Scoping Review of 5 Common Occupational Cancers and Their Related Exposures

Ahmad Naghibzadeh-Tahami¹, Yahya Khosravi², Mahboubeh Es'haghi³, Ali-Akbar Haghdoost⁴* ¶

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Abstract

**Background:** Occupational cancers can be avoided by removing dangerous chemicals from the workplace or limiting occupational exposure. Approximately, 10 major risk factors account for 85% of all occupational cancers. This scoping review study aimed to determine the most important chemical carcinogens related to 5 known occupational cancers.

**Methods:** In this scoping review, we followed Arksey and O’Malley’s 5-step framework. Four databases (PubMed, Web of Science, Google Scholar, Scopus) were systematically reviewed for relevant published papers from January 2000 to September 2021. Studies were included in this scoping review, which examined the effect of carcinogenic (definite and probable) chemical exposures on 5 known occupational cancers (lung, bladder, laryngeal, leukemia, and liver). We reported the types of occupational carcinogens, the geographical diversity of studies, extraction of relative risks (RRs), hazard ratios (HRs), or odds ratios (ORs), and identified gaps in the existing literature.

**Results:** The highest number of studies was related to lung cancer (LC) (n = 26), bladder cancer (BC) (n = 11), laryngeal cancer (LaC) (n = 8), leukemia (LeC) (n = 3), and primary liver cancer (PLC) (n = 2), respectively. Most studies were performed in France and Canada (n = 8), Germany (n = 4), Finland (n = 3), Netherlands (n = 2), and Finland (n = 2), respectively. Furthermore, the most common occupational chemical carcinogens associated with the 5 known occupational cancers were asbestos, benzene, crystalline silica, polycyclic aromatic hydrocarbons (PAH), and diesel motor exhausts (DME).

**Conclusion:** Although the attributable risk of occupational cancers in developing countries is much higher, a small proportion of studies were performed in these countries.

**Keywords:** Occupational Carcinogens, Cancer, Risk Factor, Developing Countries

**Conflicts of Interest:** None declared

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Introduction

Occupational exposures were among the earliest carcinogens identified (1, 2). The term "occupational carcinogens" refers to occupational exposures, particularly chemical exposures, which are used or released as intermediate compounds during manufacturing and have been proven or suspected to cause cancer (3). According to estimates of the current and future burden of occupational diseases, occupational cancers are still a concern and they will continue to be so in the future due to workers being exposed to carcinogens (4).

According to the World Health Organization (WHO) estimates, carcinogen exposures in the environment and...
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Workplace cause around 19% of cancer diagnoses worldwide, resulting in nearly 1.3 million deaths per year (5). These carcinogens are one of the major categories of risk factors that can be reduced using preventative actions (6). According to the International Agency for Research on Cancer (IARC) classification, carcinogens are divided into 2 categories; Group 1 (known human carcinogen), and Group 2A (probable human carcinogen). The IARC monographs have designated nearly 200 exposures as carcinogenic or probably carcinogenic to humans. A high proportion of these exposures occur in industrial contexts. As a result, the impact of occupational exposure on cancer burden is a major public health concern in many nations (7-9).

BTEXs (benzene, toluene, ethylbenzene, and xylene), asbestos, crystalline silica, heavy metals such as arsenic and its inorganic compounds, beryllium and its compounds, cadmium and nickel compounds, wood dust, and pollution caused by diesel equipment are all known or probable carcinogens for workers in these industries (10). Also, lung cancer (LC), bladder cancer (BC), laryngeal cancer (LaC), primary liver cancer (PLC), and leukemia cancer (LeC) are among the 5 most common occupational cancers globally, according to the Institution of Occupational Safety and Health (IOSH) (11).

However, it does not appear that in the last 30 years in developed countries, including the United States, a coherent study has been conducted to evaluate occupational cancers and provide preventive strategies to control these cancers (12). According to the WHO, initiatives aiming at eliminating or reducing established risk factors for cancer, such as occupational exposures, are the most effective in reducing the global burden of cancer (13).

In total, evidence-based information on carcinogenic agent exposures and cancer risks in workers is needed for national and worldwide efforts to minimize the burden of occupational cancers (14). Furthermore, studies that estimate the number of cancers caused by historical occupational exposures, such as chemical, physical, or circumscriptional carcinogens, are critical for guiding public health and prevention priorities, as well as developing and enforcing labor regulations for various occupational exposures (15).

Based on the above, it can be said that occupational malignancies can be avoided by removing harmful compounds or limiting worker exposures. It is vital to understand the types of occupational carcinogens and their prevalence in this regard (14).

In total, despite the extensive scientific work done on occupational carcinogens, it seems that so far, a coherent review study has not been done to identify gaps in scientific evidence related to occupational carcinogens. Scoping reviews are comprehensive studies used to map available literature and to identify potential gaps based on evidence (16). As far as we know, no comprehensive review study based on occupational cancers has ever been done elsewhere in the world. On the other hand, knowing the major occupational carcinogens is critical for estimating the burden of cancers attributable to these exposures and implementing control and preventative measures to limit these exposures. Accordingly, it is necessary to conduct a comprehensive study to identify important occupational carcinogens.

Therefore, the present scoping review aimed to review studies conducted worldwide based on 5 known occupational cancers (LC, BC, LaC, PLC, and LeC) and determine the most common chemical exposures in occupational and industrial environments. Overall, the present study follows 4 objectives: (a) determine the geographical diversity for studies on occupational carcinogens and 5 cancers attributed to these exposures; (b) identify the types of occupational carcinogens associated with these 5 common cancers and the main outcomes (mortality/incidence) that were assessed about these exposures; (c) assess the quality and characteristics of studies in the field of occupational carcinogens; (d) report on the observed associations between occupational carcinogens and 5 known cancers and extraction of relative risks (RRs), hazard ratios (HRs), or odds ratios (ORs) of 5 common cancers attributed to occupational carcinogens; and (e) conduct a thorough examination of the field as a whole and identify gaps in the existing literature. Thus, to achieve the above goals, we conducted a systematic scoping review on occupational carcinogens and 5 common cancers associated with these exposures.

The present study has some implications to provide evidence to pave the path for future estimates of the burden of occupational cancers.

Methods
This scoping review is registered with the research registry (reviewregistry1271). Arksey and O'Malley's published a methodological framework for a scoping review in 2005 (17). The goal of this framework is to map the key concepts underpinning a research area, as well as the main sources and types of evidence available. The framework consists of 5 stages, which are as follows:

Stage 1: Determining the Primary Research Question
Our study query was as follows: What is known about the association between occupational carcinogens and the 5 known occupational cancers?

Our research question was as follows: What is known about the relationship between occupational carcinogens and the 5 known occupational cancers in the literature?

Stage 2: Identifying Relevant Studies
The review's research objectives were to identify occupational carcinogens and cancers in the world. Five cancers of LC, BC, LaC, PLC, and LeC were selected as the main cancers due to occupational exposures based on the literature review and expert opinion. However, to increase the sensitivity of the search strategy, it did not focus on these cancers, but in the review of the title and abstract and body text for screening articles, only these cancers were considered. Through 4 bibliographic databases, a literature search was done to find papers relevant to occupational cancer worldwide (PubMed, Web of Science, Google Scholar, Scopus) from January 2000 to August 2021. To find relevant papers, the titles, abstracts, and body texts were all searched for specific keywords.

