Indoor and Outdoor Exposure: Recent Advances in Research on Lung Cancer in Women and Its Association with Household Cooking Fumes, Secondhand Smoke, and PM2.5

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Abstract: This study provides important theoretical basis for etiological research and the development of prevention and control strategies for lung cancer in women. By systematically examining the independent and synergistic exposure effects of household cooking fumes, secondhand smoke, and PM2.5, the study overcomes the limitations of traditional single-factor research and, for the first time, reveals the environmental pathogenic mechanisms underlying the high incidence of lung cancer in non-smoking women from an integrated perspective of indoor and outdoor dual exposure. The study elucidates the synergistic carcinogenic effects of the three environmental factors through common pathways such as oxidative stress, DNA damage, chronic inflammation, and epigenetic changes, providing a new theoretical framework for explaining the lung cancer incidence risk in this special population of non-smoking women. These findings not only enrich environmental carcinogenesis theory but also lay a scientific foundation for developing targeted lung cancer prevention and control strategies for women, particularly providing critical theoretical support for individual protective measures and social intervention policies in high-risk environments.

***Keywords*:**Female lung cancer, Environmental factors,Synergistic carcinogenesis,Oxidative stress,Prevention strategies

1. Introduction

In recent years, lung cancer has become one of the most common and deadly forms of cancer worldwide. Although the traditional view still believes that lung cancer is mainly related to male smoking, epidemiological data show that the incidence of female lung cancer is showing a rapid upward trend. According to the 2019 Global Cancer Statistics (Global Cancer Statistics 2019) and data from the International Agency for Research on Cancer (IARC)'s CA: A Cancer Journal for Clinicians, female lung cancer incidence ranks third among malignant tumors, with mortality rates ranking second, and the growth rate is significantly higher than that of males [1]. Notably, 15%-20% of lung cancer patients are non-smokers, and among them, women have twice the risk of developing the disease compared to men. In Asian countries, the proportion of non-smokers in China is particularly striking, reaching as high as 80% [2].

The pathological type of lung cancer in women is primarily adenocarcinoma within non-small cell lung cancer, accounting for approximately 80%, and its pathogenesis is closely related to environmental factors [3]. A study by the University of Buffalo published in the journal Cancer Causes & Control confirmed that even breathing indoor polluted air significantly increases the risk of cancer, particularly for women. The study found that indoor air pollution, especially fine particulate matter, is a key factor contributing to the high risk of lung cancer among Chinese women. In addition to smoking, which is a well-established risk factor, household cooking fumes, secondhand smoke, and exposure to environmental PM2.5 are also considered important risk factors for lung cancer in women. At the same time, these factors may act through multiple biological mechanisms to synergistically promote carcinogenesis, significantly increasing the risk of lung cancer in women. As urbanization accelerates and lifestyles change, women face growing dual exposure risks in both indoor and outdoor environments, necessitating systematic research to elucidate the underlying mechanisms and provide evidence for public health interventions.

2. Household cooking fume exposure and female lung cancer

2.1 Composition and carcinogenic mechanisms of household cooking fumes

Domestic cooking fumes primarily originate from the volatilization of cooking oils and smoke produced by fuel combustion during cooking. Their composition is complex, containing over 200 harmful substances such as polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), fine particulate matter (PM2.5), and aldehydes [4]. High-temperature cooking methods (such as frying, deep-frying, and stir-frying) significantly increase the concentration of harmful substances in cooking fumes. Studies have shown that when cooking oil is heated above 270°C, PM2.5 concentrations

can reach levels dozens of times higher than those of severe pollution [5]. These complex components can increase oxidative stress through mechanisms such as stimulating airway reactivity, damaging DNA, and promoting lipid peroxidation, thereby exacerbating pulmonary inflammatory responses and affecting the activity of surfactants. If you inhale kitchen soot for a long time, its harmful substances will accumulate in the body, thus increasing the risk of lung cancer.

