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How Iron Intake and Iron Biomarkers Affect the Risk of Developing Lung Cancer

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Abstract

It is unclear how iron intake and iron indicators affect a person's vulnerability to lung cancer. This study's objective was to conduct a systematic review and meta-analysis to evaluate the association between body iron stores or dietary iron consumption with the risk of developing lung cancer.

Keywords: Iron biomarkers • Iron intake • Lung cancer • Meta-analysis • Systematic reviews

Introduction

With an expected 2.2 million new cases and 1.8 million estimated deaths from lung cancer in 2020, it is the most prevalent cancer in the world. Lung cancer is thought to be more common due to genetic factors such heritable and somatic mutations. Smoking, air pollution, and exposure to carcinogens are other environmental variables that increase the risk of lung cancer. Dietary changes have recently offered promising ways to lower the incidence of lung cancer. In the meanwhile, dietary and lifestyle changes may reduce the risk of getting lung cancer, according to the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR). The human body requires iron to function. Numerous biological functions, such as cellular respiration, oxygen sensing, oxygen transport and metabolism, energy metabolism, DNA synthesis and repair, and signaling, depend on proteins that contain iron. However, an excess of iron may be hazardous, leading to DNA damage, oxidative stress, and carcinogenesis. Also connected to the start and growth of cancer, including lung cancer, is iron dysregulation.

Despite biological plausibility, population-based studies have not adequately summarised the relationship between iron and the risk of developing lung cancer. 13 papers altogether, published between 1989 and 2013, were found in Chen et al2019.'s meta-analysis on blood iron and lung cancer risk. The evaluation, which primarily included research from Chinese databases, did not examine the relationship between the incidence of lung cancer and any other iron biomarkers. Three studies were included in a previous meta-analysis that looked at the connection between heme iron consumption and the chance of developing lung cancer, and two more cohort studies followed. However, no meta-analyses have examined the relationship between iron and non-heme intake with the risk of lung cancer. Therefore, the current study's goal was to conduct a systematic review of the literature and meta-analysis of case-control and prospective cohort studies evaluating the relationship between the risk of lung cancer and both iron biomarkers and iron intakes, while taking into account potential differences based on regions, study designs, and iron status (including serum ferritin, serum iron, serum transferrin, TSAT, Total Iron-Binding Capacity (TIBC), lung tissue iron, and BALF ferr) (i.e., heme iron, nonheme iron). According to pooled ORs with 95% CIs, meta-analyses were utilised to calculate the link between iron intake and lung cancer risk. To compare and evaluate the correlations between iron biomarkers and lung cancer risk, the SMDs and 95% CIs were also elicited. By performing the Cochran Q test and computing the inconsistency index (I2), the heterogeneity was determined. We defined statistical significance as I2 > 50% or P 0.10 for the Q test. The random effects model was employed if the I2 value was greater than 50%; otherwise, the fixed-effects model was utilised. Sensitivity analysis was done to see if leaving out one study could change the outcome as a whole. To identify publication bias, Begg's and Egger's tests were also applied. We conducted subgroup analyses based on the study's design, geographic region (Asia, Europe, or South America), and participant characteristics (cohort or case-control).

After eliminating duplicates, a primary search of the PubMed, Web of Science, Scopus, Embase, and Cochrane databases yielded a total of 139,946 articles. 131,163 items were removed because they had irrelevant titles or abstracts, however. 20 studies were included in the systematic review and meta-analysis after the full texts of the publications were assessed. Two researches indicated blood ferritin concentrations in patients with lung cancer were considerably higher than that of healthy controls, while other studies showed no difference. The analysis included five studies showing the connection between serum ferritin with lung cancer risk. Serum ferritin concentrations in lung cancer patients were found to be substantially higher than those in healthy controls in the pooled analysis (SMD, 0.235, 95% CI, 0.129, 0.341). The blood transferrin levels in lung cancer patients were significantly lower than those in healthy controls, according to a meta-analysis that combined the results for transferrin and TIBC (a proxy measure of transferrin) (SMD, 0.591, 95% CI, 1.18, 0.003, I2 = 87.7%). In subgroup studies, the case-control study design studies with a methodological quality score of 7 and the studies using colorimetry to measure transferrin concentrations (SMD, 0.755, 95% CI, 1.634, 0.123) all showed a significant difference in the serum transferrin level between lung cancer patients and healthy controls.