According to the PICO statement, the search queries
Stage 3: Study Selection

We included articles reporting that occupational agents were limited to chemical agents evaluated by the IARC Monograph Programme on the Identification of Carcinogenic Hazards to Humans; that is, group 1 (carcinogenic to humans), group 2A (probably carcinogenic to humans), and group 2B (possibly carcinogenic to humans). Studies conducted before 2000 or other harmful environmental factors, including noise, radiation, and shift work, were excluded.

Duplicates were deleted after importing the identified articles into EndNote reference management. To ascertain potential eligibility, the titles and abstracts of all identified references in the original search were examined. If there was a disagreement between reviewers (A.N.T. and M.E.), the full-text publication was studied and discussed to reach a consensus. The full texts of the relevant references were acquired after the primary screening.

If additional information or study procedures were not otherwise available, we made one attempt to contact the authors of the included articles. It should be noted that HRs, ORs, and RRs, as parameters to show effect measures of the associations between carcinogens and studied common cancers, were extracted from the included studies.

Stage 4: Extracting the Data

We did not assess the quality of the individual studies or the risk of bias by scoping review methodology because our goal was to map the evidence and/or summarize the study results (17).

We used a data extraction form developed by Udoh et al (19) to aid our extraction process, as shown in Table 2.

| Table 1. Search strategy in this scoping review |
|------------------------------------------------|
| 1. Occupational exposure* OR job-related exposure OR occupant* OR workplace* OR job |
| 2. Neoplasms OR cancer* OR carcinoma* OR tumor |
| 3. Incidence OR mortality OR risk |
| 4. 1 AND 2 AND 3 |
| 5. 1 AND 2 AND 3 NOT animal |

| Table 2. Data extraction form adapted from Udoh et al (2020) |
|------------------------------------------------------------|
| Author in chief |
| Publication date |
| The study’s title |
| Design of the study |
| Setting for research (country) |
| Population under study |
| Number of participants in the study |
| Findings from the study |
| Significant findings |
| Conclusions |

Stage 5: Collating, Summarizing, and Reporting the Results

The final stage of a scoping review involves collating, summarizing, and reporting the findings. According to Arksey and O’Malley, a framework should be used to collate results. We used the Preferred Reporting Items for Systematic Reviews and the Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) (20).

For study characteristics, we created a data table. To answer our research questions, we compared characteristics and settings across all studies using these tables. Then, studies were reviewed to examine the most common effect measures. Finally, for the final inference, the conclusions of each study were evaluated.

Results

Figure 1 shows a modified PRISMA flow diagram that displays the publishing selection process. A total of 2349 publications were found during the initial systematic search (922 from PubMed, 1037 from Web of Science, and 390 from Google scholar). A total of 1149 publications remained after eliminating the duplicates (n = 1200). By screening the titles and abstracts, 1080 were removed. A total of 24 studies evaluating occupational exposure about health outcomes other than cancer were also eliminated from the remaining 69 full-text articles. In total, 45 relevant publications (10 cohort studies, 35 case-control studies) were retained for data extraction (summarized in Tables 3 and 4, some papers examined more than 1 outcome).

The Geographical Diversity of Studies Conducted Worldwide

Figure 2 shows the geographical distribution of studies worldwide. Eight studies were conducted in France (21-28) and Canada (29-36). There were also 4 papers in Germany (37-40), and 3 in Finland (41-43). Two studies were conducted in the Netherlands (44, 45) and Sweden (46, 47). Poland (48), China (49), the U.S. (50), England (51), Hong Kong (52), Turkey (53), Indonesia (54), Iran (55), and Italy (56) each had 1 article. Also, 9 studies were conducted jointly in several countries (57-65).

http://mjiri.iums.ac.ir
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### Table 3. The basic information of the studies included in this review

| First author /Location/ Date of publication | Population Size/Description | Cancer Sites | Exposure Agents Assessed | Covariates Controlled for in Modeling |
|--------------------------------------------|----------------------------|--------------|--------------------------|--------------------------------------|
| Sciannameo/ Italy/2019 | A cohort of 2991 (790 females & 2201 males) Italian electroplaters, workers who were potentially exposed to the hazards of galvanic production, cases:162 | LC *, BC | Nickel, chromium | Age, sex, calendar period |
| Liu, China/2013 | In a cohort in China (1960–2003), 34018 workers, with an average of 34.5 years of follow-up from seven metal mines and four pottery factories, cases:546, data collection: interviews, exposure assessment: JEM | LC | Silica Exposure | Sex, year of birth, and smoking |
| Siew/ Finland /2012 | Cohort of all Finnish men (born 1906 -1945) (1.2 million) followed up through the Finnish cancer registry (FCR), cases: nose (n = 292), nasopharynx (n = 149), and lung (n = 30,137) 1971–1995, exposure assessment: JEM | LC | Wood dust, formaldehyde | Smoking, socioeconomic status, and exposure to asbestos and/or silica dust |
| Offermans/Netherlands/2014 | Netherlands Cohort Study (NLCS) (58279 males aged 55 - 69), cases: after 17.3 years of follow-up 2324 LC cases available, data collection: self-administered questionnaire, exposure assessment: JEM | LC, LaC | Asbestos | Cigarette smoking, the number of cigarettes smoked per day, years of smoking cigarettes, exposure to crystalline silica, PAH |
| Lindbohm/Finland/2009 | A cohort of economically active Finns (1.2 million) (born 1906 -1945) was followed up (1.2 million, 1971–1995) by FCR, cases: 2474, exposure assessment: JEM | PLC | Organic solvents and gasoline vapors, Aliphatic and alicyclic HC | Alcohol, smoking, socioeconomic status (SES) |
| Bourkgard/ France/2009 | Historical cohort, all male (1672) and female (959) workers ever employed in a French carbon steel-producing factory, causes of death: via death certificates, data collection: interviews with and a review of historical documentation, exposure assessment: JEM. Cases: male workers who died from LC who had a known history of uranium mining, total sample: 8066 uranium miners, where 3174 died from LC exposure assessment: JEM | LC BC | Iron oxide | Asbestos, PAH, silica, smoking |
| Taeger/Germany /2008 | Cases: male workers who died from LC who had a known history of uranium mining, total sample: 8066 uranium miners, where 3174 died from LC exposure assessment: JEM | LC | Arsenic, quartz | Silicosis |
| Lohi/Finland/2008 | A cohort of all economically active Finns was followed up for BC. cases: All cancers diagnosed between 1971 and 1995 (10277) among people born between 1906 and 1945 were extracted from the nationwide FCR, exposure assessment: JEM. | BC | Chlorinate HC Solvents | Smoking, obesity, social class |
| Purdue / Swedish/2006 | Cases:510 H&NC (171 in the oral cavity, 112 in the pharynx, 227 in the larynx) were identified among 307799 male workers in the Swedish construction industry, exposure assessment: JEM | LaC | Asbestos mineral wool cement dust wood dust | Age, smoking |
### Table 3. Continued

