liver cancers [28]. Screening and preventive strategies should focus on people who have reduced breathing capacity and heart disease, as they face elevated cancer risks, especially when combined with smoking history and environmental pollution exposure.

Research through cohort studies demonstrates that subjects experiencing both cardiovascular disease and poor lung function develop cancer at greater rates than patients who have single health issues [29]. Healthcare integration must focus on addressing multiple diseases at once because it offers an effective approach to preventing cancer and detecting it early. The cohort studies exploring lung disease are mentioned in Table 2.

Table 2: Cohort Studies Exploring Lung Function Decline and Cancer Risk

Study	Lung Function Decline	Cancer Types Affected	Key Findings	Population	References
Framingham Heart Study	Decreased lung function	Lung, pancreatic, liver	Significant association between lung function decline and lung cancer risk	General population, long-term	[30]
American Cancer Society Cohort	Lower FEV1	Lung, colorectal, liver	Increased risk of lung cancer and gastrointestinal cancers in COPD patients	Smokers, age 50+	[31]
NHANES	Reduced lung function	Lung, colorectal, liver	Decline in lung function correlates with a higher incidence of cancer	U.S. adult population	[32]
EPIC	Decreased FEV1 and FVC	Lung, esophageal, liver	Strong correlation between impaired lung function and various cancers	European adults, long-term	[33]
Danish Cancer Registry	Chronic airflow limitation	Lung, bladder, colorectal	Increased risk of lung and bladder cancer	Danish adult population	[34]
National Health Service (UK)	Decreased FEV1 and FVC	Lung, colorectal, liver	Lung function decline is associated with higher cancer incidence	UK population, long-term	[35]
Multi-Ethnic Study of Atherosclerosis	Reduced lung function	Lung, pancreatic, colorectal	Lung function decline correlates with elevated cancer risk, especially lung and colorectal cancers.	U.S. multi-ethnic adult population	[36]
Lung Health Study	Decline in FEV1	Lung, respiratory tract, lung	Lung function decline is associated with poor lung cancer outcomes	Adults with chronic respiratory disease	[37]
Kaiser Permanente Health System	Decreased FVC	Lung, gastrointestinal	Increased incidence of cancers, especially lung and gastrointestinal cancers	U.S. adult population	[38]

FEV1-Forced Expiratory Volume in 1 second; FVC-Forced Vital Capacity; COPD-Chronic Obstructive Pulmonary Disease; NHANES-National Health and Nutrition Examination Survey; EPIC-European Prospective Investigation into Cancer and Nutrition; UK-United Kingdom; U.S.-United States

4. The Synergistic Relationship Between Cardiovascular Health and Lung Function

Many causal elements and physical processes that affect cardiovascular function also impact lung performance, leading to combined deterioration of these two systems. The decline in both cardiovascular health and lung function results from smoking, together with a sedentary lifestyle, poor diet, and obesity. Smoking creates a rapid progression of atherosclerosis and generates oxidative stress and sustained inflammation; thus, it damages vascular function, which causes COPD and lung cancer

development [39]. Elevated cholesterol, hypertension, and insulin resistance occur from combined factors of a sedentary lifestyle with poor eating habits, and these conditions substantially harm cardiovascular health, along with respiratory functioning. The common risk factors increase cancer susceptibility, as inflammation and metabolic problems created by these factors enable tumor growth and metastasis across both the lungs and the heart. The development of CVD through endothelial dysfunction produces vascular complications that raise the risks for atherosclerosis and ischemic events while simultaneously contributing to chronic respiratory diseases in the lungs [40]. A self-perpetuating mechanism thus forms because both conditions intensify each other, leading to faster disease progression and heightened cancer risks. The persistent lung inflammation, which stems from impaired breathing, results in both cardiovascular disease worsening and promotes tumor development, leading to higher lung cancer risk [41]. Heart diseases that obstruct oxygen delivery produce unfavorable effects on lung function while simultaneously deteriorating overall respiratory health. The shared risk factors of cardiovascular and lung health are illustrated in Figure 2.