In three case-control investigations, the iron concentrations of healthy lung tissue and cancer tissue were compared in lung cancer patients, and the results showed no significant differences between the two types of pulmonary tissue (SMD, 0.16, 95% CI, 0.068, 0.389, I2 = 0%). The meta-analysis also showed that there were no appreciable differences in BALF ferritin levels between lung cancer patients and healthy controls, despite the fact that the number of studies was small (SMD, 0.299, 95% CI, 0.799, 1.397), and the heterogeneity was considerable (I2 = 90.1%). There was no statistically significant correlation between toenail iron concentrations and lung cancer incidence in a population-based case-control study conducted in Appalachian Kentucky (OR, 0.993, 95% CI, 0.67-1.46). The correlations between dietary iron consumption and lung cancer incidence across the subgroups stratified by study design, BMI adjustment or lack thereof, and adjustments for physical activity or inclusion or exclusion did not reach statistical significance. Studies of low quality and alcohol consumption correction showed a significant inverse correlation when stratified by geographic location, methodological quality, adjusting for alcohol intake or not, and adjusting for meat intake or not. In contrast, research conducted in South America and studies that were corrected for meat consumption and family history of lung cancer did not find any significant relationships. This detailed summary and evaluation of the relationships between various iron biomarkers, iron intakes, and lung cancer was carried out as part of a systematic review and meta-analysis.

The results showed that lung cancer patients had significantly greater blood ferritin and TSAT levels and lower serum transferrin levels than healthy controls. Our findings, however, do not confirm associations between lung cancer risk with iron intakes or other iron indicators. This is the first systematic review and meta-analysis that particularly includes different measures of iron intakes and body iron indicators, as far as we are aware.

An essential protein in the body that stores iron, ferritin can be used to detect either an iron deficiency or an iron overload. According to earlier research, tumour cells contain a lot of ferritin, and higher ferritin levels, a sign of a high iron load, may have the power to cause mutations, control the production of hydroxyl radicals, and encourage carcinogenesis through dietary pathways. Overall, our findings offered fresh proof of the link between lung cancer risk and serum ferritin levels, which were shown to be considerably higher in lung cancer patients compared to healthy controls. Additionally, an increased risk of lung cancer may be linked to reduced serum transferrin levels, another sign of iron overload. However, these findings should be evaluated given the small number of research.

Conclusion

The success rate of cancer organoid establishment has significantly increased in recent years thanks to advancements in culture technology. Even yet, different cancer types, people, and laboratories may use different culture media and passaging techniques. Additionally, although CRISPR/Cas9-mediated targeted gene editing in primary organoids demonstrated the potential to produce cancer organoids with appropriate genomic characteristics, it was limited to a small number of genes and, for the most part, was less effective. Additionally, organoid culture is still expensive, which restricts the use of this technology in cancer research on a larger scale. The reason that the therapeutic uses of cancer organoids have not been widely extended is due of these barriers to cancer organoid research. Cancer organoids are a promising area for future fundamental and translational cancer studies, according to mounting evidence. We shouldn't limit ourselves to biological methods in order to increase the potential for organoid utilisation. Combining expertise from other disciplines, such as biochemistry and biophysics, is extremely beneficial to the development of organoid technology and should be the norm for future scientific research in all areas. For instance, our team has described a multidimensional biosensor system that included cellular impedance biosensors and 2D and 3D LUAD patient-derived cell models to test various anticancer medications and further direct LUAD individual treatment.

Overall, improved standards, more advanced technologies and multidisciplinary intersections are mostly needed to further employ organoids in cancer research; only then can disease modelling, mechanism exploration, and drug testing with cancer organoids be more successful and credible. Normal explanations for the potential link between iron status and lung cancer involve dysregulated iron homeostasis.

A tumor's appearance and growth may be aided by iron overload since it can produce hydroxyl radicals, damage lipid, protein, nucleic acids, and numerous signalling pathways that can result in either cell death or cell transformation. Cancer cells typically have altered iron metabolism and are more dependent on iron than healthy cells are. Increased cell proliferation, angiogenesis, cell invasion, and metastasis are all effects of altered iron metabolism-related protein expression and/or activities in cancer cells.

Although there may be a biological connection between low iron levels and lung cancer, the validity of the evidence from human research is still debatable. We found no correlation between the risk of lung cancer and blood iron, TSAT, BALF ferritin, or lung tissue iron in our investigations. The outcomes agreed with a current meta-analysis by Chen and associates. Serum iron levels in lung cancer cases were not statistically different from controls when compared to those in this group's meta-analysis, which they conducted in 2019 and included 13 papers (SMD, 0.125, 95% CI 0.439, 0.189, I2 = 89.9%).

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