| First author /Location/ Date of publication | Population Size/Description | Cancer Sites | Exposure Agents Assessed | Covariates Controlled for in Modeling |
|--------------------------------------------|-----------------------------|--------------|--------------------------|--------------------------------------|
| **Cohort studies**                         |                             |              |                          |                                      |
| Zhao/US/2005                               | A cohort of 55,000 workers employed (1950 - 1993) at several Boeing North America. cases:5049 of workers who were alive and at risk of being diagnosed with cancer. exposure assessment: JEM | LC, BC | PAH, mineral oils, benzene | Age                                   |
| **Case-control studies**                   |                             |              |                          |                                      |
| Sce`lo in six Central and Eastern Europe countries/2004 | Cases:2861, controls: 3118, occupational agents: collected based on detailed occupational questionnaires | LC | Vinyl chloride, acrylonitrile, styrene | Center, gender, age, tobacco consumption |
| Radoi/France /2019                         | Cases:2161 H&NC, controls:3555 population controls, data collection: standardized questionnaire and interview, exposure assessment: JEM | LaC | Leather dust | Age, area of residence, SES, smoking status |
| Warden /Canada/2018                        | Cases:733, controls:894 population controls. data collection: obtained via interview | LC | Benzene, toluene, xylene (BTX) | Age, smoking |
| Latifovic /Canada/2020                     | Cases: 658, controls:1360 age-frequency matched population control, data collection: self-administered questionnaires, exposure assessment: JEM | BC | Silica, asbestos | Province of residence, age, proxy respondent, pack-years, smoking, exposure to mineral/lube oil at work |
| Suraya /Indonesia/2020                     | Cases: 336, controls:360, data collection: questionnaire and interviews, exposure assessment: JEM | LC | Asbestos | Gender, age, ethnicity, education, smoking, environmental exposure |
| Hall /Western Europe12 and Latin America, Germany/2020 | Cases: 2256, controls:7857 population controls (1604 females; 6253 males), data collection: structured questionnaire and interview, exposure assessment: JEM | LaC | Asbestos, crystalline silica, chromium-VI, chromium-VI and nickel combined | The study, age, alcohol, tobacco smoking |
| Colin/France /2018                         | Cohort: included 22795 male workers from six French steel-producing factories, cases:84, controls:251, data collection: face-to-face interviews and questionnaires, exposure assessment: JEM | BC | MWFs (straight, soluble, and synthesized) | Smoking, age |
| Khedher /France/2017                       | Cases: 2926 incident cases with a histologically confirmed (18-75), controls:3555 population controls, data collection: questionnaire. exposure assessment: JEM | LC | Textile dust, cotton fibers | Asbestos, smoking, gender, age, geographic area of residence |
| Barul/France/2018                          | Cases: 454 histologically confirmed (18-75) controls:2780 Population controls | LaC | Petroleum-based solvents, oxygenated solvents | Smoking, alcohol |
| Talibov/Finland, Iceland, Norway, and Sweden/2017 | The study was nested in the Nordic Occupational Cancer Study (NOCCA) cohort. Cases: 20615 (diagnosed in 1961-2005), controls:103075 population controls, exposure assessment: JEM | LeC | Occupational solvent exposure | Age, year of birth |
| Ilar/Swedish/2017                          | Cases:993, controls:2359 (two groups, population-controls and mortality-matched population controls), data collection: questionnaire and telephone interviews, exposure assessment: JEM | LC | DME | Tobacco smoking, alcohol, age, sex, year of study, exposure to air pollution from road traffic |
| Hadkhale/Finland, Iceland, Norway, and Sweden/2017 | NOCCA database, cases: 113343 (1961-2005), controls: 566715 population controls, exposure assessment: JEM | BC | TCE, benzene, toluene, aromatic HC, aliphatic & alicyclic HC | Age, sex, birth year, country |
### Table 3. Continued

| First author / Location / Date of publication | Population Size / Description | Cancer Sites | Exposure Agents Assessed | Covariates Controlled for in Modeling |
|---------------------------------------------|-------------------------------|--------------|--------------------------|--------------------------------------|
| **Case-control studies**                    |                               |              |                          |                                      |
| Barul/France/2017                           | Cases: 1857, controls: 2780 population control, data collection: face to face interviews using a standardized questionnaire, exposure assessment: JEM | LaC          | Perchloroethylene (PCE), trichloroethylene (TCE), methylene chloride (MC), chloroform (CF), carbon tetrachloride (CT) | Age, tobacco smoking, alcohol consumption, asbestos exposure, |
| Switkowska/Poland/2015                      | Case-control studies were carried out within a cohort including 7374 former workers of asbestos processing plants (employed 1943-1998), cases: 165, controls: 825 population control | LC          | Asbestos                 | Cigarette smoking                    |
| Mattrat/France/2015                         | Cases: 2926 (18-75), identified during the study period (2001-2007), controls: 3555 population controls, exposure assessment: JEM | LC          | DME                      | Age, asbestos, silica, residential history, education, occupation, lifelong cigarette smoking, and alcohol consumption |
| Kachuri/Canada/2014                         | Cases: 1681 (1994-1997), population controls: 2053, data collection: self-administered questionnaire | LC          | Crystalline silica       | Cigarette smoking, second-handed smoke, DME |
| Latifovic/Canada/2015                       | Cases: 658, controls: 1360 population data collection: self-administered questionnaire controls, exposure assessment: JEM | BC          | DME                      | Cumulative silica exposures, cigarette pack-year, sex, 5-year age group |
| Colt/England/2014                           | Cases: 895 histologically confirmed (30-79) between 2001-2004, population controls: 1031, data collection: questionnaire and interviews | BC          | MWFs                     | State, gender, and age at diagnosis (within 5 years), smoking |
| Pesch/Germany/2013                          | Case-control study nested in the European Prospective Investigation into Cancer and Nutrition (EPIC), cases: 754, controls: 833, exposure assessment: JEM | BC          | PAH, aromatic amines     | Gender, age, smoking cigarettes, smoking of other tobacco types, age, research center |
| Mo’Hner/Germany/2013                        | A cohort study that followed up on approximately 6,000 German potash miners, cases: 68, controls: 340, exposure assessment: JEM | LC          | DME                      | Cigarette smoking                    |
| Guida/ France/2013                          | Cases: 1350 histologically confirmed LC in men (18-75), controls: 1912 population controls, data collection: face-to-face interviews via standardized questions, exposure assessment: JEM | LC          | MWS, asbestos, silica   | Age, cigarette smoking, gender, education, lifetime alcohol consumption |
| Villeneuve/Canada/2012                      | Cases: 1,681, controls: 2053 (recruited between 1994 and 1997), data collection: self-reported questionnaires | LC          | Asbestos                 | Age, cigarette smoking, SES, secondhand smoking, occupational exposure to silica, DME |
| Tse/Hong Kong/2012                          | Cases: 1208 male, controls: 1069 age-matched male population controls (2004–2006), data collection: face-to-face interviews via standardized questions | LC          | Asbestos, silica dust, welding fume, DME, MMMF | Smoking, indoor air sources pollutants, tobacco smoking alcohol, dietary habits, history of diseases |
| Villeneuve/Canada/2011                      | Cases: 1681 (men 40 years of age), 2053 population controls: data collection: self-reported questionnaire | LC          | DME                      | Crystalline silica, asbestos, cigarette smoking |
| Mannetje/Central Eastern Europe and UK/2011 | Cases: 2853, controls: 3104, data collection: face-to-face interviews via a questionnaire | LC          | Chromium, cadmium, nickel, arsenic | Cigarette smoking, age, center, sex, |
### Table 3. Continued