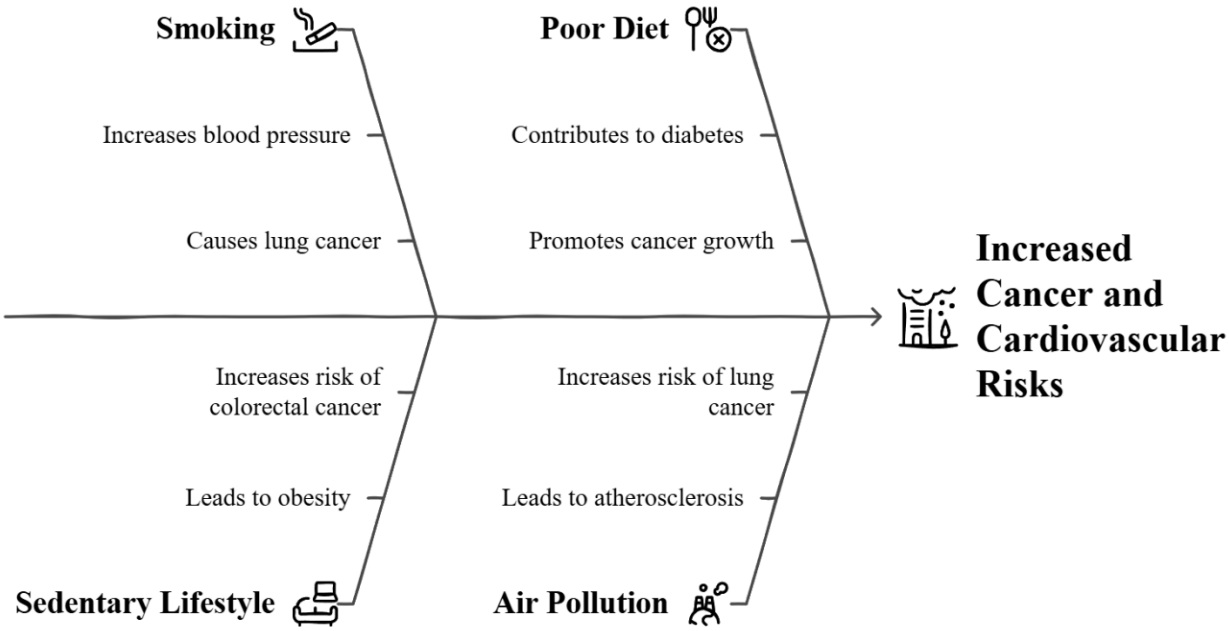

Figure 2: Shared Risk Factors and Their Impact on Cardiovascular and Lung Health

The mutual influences between cardiovascular disease and lung function work together as a single reinforcing mechanism that raises the risk of cancer development. Lung function deterioration, such as in COPD patients, leads to cardiovascular problems through systemic inflammation and blood oxygenation changes that worsen respiratory symptoms [42]. The reciprocal relationship causes disease severity to escalate since each condition speeds up the progression of the other. Such conditions combine to boost cancer risk most notably among patients who suffer from both CVD and respiratory diseases [43].

5. Biological Mechanisms Underlying the Interplay

The connection between CVD and lung dysfunction, together with cancer, develops through intertwined inflammatory reactions and oxidative mechanisms. Chronic inflammation serves as the main cause of all three conditions since pro-inflammatory cytokines TNF-α, IL-6, and IL-1β remain elevated in patients who have cardiovascular disease and impaired lung function, and cancer [44]. Endothelial dysfunction, which characterizes CVD, develops from these cytokines while they simultaneously pull immune cells to both lungs and other tissues to build conditions suitable for tumor expansion. These diseases experience pathogenic development due to oxidative stress. ROS produced

during inflammation induce DNA damage along with compromised repair mechanisms that help trigger tumorigenic mutations [45]. This continuous cycle of oxidative stress speeds up vascular damage, damages lung function, and makes individuals more susceptible to cancer development, with a special risk in the lungs where tobacco smoke and air pollution exposures enhance these processes. The differential mechanism of oxidative stress and inflammation in diseased vs. healthy hearts is depicted in Figure 3.