Harmful ingredients in cooking soot can promote the occurrence of lung cancer through a variety of molecular mechanisms. First, carcinogens such as PAHs can enter cells through cell membranes and, under the action of enzymes in organs like the liver, be converted into active metabolites that form adducts with DNA, inducing gene mutations and triggering abnormal cell proliferation, thereby promoting tumor formation [6]. Second, free radicals and reactive oxygen species in cooking fumes can trigger oxidative stress within cells, causing oxidative damage to lipids, proteins, and DNA. Additionally, prolonged exposure can induce chronic inflammatory responses in the lungs, with inflammatory cells releasing numerous mediators that create a favorable microenvironment for tumor growth [7]. Additionally, long-term exposure to cooking fumes can suppress immune cell function, damage the local respiratory tract barrier, weaken immune defense capabilities, and prolong the retention time of carcinogens in the lungs [8]. These mechanisms collectively form the "DNA damage-oxidative inflammation-immune suppression" carcinogenic pathway, systematically elucidating the pathophysiological basis of cooking fumes causing lung cancer.

2.2 Epidemiological evidence

Wu Mengru's study showed that women who were continuously exposed to cooking fumes during adulthood had a 2.73-fold higher risk of lung cancer than those without exposure (95% CI: 1.04-7.17), while those exposed from childhood had an even higher risk of 4.29-fold (95% CI: 1.52-12.13). Additionally, households using solid fuels (such as coal or wood) for cooking or heating produce large amounts of smoke and harmful gases due to incomplete combustion, resulting in a 5–10-fold higher risk of lung cancer compared to households using clean energy (95% CI: 1.39–18.72) [9]. Multiple case-control studies have confirmed that women exposed to kitchen cooking fumes over the long term have a significantly increased risk of lung cancer. In recent years, with the popularization of clean fuels, public health awareness has increased, health commissions in multiple provinces have called for "reducing kitchen fumes and maintaining cooking safety standards," emphasizing that improving kitchen ventilation, promoting the use of high-efficiency range hoods, and adopting clean energy are key measures to reduce the risk of lung cancer in women.

3. Passive Smoking and Lung Cancer in Women

3.1 The Carcinogenicity of Passive Smoking

Secondhand smoke contains over 7,000 chemical substances, including 70 known carcinogens such as benzo[a]pyrene and 4-(methylnitrosamino)-1-(3-pyridinyl)-1-butanone (NNK) [10].

The harmful components in secondhand smoke primarily damage the respiratory system and induce lung cancer through two key pathways. On one hand, the microscopic particles it contains can directly deposit in the small airways of the lungs, causing sustained damage to respiratory epithelial cells and leading to precancerous conditions such as chronic bronchitis and epithelial metaplasia [11]; on the other hand, these harmful compounds can also exert effects through epigenetic mechanisms, particularly by causing abnormal methylation of the promoter regions of key tumor suppressor genes such as p16 and RASSF1A, leading to gene silencing and loss of tumor suppressor function [12]. This dual mechanism of "direct cellular damage-epigenetic regulation" systematically explains the pathological basis of lung cancer development caused by secondhand smoke exposure.

3.2 Epidemiological Data

A meta-analysis of non-smoking women in China showed that passive smoking increases the risk of lung cancer by 23% (RR=1.23, 95% CI: 1.04-1.45) and the risk of death by 16% (RR=1.16, 95% CI: 1.04-1.28), revealing the significant harm of secondhand smoke to non-smoking women. Among these, the risk of passive smoking in the home environment is particularly prominent, with women whose spouses smoke having a 34% increased risk of lung cancer (RR=1.34, 95% CI: 1.06–1.69) [13]. This increase in risk is significantly higher than the impact of general passive smoking exposure, indicating that long-term, close exposure to spousal smoking has a stronger carcinogenic effect.