| First author /Location/ Date of publication | Population Size/Description | Cancer Sites | Exposure Agents Assessed | Covariates Controlled for in Modeling |
|--------------------------------------------|----------------------------|--------------|--------------------------|--------------------------------------|
| **Case-control studies**                   |                            |              |                          |                                      |
| Preller /Netherlands/2010                   | Men (58279) from the NLCS, cases:1667 after 11.3 years of follow-up, data collection: self-reported questionnaire, exposure assessment: JEM | LC | Silica | Age, family history of LC; smoking behavior, fruit/vegetable, asbestos |
| Olsson/ seven European countries and Liverpool (UK)/2010 | Cases:2852, controls:2936 population or hospital (1998-2002), data collection: questionnaire via interviews | LC | PAH | Age, sex, center, tobacco pack years, occupational exposure to silica, asbestos, metals (arsenic, chromium, cadmium) |
| Kiran/ Czech Republic, France, Germany, Italy, Ireland, and Spain /2010 ELCI/ Turkey/ 2009 | Cases:406, controls:2463 population controls, data collection: self-reported questionnaire (between 1998–2004) | LeC | Ethylene oxide | Age, sex, and participating center. |
| Cases:189 pathologically confirmed male NSND, controls: 536 NSND hospital-based controls, data collection: face-to-face interviews via a questionnaire | LC | Silica, grain dust, leather dust, asbestos, wood dust, cotton dust, PAH, DME, formaldehyde, solvent | Age, smoking, alcohol |
| Richardson /Canada/2007                    | Cases: 1062 adult male (diagnosed between 1983 and 1990), controls: 8057 population controls, data collection: self-administered questionnaire, exposure assessment: JEM | BC | Coal-tar pitches, mineral oils, Benz (a) anthracene, DME, Direct black 38,4-Chloro-ortho-toluidine, ortho-Toluidine | Ethnic origin, marital status, education, alcohol, cigarette smoking |
| Richardi/ Germany /2006                    | Cases: 595 histologically confirmed, controls: 845 population controls. data collection: structured questionnaire and through interviews, exposure assessment: JEM | LC | DME | Sex, smoking |
| Berrino/ four European countries /2003     | Cases: 315 male of hypopharyngeal/ LaC, controls:819 population controls (during 1979–1982), exposure assessment: JEM | LaC | Asbestos, PAH, chromium, arsenic, and compounds, wood dust, formaldehyde, solvents, | Age, center, tobacco, alcohol, diet, SES |
| Heinemann/six European countries /2000     | Cases: 317 women hospital cases, controls:1789 (1060 hospital controls and 719 population controls), exposure assessment: JEM | PLC | Beryllium, cadmium, formaldehyde, PAH, lead, mercury | Age, center, hepatitis infection, smoking, alcohol, oral contraceptive use |
| Roussua/ Canada/2007                      | Cases:3730 Men, controls: 533 population controls were interviewed. data collection: structured questionnaire and interviews | LC | Lead (organic, inorganic, gasoline emissions) | Age, tobacco, SES |
| Hosseini /Iran /2009                      | Cases:242 histologically confirmed (178 male, 64 female), controls: two controls for each patient (242 hospital controls and 242 visiting healthy controls), data collection: structured questionnaire and through interviews | LC | Asbestos, heavy metals, coal tar, soot, DME, Inorganic dust, wood dust, cotton dust, silica | Age, sex, place of residence |

LC: Lung cancer; BC: Bladder cancer; LaC: Laryngeal cancer; PLC: Primary Liver cancer; LeC: Leukemia
### Table 4. The results of the studies reviewed, about the association between occupational exposures and five common occupational cancers