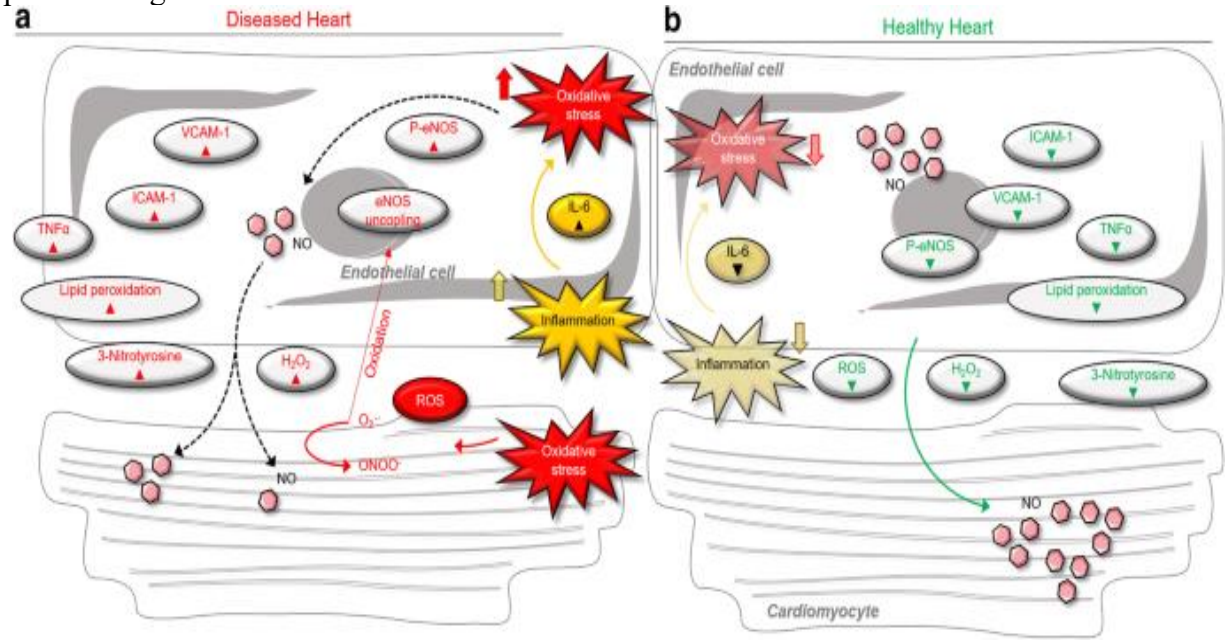

Figure 3: Differential Mechanisms of Oxidative Stress and Inflammation in Diseased vs. Healthy Hearts

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The development of CVD, together with respiratory diseases and cancer, is powerfully affected by genetic and epigenetic elements. Genetic susceptibility factors, including TP53 tumor suppressor gene mutations and KRAS oncogene mutations, make people more prone to experiencing both lung and cardiovascular health problems since these diseases share genetic connection points [46]. DNA methylation as well as histone modification function alongside epigenetic processes to control the expression of genes that determine disease progression. CVD experiences modifications that influence gene expression related to endothelial function, whereas cancer develops through tumor expansion along with metastasis activation from these modifications. The development of health disorders becomes challenging due to genetic predisposition along with environmental elements like smoking, combined with dietary factors and pollution exposure, because gene-environment interactions drive disease initiation and advancement [47].

All three diseases feature chronic inflammation as their vital component which actively advances their development. The occurrence of extended inflammation results in atherosclerotic plaque generation in CVD patients and causes airway remodeling and lung tissue deterioration in respiratory diseases like COPD [6]. The tumor microenvironment transforms due to inflammation, which helps cancer initiate new tumors while allowing them to grow and spread throughout the body. The disease advancement becomes worse through immune system weakness that develops from chronic inflammation, as immune cells end up either fatigued or supporting tumor growth. The therapeutic use of anti-inflammatory treatments, which include IL-6 inhibitors along with corticosteroids, demonstrates potential benefits for cardiovascular and respiratory disease management as well as cancer prevention and better patient results [48].

6. Public Health Implications and Interventions

Preventive strategies help control CVD, lung dysfunction, and cancer risks the most by focusing on lifestyle changes. Various studies indicate that physical exercise minimizes every condition as it enhances cardiovascular health and lung capacity and prevents cancer formation, particularly in colorectal, lung, and breast tissues [49]. The greatest preventive measure available today is smoking cessation, since cigarette use dramatically increases the risk for CVD, COPD, and multiple cancers, starting with lung cancer. Research has proven that smoking cessation makes people less likely to develop these diseases and creates better overall health results [50]. Also, it is believed that following a Mediterranean diet with its focus on fruits vegetables, and healthy fats decreases inflammation, together with enhancing cardiovascular health and preventing cancer [51]. Such lifestyle modifications both stop diseases from developing and support the management of current health problems to enhance life quality while extending lifespan.