4. PM2.5 Exposure and Lung Cancer in Women

4.1 Sources and Toxicity of PM2.5

PM2.5 (particulate matter 2.5) refers to particles with an aerodynamic diameter ≤2.5 μm. It primarily originates from

industrial emissions, vehicle exhaust, and household fuel combustion. The polycyclic aromatic hydrocarbons (PAHs) and heavy metals (such as arsenic and cadmium) adsorbed on its surface possess strong carcinogenic properties [14].

PM2.5 promotes the development of lung cancer through multiple synergistic pathways. On one hand, after PM2.5 enters the respiratory tract and is phagocytosed by macrophages, it releases a large amount of oxidants and free radicals, triggering oxidative stress reactions, leading to DNA oxidative damage and cellular dysfunction [15]. Simultaneously, the oxidants inherent in PM2.5 can activate inflammatory cell oxidative stress pathways, exacerbating cellular damage. On the other hand, long-term exposure to PM2.5 induces hypermethylation of the promoter regions of key tumor suppressor genes such as p53 through the ROS-Akt-DNMT3B pathway, leading to their silencing [15]. This dual mechanism of "oxidative stress-epigenetic regulation" systematically elucidates the molecular basis for the close association between even low-concentration long-term exposure to PM2.5 and the occurrence and development of lung cancer.

4.2 Epidemiological studies

A cohort study in southern China showed that for every 10 μg/m³ increase in PM2.5, the lung cancer mortality rate increased by 16% (RR=1.16, 95% CI: 1.09-1.23) [16]. This finding is consistent with results from multiple global studies, confirming the existence of a clear dose-response relationship between PM2.5 exposure and lung cancer risk. Of particular note is indoor PM2.5 exposure in rural areas due to the use of solid fuels, which increases the risk of lung cancer in women by 2–3 times. These data clearly demonstrate that elevated PM2.5 concentrations and prolonged exposure significantly increase the risk of lung cancer, with particularly pronounced effects on women who are chronically exposed to indoor pollution.

5. Synergistic effects of three factors

Kitchen fumes, secondhand smoke, and PM2.5 often coexist in real-world environments, and their synergistic effects may further exacerbate the risk of lung cancer in women. For example, in poorly ventilated kitchens, cooking fumes and secondhand smoke can release large amounts of fine particulate matter, leading to cumulative damage to respiratory cells. Additionally, these three exposure factors share high overlap at the molecular mechanism level, all capable of inducing carcinogenic effects through oxidative stress and activation of chronic inflammatory pathways. This shared mechanism suggests that they may not merely exhibit cumulative exposure effects but also result in synergistic carcinogenic effects, exerting an additive influence on lung cancer development .

6. Public Health Intervention Recommendations

In recent years, the incidence of lung cancer in women has shown a continuous upward trend, a phenomenon closely related to multiple environmental exposure factors. Among them, the triple factors of fumes, passive smoking and PM2.5 pollution caused by home cooking increase women's risk of lung cancer. In the face of this severe indoor and outdoor environment situation, it is urgent to build a public health intervention system covering policy regulation, environmental improvement, health education and early screening, and implement source prevention and control, risk control and accurate health promotion through multi-faceted and multi-level cooperation, so as to effectively curb the epidemic trend of women suffering from lung cancer.Specific recommendations are as follows.

6.1 Strengthen health education and raise awareness of prevention and control

Conduct targeted health education activities, focusing on high-risk groups such as housewives and food service workers, through community lectures, short videos, social media, and other channels to promote awareness of the hazards and prevention methods related to household cooking fumes, secondhand smoke, and PM2.5 pollution in an easy-to-understand manner.In terms of lifestyle. On the one hand, we should vigorously advocate low-smoky cooking methods. Promote healthy cooking methods such as steaming, cooking and stewing through public service advertisements and cooking courses to reduce high-temperature frying. On the other hand, it is necessary to implement "smoking-free families" and create activities to encourage family members to quit smoking together.Specific protective measures include the proper use of high-efficiency range hoods (turning them on in advance and delaying shutdown), selecting refined cooking oils, adopting low-temperature cooking methods, and improving kitchen ventilation conditions. Research has shown that these measures can effectively reduce exposure to cooking fumes. Through systematic health education and behavioral interventions, the self-protection capabilities of women can be effectively enhanced.