| First author /Location/ Date of publication | Type of assessment | Outcome evaluated | Main results | Conclusion |
|--------------------------------------------|-------------------|------------------|--------------|------------|
| Suraya/ Indonesia/2020 ever exposure       | LC incidence      | Asbestos: risk was elevated forever exposure (OR = 2.04, 95% CI = 1.21–3.42), Exposure ≥10 (OR = 2.31, 95% CI = 1.26–4.26) | Elevated LC risk attributable to asbestos exposure. The disease risk is consistent with a dose-response relationship. |
| Latifovic/Canada/2020 Ever exposure       | BC incidence      | Silica: ever exposure (OR=1.29, 95%CI: 1.00–1.61), for ≥27 years 1.41 (95%CI: 1.01–1.98). Asbestos: ever exposure: (OR:1.32,95%CI:0.98–1.77), exposures ≥20 years ago (OR:2.04, 95%CI:1.25–3.34), <10 years (OR:1.75, 95%CI:1.10–2.77), lower tertile of cumulative exposure (OR:1.69, 95%CI:1.07 2.65) | Occupational silica and asbestos increase the risk of BC, silica exposure: an exposure-response relationship. |
| Hall/ Western Europe12 and Latin America/Germany/2020 Ever exposure Duration of exposure Cumulative exposure | LaC incidence | Asbestos: at >90 percentile cumulative exposure (OR: 1.3, 95% CI = 1.0, 1.6), Respirable crystalline silica: >30 years duration (OR: 1.4, 95% CI = 1.2, 1.7), 75th–90th percentile cumulative exposure (OR: 1.4, 95% CI = 1.1, 1.8), chromium-VI: at >75th percentile cumulative exposure (OR: 1.9, 95% CI = 1.2, 3.0), chromium-VI and nickel combined: at 20–29 years duration (OR: 1.5, 95% CI = 1.1, 1.9) | Exposure to asbestos, respirable crystalline silica, chromium-IV, and chromium-VI with nickel) increase the risk of LaC |
| Sciannameo/ Italy/2019 Cumulative exposure | LC & BC mortality | Chromium & LC: Not any association Chromium & BC: Not any association Nickel & LC: Increased risks for a cumulative exposure of (HR:6.03, 95% CI 2.94 -12.37) | Exposure to nickel compounds may increase the risk of LC |
| Offermans/Netherlands/2014 Ever exposure Duration of exposure Cumulative exposure | LC & LaC incidence | Asbestos & LC: Ever exposure (HR = 1.50; 95% CI: 1.27–1.78) duration of exposure (Lowest (HR=1.47; 95% CI: 1.15-1.87), Middle (HR=1.58; 95% CI: 1.21-2.07), Highest (HR=1.46; 95% CI: 1.2-1.9)) was associated with LC. The risk of LC increased with cumulative exposure to asbestos ((Lowest HR=1.44; 95% CI: 1.12-1.86), Middle (HR=1.40; 95% CI: 1.09-1.79), Highest (HR=1.76; 95% CI: 1.3-2.38)). Asbestos & LaC: No statistically significant relationship was observed | Asbestos exposure increased risk for LC |
| Liu/China/2013 Ever/never exposure Cumulative exposure | LC mortality | Silica: Quartiles of cumulative exposure yielded HR of 1.26(0.98, 1.60), 1.54 (1.16, 2.05), 1.68 (1.26, 2.24), and 1.70 (1.23, 2.34), respectively. | Silica exposure is associated with a significant increase in LC risk |
| Siew/Finnish/2012 Cumulative exposure | LC incidence | Formaldehyde: cumulative exposure to formaldehyde was associated with an elevated risk of LC (RR, 1.18; 95% CI: 1.12–1.25). Wood dust: not any association | Elevated LC risk attributable to cumulative exposure of formaldehyde |
| First author /Location/ Date of publication | Type of assessment | Outcome evaluated | Main results | Conclusion |
|--------------------------------------------|-------------------|-------------------|--------------|------------|
| Lindbohm/Finland/2009                       | Cumulative exposure| PLC incidence     | Aromatic HC: the highest exposure category (RR: 1.77; 95% CI: 1.30–2.40), Aliphatic/alicyclic HC: the highest exposure category (RR: 1.47; 95% CI: 0.99–2.18), Chlorinated HC: the highest exposure category (RR: 2.65; 95% CI: 1.38–5.11), The highest exposure category other solvents (RR: 2.14; 95% CI: 1.23–3.71). | Men who are exposed to chlorinated HC have a higher risk of PLC. |
| Bourgkard/France/2009                       | Ever exposure     | Duration of exposure | Iron oxide & LC: No excess was observed for ever exposure (RR= 0.80, 95% CI: 0.55–1.17), Oil mist & BC: Excess was observed for ever exposure: (RR =2.44; 95% CI: 1.06–5.60), duration of exposure: (RR=1.85; 95% CI: 1.07–3.19) and cumulative of exposure: (RR= 1.69; 95% CI: 1.03–2.79). | Exposure to Oil mist increases the risk of BC. |
| Taeger/ Germany /2008                       | Cumulative exposure| LC mortality      | Cumulative exposure to quartz (OR, 1.78; 95% CI: 1.39–2.26) and arsenic (OR, 1.18; 95% CI: 0.99–1.4) were determined as risk factors for LC and Middle levels of chlorinated HC solvents (1.7, 95% CI: 1.2–2.5) and a low level of aromatic HC solvents (1.6, 95% CI: 1.3–2.1) were associated with BC. | Evidence indicated that quartz and arsenic are risk factors for LC. |
| Lohi/Finland/2008                           | Cumulative exposure| BC incidence      | Evidence indicated that quartz and arsenic are risk factors for LC. | Evidence indicated that quartz and arsenic are risk factors for LC. |
| Purdue/Swedish/2006                         | Ever exposure     | cumulative exposure | Asbestos: Ever exposure was related to an increased LaC incidence (RR=1.9, 95% CI 1.2–3.1). | Asbestos and Mineral wool increases the risk of LaC. |
| Zhao/America/2005                           | Cumulative exposure| LC, BC & LeC incidence | Other exposures did not show a significant association. | Mineral wool experienced an increased risk of developing and/or dying from LC and LeC. |
| See’s in six Central and Eastern Europe countries/2003 | Ever exposure | Duration of exposure | Acrylonitrile: Ever exposure was associated to LC (OR: 2.20; 95% CI: 1.11–4.36). No association between exposure to styrene, vinyl chloride and LC risk was found. | Exposure to acrylonitrile increases the risk of LC. |
| First author / Location/ Date of publication | Type of assessment | Outcome evaluated | Main results | Conclusion |
|---------------------------------------------|-------------------|-------------------|--------------|------------|
| Radoï/France /2019 Ever exposure Cumulative exposure | Leather dust: Cumulative exposure was associated (OR = 2.26; 95% CI: 1.07–4.76); ever exposure was not associated (OR = 1.40; 95% CI: 0.77–2.56) | Increased cases of LaC attributable to leather dust | |
| Warden/Canada/2018 Ever exposure duration of exposure | Benzene: Exposure (OR: 1.35; 95% CI: 0.99–1.84) and exposure>10 years (OR: 1.44; 95% CI: 0.94–2.21) were associated with LC | Exposure to one or more of the BTX agents may be associated with LC | |
| Colin/France /2018 Duration of exposure, Cumulative exposure | Textile dust: Inverse association between working in textile dust and LC, although this relationship was not statistically significant (OR = 0.84, 95%CI 0.67–1.07). Cotton fibers: LC was significantly decreased among workers exposed (OR = 0.70, 95% CI 0.48-0.97). | Decreased risk of LC associated with exposure to textile dust, particularly cotton. | |
| Khedher/ France/2017 Ever exposure | Textile dust: Inverse association between working in textile dust and LC, although this relationship was not statistically significant (OR = 0.84, 95%CI 0.67–1.07). Cotton fibers: LC was significantly decreased among workers exposed (OR = 0.70, 95% CI 0.48-0.97). | Exposure to synthetic MWFs: not any association | |
| Barul/France/2018 Ever exposure Cumulative Exposure | Benzene: No significant association was found forever (OR: 0.94; 95%CI: 0.71–1.24) and cumulative exposure to low, medium and high | Exposure to petroleum-based or oxygenated solvents is not a substantial role in LaC risk | |
| Talibov/Finland, Iceland, Norway, and Sweden/2017 Cumulative Exposure | Perchloroethylene: Significantly risks were observed for cumulative exposure (OR = 1.61, 95% CI:1.01-2.56) among women non-significant associations were observed forever exposure to methylene chloride, perchloroethylene, and 1,1,1-trichloroethane in both exposure levels | There is not any association between solvent exposure and adult LeC | |
| Ilar/Swedish/2017 Ever exposure Duration exposure Cumulative Exposure | DME: OR forever exposure was 1.15 (95% CI:0.94–1.41). | Elevated risk for LC attributable to DME exposure | |
| Hadkhale/Finland, Iceland, Norway and Sweden/2017 Cumulative Exposure | Increased risks for TCE (HR=1.23; 95% CI:1.12–1.40), toluene (HR = 1.20, 95% CI: 1.00-1.38), benzene (HR= 1.16, 95% CI: 1.04-1.31), aromatic HC solvents (HR= 1.10; 95% CI: 0.94-1.30) and aliphatic & alicyclic HC solvents (HR =1.08, 95% CI:1.00-1.23) at high exposure level | Exposure to TCE, perchloroethylene, aromatic hydrocarbon solvents, benzene and toluene and an elevated risk for BC | |
| Barul/France/2017 Ever exposure cumulative exposure | The OR for LeC was 3.86 (95% CI = 1.30–11.48) for those exposed to the highest levels of PCE. There was no increased risk of exposure to TCE MC, CF, CT, and LeC | High exposure to PCE increases the risk of LeC | |
| Swiatkowska/ Poland/2015 Cumulative Exposure | Risk in the group with the highest exposure was two times higher (OR= 1.99; 95%CI: 1.22–3.25) | LC risk is associated with asbestos exposure and it increases along with the increasing exposure. | |
| First author /Location/ Date of publication | Type of assessment | Outcome evaluated | Main results | Conclusion |
|--------------------------------------------|-------------------|-------------------|--------------|------------|
| Matrat/France/2015                         | Ever exposure     | LC incidence      | DME: Ever exposure was associated with LC (OR = 1.3; 95% CI: 1.1–1.6). The more the cumulative exposure increases, the more the risk of LC increases (OR= 1.4; 95% CI: 1.1–1.6) for the highest IEC | DME exposure as a risk factor of LC |
| Offermans/Netherlands/2014                 | Ever exposure     | LaC incidence     | LaC showed a positive association after prolonged higher asbestos exposure (HR per10 years increment, 1.95[95% CI: 1.36 - 2.80]. | Asbestos levels may be associated with an increased risk of LaC |
| Kachuri/Canada/2014                        | Ever exposure     | LC incidence      | Silica: Increasing duration of exposure was associated with a significant risk to LC (OR≥30 years: 1.67; 95% CI: 1.21 -2.24), cumulative exposure was associated with LC risk (OR=1.81; 95% CI: 1.34–2.42), ever exposure was related to LC (OR=1.20; 95% CI: 1.0 –1.43) | Occupational exposure to silica is a risk factor for LC |
| Latifovic/Canada/2015                      | Ever exposure     | BC incidence      | DME: Exposure was not associated with BC; duration >10 years of exposure had a greater than two-fold increase in the risk of BC (OR = 2.45; 95% CI: 1.64 -5.74) | Exposure to high concentrations of DME may increase the risk of BC |
| Colt/England/2014                          | Ever exposure     | BC incidence      | Ever exposure: risk was elevated among men who reported using straight MWFs (OR=1.7; 95% CI: 1.1–2.8). Cumulative exposure to straight MWFs: was associated with BC (OR=2.2; 95% CI: 1.02 –4.8) | MWFs exposure was associated with a significantly increased BC risk |
| Pesch/German/2013                          | Cumulative exposure| BC incidence      | Exposure to aromatic amines and PAH was associated with an increased BC risk (highest exposure: OR=1.37; 95% CI: 1.02–1.84, and OR=1.50; 95% CI: 0.09–2.05, respectively) | Excess risks of BC are associated with occupational exposure to aromatic amines and are supportive of the role of PAHs in the development of BC |
| Mo¨hner/ German/2013                        | Cumulative reparable elemental carbon (REC) exposure as a continuous variable | LC mortality | Introducing cumulative REC exposure as a continuous variable yielded an odds ratio of 1.04 [0.70 –1.53] | Occupational exposure to asbestos & crystalline silica were associated with an increased risk of LC, no firm evidence that MWFs was not associated with LC |
| Guida / France /2013                        | Ever exposure     | LC incidence      | MWs: Ever and cumulative exposure was not associated with LC Asbestos: ever exposure was associated with significantly increased risk of LC (OR = 1.46; 95% CI: 1.17 -1.83). Crystalline silica: ever exposure was related to LC (OR=1.35; 95% CI: 1.03 -1.77) | Crystalline silica & asbestos were associated with an increased risk of LC. No firm evidence that MWFs was not associated with LC |
| Villeneuve / Canadian/2012                 | Ever exposure     | LC incidence      | Asbestos: cumulative exposure to medium or high concentrations of had OR for LC of 2.16 (95% CI=1.21–3.88, ever exposure increased risk of LC (OR = 1.28; 95% CI: 1.02 to 1.61) | Exposure to asbestos has contributed to an increased risk of LC |
| Tse / Hong Kong /2012                      | Ever/never exposure, duration of exposure | LC incidence | Significantly elevated risk for ever exposure to silica dust (1.75; 95% CI: 1.16–2.62), welding fumes (1.74; 95% CI: 1.13–2.68), DME (2.18; 95% CI: 1.23–3.84), and MMMF (7.45; 95% CI: 1.63–34.00), significantly reduced risk of LC (OR = 0.67; 95% CI: 0.47–0.95) was linked to ever exposure to cotton dust, ever exposure to asbestos showed no association with LC | Silica dust, welding fumes, DME, MMMF were at a significantly increased risks of LC, while long-term exposure to dust seemed to be protective of LC |