The integrated healthcare system must handle CVD together with lung dysfunction and cancer, as their medical conditions deeply intersect with each other. Medical strategies need to approach healthcare through complete systems that simultaneously treat cardiovascular concerns alongside respiratory needs and cancer health [12]. Proficient heart healthcare requires joint efforts between medical experts in cardiology and pulmonology, and oncology, with nutritionists and physical therapist specialists. The combination approach aids in prompt detection and effective treatment, as well as aids in avoiding complications that occur from connected medical conditions. Multiple interventions that focus on shared risk elements, such as weight problems combined with cigarette usage and inactive lifestyle patterns, help stop medical conditions from worsening and achieve better patient results [26]. Integrated care approaches decrease health-related expenses through optimal resource management which aids in stopping chronic conditions from becoming advanced illnesses needing intensive medical care.

Public health policies function as key components in decreasing the weight of these illnesses. Early detection screening programs for CVD, along with lung dysfunction diseases (COPD and lung cancer) and cancers, generate early interventions that result in better health outcomes as well as improved survival rates. Generic population-based lung cancer screening of individuals at high risk, like smokers, produces major mortality rate declines [52]. Environmental health policies need to control air pollution, along with smoking exposure and industrial toxins, as these regulations help stop cardiovascular and respiratory diseases and cancer development. Public health outcomes will improve because legislative measures supporting clean air standards, working environment safety and tobacco regulations help reduce disease effects.

7. Research Gaps and Future Directions

The expanding research about cardiovascular health relationships with lung function and cancer risk maintenance fails to address important underlying mechanisms. The current research lacks thorough investigations that would study multiple interconnecting elements among these medical conditions simultaneously. Research on CVD and respiratory dysfunction, and cancer relationships exists, but insufficient studies investigate their combined effects on disease progression through time. The majority of studies examine how individual diseases like CVD, lung disease, or cancer affect cancer risk, yet they do not analyze the combined impact of having multiple diseases at once. The pathways that unite CVD with lung dysfunction and cancer remain unclear despite the lack of understanding about how persistent inflammation and oxidative stress from CVD and lung diseases work together to advance cancer development [53]. Research regarding how particular environmental factors, such as air pollution and occupational hazards, influence combined disease progression remains very limited.

Future investigations must conduct comprehensive longitudinal cohort studies that monitor cardiovascular health, combined with lung functions and cancer susceptibility across extensive time frames. Research studies should examine the joint disease development process between these conditions while investigating the extended impacts of mutual risk factors. Research involving

experiments should explore the direct mechanisms by which cardiovascular problems and lung efficiency issues lead to cancer development. Studies should investigate how particular biomarkers and inflammatory mediators, together with genetic predispositions, contribute to the pathophysiological processes between these diseases. Future research should include representative populations from different cultural backgrounds, together with socio-economic levels and geographical areas, to achieve widespread applicability of results. The proposed research method would yield an expanded understanding regarding the impact of worldwide health inequalities on cardiovascular health, together with lung function and cancer susceptibility. Such research directions will enhance scientific knowledge about disease connections between lung health challenges, heart conditions, and cardiovascular threats, as well as support the development of optimized intervention strategies.

8. Conclusion

Cardiovascular health, lung function, and cancer risk are important public health challenges intertwined with each other. The three health conditions advance due to common risk elements that combine smoking habits with inactivity and dietary neglect. The diseases become more severe due to biological mechanisms that combine chronic inflammation with oxidative stress and metabolic disturbances to form a dangerous cycle that raises cancer risk. Scientific research using epidemiological approaches and cohort studies demonstrates that cancer risk increases when people have poor lung function and heart disease; thus, it becomes vital to screen these patients intensively. The relationship between these diseases at the molecular and genetic levels requires more extensive research due to existing knowledge gaps. Longitudinal cohort investigations should lead future research to refine complex disease relationships because they will advance the development of unified healthcare programs. Early cancer detection programs and environmental safety frameworks through public health policies decrease the collective impact of these interrelated diseases on communities. The reduction of global cardiovascular diseases and lung dysfunction, along with cancer, requires preventive measures to function with integrated healthcare and public health initiatives.

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