6.2 Promoting environmental and facility improvements

Promoting comprehensive smoking bans in public places and strengthening smoke-free home environments are key to reducing exposure to secondhand smoke. Ding Shiyas survey found that although 89% of lung cancer patients had been

exposed to secondhand smoke, only 4% supported comprehensive smoking bans in public places [17]. To effectively reduce the risk of lung cancer in women, it is necessary to strengthen comprehensive smoking bans in public and workplace settings, improve laws and regulations, and increase enforcement efforts; promote smoke-free home environments, advocate for smoke-free family values, and build a safe environment for family members. Among them, it is necessary to focus on promoting the environmental transformation project of the family kitchen, ensuring the basic smoke exhaust system of housing, and reducing home cooking pollution from hardware facilities. Additionally, efforts should be made to improve indoor air quality through measures such as promoting the use of air purification equipment and implementing green building standards to effectively reduce the accumulation of carcinogenic substances like radon and formaldehyde, thereby creating a healthier and safer living environment for women.

6.3 Improve early screening and precise prevention and control

To improve the early screening and precise prevention and control system, a low-dose spiral CT (LDCT) screening mechanism should be established for women who have been exposed to cooking fumes, secondhand smoke, or high-pollution areas for an extended period. By developing community health record systems and combining individual environmental exposure histories, women's lung cancer risks can be scientifically stratified and managed, with personalized health guidance plans provided. Concurrently, it is essential to strengthen the capacity-building of primary healthcare institutions. Through systematic training, the ability of community healthcare workers to identify early symptoms of lung cancer should be enhanced, thereby effectively improving the early diagnosis rate of lung cancer and improving patient outcomes. These measures will establish a comprehensive prevention and control chain from risk assessment to early detection and precise intervention, providing robust support for the prevention and control of lung cancer in women.

6.4 Adopting multiple measures to reduce PM2.5 exposure risks

Research indicates that comprehensive protective measures can significantly reduce PM2.5 exposure risks. During periods of high smog incidence, it is recommended to minimize outdoor activities and wear masks compliant with national standards when necessary; indoor environments should be equipped with high-efficiency air purification devices and maintained regularly to ensure filtration effectiveness; Additionally, efforts should be accelerated to promote clean energy replacement projects, vigorously promoting the use of clean energy sources such as natural gas in residential and industrial sectors to reduce PM2.5 emissions at the source. These measures have been proven through empirical research to effectively lower air pollution exposure levels [18].

7. Conclusion

In recent years, the rapid rise in lung cancer incidence among women, particularly the significant increase among non-smokers, has become a major global public health challenge. Research indicates that this phenomenon is closely associated with dual exposure to indoor and outdoor environmental factors, with household cooking fumes, secondhand smoke, and PM2.5 being the three key risk factors. Carcinogens such as polycyclic aromatic hydrocarbons (PAHs) in household cooking fumes promote lung cancer development through mechanisms including DNA damage, oxidative stress, and immune suppression; passive smoking directly damages respiratory epithelial cells and triggers epigenetic changes; PM2.5 exacerbates lung lesions through oxidative stress and chronic inflammation. These three factors not only pose significant risks when acting alone but may also amplify risks through synergistic effects, especially in poorly ventilated indoor environments. To address this trend, comprehensive intervention measures are urgently needed. At the individual level, women's health education should be strengthened to enhance their awareness of the hazards of environmental exposure, and protective knowledge should be disseminated through social media platforms, such as improving kitchen ventilation, reducing exposure to secondhand smoke, and wearing masks on hazy days. At the societal level, governments need to improve legislation and strengthen interdepartmental collaboration, using policy guidance, legal constraints, and public education to reduce the risk of lung cancer in women. This study innovatively explored the interactive effects of household cooking fumes, secondhand smoke, and PM2.5, revealing their synergistic impact mechanisms on female lung cancer and providing new insights for etiological research. However, the study also has limitations, such as relying on secondary data that may not fully reflect the actual situation, failing to comprehensively analyze all influencing factors (such as genetic background and lifestyle habits), and lacking in-depth exploration of individual differences. Future research should further clarify the synergistic mechanisms of environmental factors, explore personalized prevention strategies using multidisciplinary approaches, and strengthen cross-disciplinary collaboration between environmental science, public health, and clinical medicine to develop more precise prevention and control policies, ultimately reducing the incidence and mortality rates of lung cancer in women.