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Med J Islam Repub Iran. 2022 (27 Jul); 36.84.
| First author / Location / Date of publication | Type of assessment | Outcome evaluated | Main results | Conclusion |
|---------------------------------------------|-------------------|------------------|-------------|------------|
| Villeneuve / Canada / 2011                  | Ever exposure     | LC incidence     | DME: Ever exposure (OR = 1.06; 95% CI: 0.89–1.25) and cumulative exposure Lowest (OR=0.93; 95% CI: 0.75–1.17), Middle (OR=1.03; 95% CI: 0.83–1.29), Highest (OR=1.12; 95% CI: 0.89–1.40) were not associated with LC | The findings of this study suggest that exposure to DME may increase the risk of LC |
| Marnette / Central Eastern Europe and UK / 2011 | Ever exposure     | LC incidence     | Arsenic: Ever exposure was associated with an increased LC risk (OR= 1.65;95% CI:1.05–2.58) | Occupational exposure to metals is an important risk factor for LC. |
| Preller / Netherlands / 2010                | Ever exposure     | LC incidence     | Cadmium fumes: highest category of cumulative exposure was associated with LC (OR=2.04; 95% CI: 1.07–3.90). No increased risk was observed for inorganic acid mist, inorganic pigment dust, Chromium, or nickel | Elevated LC risk attributable to crystalline silica exposure |
| Olsen / seven European countries and Liverpool (UK) / 2010 | Ever exposure     | LC incidence     | Silica: Exposure was not associated with LC (RR=1.06; 95% CI: 0.89–1.25) and cumulative exposure Lowest (OR=0.93; 95% CI: 0.75–1.17), Middle (OR=1.03; 95% CI: 0.83–1.29), Highest (OR=1.12; 95% CI: 0.89–1.40) were not associated with LC in the CEE countries. | Occupational PAH exposure may contribute to the burden of LC in some countries |
| Kiran / Czech Republic, France, Germany, Italy, Ireland, and Spain / 2010 | Ever exposure     | LaC incidence    | The OR forever exposure to ethylene oxide and LeC was 2.0 (95% CI= 0.8–4.1), and for medium/high duration of exposure was 6.2(1.3–29.3). Cumulative exposure was not related to LeC | Ethylene oxide is a risk factor for LeC |
| ELCU / Turkey / 2009                        | Cumulative exposure | BC incidence   | An excess of LaC occurred with silica (OR, 1.7; 95%CI: 1.1–3.0) and PAH (OR, 1.5; 95% CI: 1.1–2.2). Other exposures did not show a significant relationship | Several specific chemical agents were significantly associated with the risk of BC |
| Richardson / Canada / 2007                  | Ever exposure     | BC incidence     | Ever exposure to Mineral oils (OR, 1.16; 95%CI, 1.01–1.32), Benz(a)anthracene (OR, 1.92; 95%CI, 1.02–3.61), and DME (OR, 1.18; 95%CI, 1.04–1.35) were associated with BC. Also, cumulative use of DME was related to BC (OR, 1.25; 95%CI, 1.04–1.49) | Occupational exposure to dyes and LaC |
| Richardi / Italy / 2006                     | Duration of exposure | BC incidence    | The OR forever exposure to DME and LC was 1.04 (95% CI: 0.79–1.37), no association was found with cumulative and duration of exposure | NO statistically significant relationship between occupational exposure to PHE and LaC risk. |
| Berrino / South Europe / 2003               | Ever exposure     | LaC incidence    | A positive association between ever exposure to wood dust (OR 1.7, 95% CI: 1.2–2.6), organic solvents (OR:1.7, 95% CI: 1.1–2.5), and asbestos (OR= 1.6;95% CI:1.0–2.5) and LaC was observed. | Occupational exposure to solvents and asbestos was associated with an increased risk of LaC |
| Heinemann / six European countries and covered the period July 1990 to June 1996 / 2000 | Ever exposure     | PLC incidence    | PLC: None of the beryllium, cadmium, Lead, Mercury, and PAHs were not associated with PLC. Although Formaldehyde (OR:3.36, 1.2–9.35) was associated with PLC No association was found for exposure to arsenic, chromium, and PAH | No association was found for exposure to arsenic, chromium, and PAH |
| Rousseau / Canada / 2007                   | Ever exposure     | LC incidence     | Ever exposure to lead was not associated with an increase in the odds of LC(OR:1.4, 95%CI: 0.6–3.2) | Little evidence for an association between lead and LC |
| Hosseini / Iran / 2009                     | Ever exposure     | LC incidence     | Occupational exposures to inorganic dust (OR 4.2, 95% CI: 2.8–6.7), chemical compounds (OR=3.4, 95% CI: 2.1–5.6), and heavy metals (OR 3.0, 95% CI: 1.3–7.0) were all found to be independent risk factors for LC | Inorganic dust, chemical compounds, and heavy metals were associated with LC etiology |