References

[1] Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin. 2021;71(3):209-249.

[2] Pelosof L, Ahn C, Gao A, et al. Proportion of Never-Smoker Non-Small Cell Lung Cancer Patients at Three Diverse Institutions. J Natl Cancer Inst. 2017;109(7):djw295.

[3] Chen Yinglan, Wan Hong. The incidence of lung cancer in women is rising rapidly; these risk factors should be taken seriously. China National Knowledge Infrastructure. 2022.

[4] Wu Mengru. The impact of kitchen fume exposure on the risk of lung cancer in non-smoking women and effective preventive measures. Anhui Medical University. 2024.

[5] Liu Shaowei. The main culprit behind lung cancer in Chinese women: kitchen fumes. Cancer Prevention and Control. 2021.

[6] Stadler R, Gastelum G, Chu C, et al. Molecular Mechanisms of Pulmonary Carcinogenesis by Polycyclic Aromatic Hydrocarbons (PAHs). Semin Cancer Biol. 2021;76:3-16.

[7] Ma Y, Deng L, Ma P, et al. In Vivo Respiratory Toxicology of Cooking Oil Fumes. J Hazard Mater. 2021;402:123455.

[8] Li Y, Xu H, Wang J, et al. Personal Exposure to PM2.5-Bound Organic Species from Domestic Solid Fuel Combustion. Chemosphere. 2019;227:53-62.

[9] Hecht SS. Recent Studies on Mechanisms of Bioactivation and Detoxification of 4-(Methylnitrosamine)-1-(3-Pyridyl)-1-Butanone (NNK). Crit Rev Toxicol. 1996;26(2):163-181.

[10] Ye Xiu, Xu Ning. Meta-analysis of cohort studies on the association between passive smoking and lung cancer in non-smoking women. Journal of Environmental Health. 2022.

[11] Xue Y, Wang L, Zhang Y, et al. Air Pollution: A Culprit of Lung Cancer. J Hazard Mater. 2022;434:128937.

[12] Li J, Li WX, Bai C, et al. Particulate Matter-Induced Epigenetic Changes and Lung Cancer. Clin Respir J. 2017;11(5):539-546.

[13] Zhou Wei. The Role of Abnormal DNA Methylation Patterns in the Process of PM2.5-Induced Lung Cancer [D]. Beijing: Academy of Military Medical Sciences, People's Liberation Army of China, 2017.

[14] Guo T, Chen S, Wang Y, et al. Potential Causal Links of Long-Term Air Pollution with Lung Cancer Incidence. Int J Cancer. 2024;154(2):251-260.

[15] Shen S, Luo M, Meng X, et al. All-Cause Mortality Risk Associated With Solid Fuel Use Among Chinese Elderly People. Front Public Health. 2021;9:741637.

[16] Wang L, Xiang Z, Stevanovic S, et al. Role of Chinese Cooking Emissions on Ambient Air Quality and Human Health. Sci Total Environ. 2017;589:173-181.

[17] Corrales L, Rosell R, Cardona AF, et al. Lung Cancer in Never Smokers: The Role of Different Risk Factors Other Than Tobacco Smoking. Crit Rev Oncol Hematol. 2020;148:102895.

[18] Ding Shiyia, Guan Yun, Huang Xianghua, et al. Survey on Smoking and Secondhand Smoke Exposure Among Lung Cancer Patients and Their Attitudes Toward Smoking Bans in Public Places. South China Journal of Preventive Medicine. 2022.