*LC: Lung cancer; +BC: Bladder cancer; +LaC: Laryngeal cancer; +PLC: Primary Liver cancer; +LeC: Leukemia*
The Types of Occupational Carcinogens

One study examined the effects of nickel and/or chromium (56). The effect of diesel motor exhausts (DME) was investigated in 5 studies (27, 33, 37, 40, 47). Silica and/or asbestos (31, 49) polycyclic aromatic hydrocarbons (PAH), and/or aromatic amines (39, 65) were examined in 2 studies. Furthermore, 2 studies examined metalworking fluids (MWFs) (23, 51).

Wood dust and/or formaldehyde (41), arsenic and/or quartz (38), textile dust and/or cotton fibers (24), benzene and/or gasoline (25), iron oxides (21), ethylene oxide (64), lead (35), leather dust (22) chlorinated hydrocarbon solvents (43) vinyl chloride, acrylonitrile and/or styrene (57), and oil mist (21) were examined in a separate study. A study also examined the effects of co-exposure to benzene, toluene, and xylene (BTX) (29).

The effect of asbestos has been studied in 4 studies (32, 44, 48, 54). Other studies investigated the impact of multiple exposures (26, 28, 42, 46, 50, 52, 53, 55, 59-63). All exposures are shown in Table 3.

The Main Outcome Evaluated

Five studies evaluated the mortality of cancer (21, 38, 40, 49, 56), 1 study evaluated the incidence or mortality (50), and other studies considered the incidence (occurrence) of cancer as the outcome (Table 4).

Characteristics and Quality of Studies

Cohort studies

Sciannameo et al (56) who evaluated the 2 outcomes of lung and bladder cancers, had a relatively small sample size but the potential confounders were almost controlled.

Confounders were successfully controlled in the analyses of Liu et al and Offermans et al (44), in addition to the large sample size.

Although the outcome was recorded and collected by the Finnish Cancer Registry (FCR) in the research of Lindbohm et al (42), Siew et al (38), and Lohi et al (43), in addition to having a large sample size and thorough management of confounders, this contributed to minimizing selection bias in these investigations. In the study by Bourgkard et al (21), the sample size was relatively small, but potential confounders, especially socioeconomic status (SES), were largely controlled. Although the study by Taeger et al (38) had a middle sample size, the control of potential confounders was relatively weak, and only exposure to silica was considered a potential confounder.

Case-control Studies

In studies by Sce’Io et al (57), as well as Radoï et al (22), in addition to having a large sample size, potential confounding factors were also well controlled. However, the choice of control in the first study was individually and
population-based, but in the second study, it was frequency matching. Warden et al (29) conducted a study with a relatively high sample size and population control selection. Only 2 variables, smoking and age, were controlled, and other confounding factors were not considered.

In the studies of Latifovic et al (31), Suraya et al (54), Hall et al (63), Ilar et al (47), Barul et al (26), Matrat et al (27), Mannetje (60), Kachuri et al (30), Latifovic et al (36), Villeneuve et al (33), Olsson et al (65), and Kiran et al (64), in addition to large sample sizes, detailed information on lifelong occupational histories was available and the dose-response rate was assessed; also the impact of other occupational carcinogens as potentially confounding agents was controlled.

The studies by Colin et al (23), Pesch et al (39), Talibov et al (58), and Mohner et al (40) were nested case-control studies with large sample sizes. In these studies, potentially confounding factors were collected, cumulative exposure over a lifetime was collected, and the incidence and dose-response could be estimated.

Switkowska et al (48) conducted their case-control study on a cohort of employees with large sample size. Because the exposure was already recorded, the possibility of information bias was minimized; however, only smoking was controlled as a confounding factor and the effect of other possible confounders was not considered.

Khedher et al (24), Barul et al (26), Colt et al (51), Guida et al (28), Richiardi et al (37), and Hosseini et al (55) studies were based on histopathologically confirmed cases, and this prevented the occurrence of selection bias. However, the studies of Richiardi et al (37), as well as Hosseini et al (55), had a smaller sample size than other studies, it seems that even these studies have good statistical power for statistical analysis.

A case-cohort study by Preller et al (45) provided a direct estimation of incidence; however, this study had a large sample size, and potentially confounding factors were well controlled.

Overall, one of the most important limitations of case-control studies is the use of job exposure matrices (JEM) that increased the occurrence of differential misclassification (26).

Main carcinogens, associations, and the strength of associations between common occupational carcinogens and 5 related occupational cancers.

LC

Among the cohort studies, 7 studies examined the effects of occupational carcinogens on LC (21, 38, 41, 44, 49, 50, 56). Also, out of 34 case-control studies, 19 studies related to LC (24, 27, 29, 30, 32, 33, 35, 37, 40, 44, 45, 47, 48, 52, 53).
The most important exposures were asbestos (28, 32, 44, 48, 52-55), silica (28, 30, 45, 49, 52, 53, 55), DME (27, 33, 37, 40, 47, 52, 55), cotton dust (24, 52, 55), benzene (29, 50), PAH (50, 65), wood dust (41, 55), nickel (56, 60), chromium (56, 60), and arsenic (38, 60), respectively. Other exposures each included a study (Table 3).

Except for 2 studies (52, 55), all findings showed a significant association between exposure to asbestos and LaC. Also, the effect of exposure to silica was not shown in 1 study (55). The relationship between occupational exposure to DME was not seen in 2 studies (33, 55), and in 4 other studies, a statistically significant relationship was observed.

In 2 studies (38, 60) conducted to investigate the effect of arsenic, both studies showed a statistically significant relationship. Only 1 of 2 studies on the effect of benzene (29), PAH (65), and nickel (56) was significant. Also, 2 studies (41, 55) conducted to investigate the effect of wood dust and chromium (56, 60) did not show any statistically significant relationship. In 2 studies (24, 52), exposure to cotton dust reduces the risk of LC; however, 1 study showed an increased risk, and this association was not significant (55). Other occupational exposures that elevated the incidence of LC include quartz (38), iron oxide (21), acrylonitrile (57), mineral oil (50), xylene and toluene (29), cadmium fumes (60), and inorganic dust, chemical compounds, and heavy metals (55). The strength of all associations and other results are shown in Table 4.

BC
Among the cohort studies, 4 studies were related to BC (21, 43, 50, 56). There were also 7 case-control studies for occupational carcinogens and BC (23, 31, 34, 36, 39, 51, 59). The most important occupational carcinogens included solvents (43, 50, 59), PAH (39, 50), mineral oils (34, 50), and DME (34, 36). Other occupational exposures are listed in Table 3.

The results of the studies suggest that exposure to solvents in 2 studies (43, 59) increased the risk of BC, although 1 study (50) showed no association. One study (39) also showed an association between PAH and BC, although another study (50) did not show this association.

Two studies (34, 36) showed that DME increases the risk of BC. In 1 study mineral oils (50) did not show a significant relationship with increased risk of BC; however, another study (34) found an increased risk of BC associated with mineral oils.

For other exposures, MWFs (23), aromatic amines (39), Benz(a)anthracene (34), oil mist (21), silica (49), and asbestos (31) increased the risk of BC. Strength of all associations and exposures that showed no association is listed in Table 4.

LaC
The effect of occupational carcinogens on LaC was evaluated in 2 cohort studies (44, 46). Also, 6 case-control studies examined the effect of occupational carcinogens on LaC (22, 25, 26, 44, 53, 61, 63). The most common occupational exposures were asbestos (44, 46, 53, 61), solvent (25, 26, 46, 53), wood dust (46, 53, 61), PAH (53, 61), DME (46, 53), leather dust (22, 53), chromium (61, 63), silica (53, 63), and formaldehyde (53, 61). Other occupational carcinogens are listed in Table 3.

The effect of asbestos on the increased risk of LaC was seen in 4 studies (44, 46, 61, 63); however, no statistically significant relationship was observed in 1 study (53).

Solvents showed a significant relationship with increased risk of LaC only in 1 study (26), and in the other 3 studies, no association was found (25, 46, 53). One study (53) found a significant relationship between PAH and LaC, although this relationship was not significant in another study (61). The 2 studies on the effect of DME on LaC were not statistically significant (46, 53).

One study (61) showed an increased risk of LaC due to occupational exposure to wood dust, but no statistically significant relationship was observed in the other 2 studies (46, 53).

Of the 2 studies investigating the relationship between exposure to leather dust and LaC, only 1 study (22), showed a statistically significant association. One study (63) found an association between chromium and LaC, although no statistically significant association was found in another study (61).

Of the 2 studies to investigate the effect of formaldehyde on LaC, only 1 study (61) showed a significantly increased risk. Exposure to silica in 1 study (63) increased the risk of LaC; however, no significant relationship was observed in another study (53).

Mineral wool (46) and the combination of nickel and chromium (63) increased the risk of LaC. The complete results are shown in Table 4.

LeC
A cohort study (50) and 2 case-control studies (58, 64) examined the effect of occupational carcinogens associated with LeC. The most common exposure was solvents (50, 58). Other exposures included PAH, benzene, mineral oils, and ethylene Oxide (50, 58, 64).

According to Table 4, in 2 studies (50, 58) there was no statistically significant association between exposure to solvents and LeC. Occupational exposure to mineral oils in 1 study (50) increased the risk of LeC, although benzene and PAH were not associated with an increased risk of LeC in this study. Exposure to ethylene oxide (64) shows an increased risk of 1 LeC in exposed individuals. Other results and the strength of the observed associations are presented in Table 4.

PLC
A cohort study (42) and 1 case-control study (62) examined the effect of occupational carcinogens on PLC. The most important exposures that have been evaluated for PLC include solvents (42, 62), beryllium, cadmium, formaldehyde, PAH, lead, mercury (62), and gasoline vapors, aliphatic, and alicyclic hydrocarbons (HC) (42).

Based on the results of Table 4, in 1 study (42), organic solvents (aliphatic and alicyclic) were associated with an increased risk of PLC. However, gasoline vapors were not significantly associated with PLC. Occupational exposure to formaldehyde (62) was significantly associated with an...
increased risk of PLC, but beryllium, cadmium, lead, mercury, and PAHs were not significantly associated with PLC. Table 4 shows the strength of associations observed in studies and other results.

Discussion
Summary of Findings
Our review found 45 papers on occupational exposure to carcinogens and cancer risk. The findings of this review strongly suggest that occupational exposures were associated with an increased risk of LC, BC, LaC, LeC, and PLC. The present review also appears to be the first study based on 5 common occupational cancers.

In this review, of the 7 cohort studies that assessed the effects of occupational carcinogens on LC, 6 studies showed a positive association between occupational exposure and LC. In addition, 3 out of 4 cohort studies on BC were associated with an increased risk of BC. In the other 3 cohort studies, the incidence of LaC, LeC, and PLC increased because of carcinogenic exposures.

Of the 19 case-control studies evaluating the effect of occupational carcinogens on LC, only 5 studies showed no statistically significant relationship. Also, all 7 case-control studies that examined the effect of occupational exposure on BC showed a statistically significant increase in the risk of BC, and this suggests that occupational carcinogen exposures strongly influence this cancer. Also, of 5 case-control studies related to occupational carcinogens and LaC, 4 studies were significant. One out of 2 case-control studies related to occupational carcinogens and LeC showed a statistically significant relationship. There were 2 case-control studies on occupational carcinogens and PLC, one of which showed an association with these carcinogens.

Interpretation Concerning Other Literature
According to previous studies, LC was the main cancer attributed to occupational exposure, followed by BC (9, 15). Among the studied exposures in this study, asbestos, silica, DME, benzene, formaldehyde, and PAH were ranked first to fifth, respectively. Previous studies have shown that crystalline silica, DME, wood dust, formaldehyde, benzene, solvents, and asbestos are the most common occupational exposures (3).

This review provides critical information for selecting carcinogenic occupational exposures and risk estimates; it is the first step in estimating the cancer burden associated with various exposures through nationwide studies, however, the methodologies, statistical approaches, and confounders used in the research evaluated were all highly diverse, which explains some of the differences in the results.

The present review study showed that developing countries have the highest exposure to occupational carcinogens but have the least published studies. Most studies were conducted in developed countries, especially in Western countries. Also, studies conducted in low- and middle-income countries had a poor methodology. Most of these studies were conducted by local authorities or by small industries. Their main goal was not to estimate the cancer burden resulting from these occupational exposures (66, 67). On the other hand, few studies conducted in developing countries have incomplete reports, and the force of association has not been well demonstrated. Even studies conducted in developed countries have high heterogeneity in controlling potential confounders and reporting other influencing factors. This heterogeneity has reduced the ability to pool the results of these studies.

Also, the present review found that a single occupational carcinogen may be linked to multiple cancer sites, and a single cancer site may be linked to multiple occupational carcinogens. According to this, in the future, the less developed countries are expected to focus more on designing studies with a stronger methodology, emphasizing common occupational carcinogens in the industries in these countries. It is also recommended that more emphasis be placed on occupational carcinogens approved by the IARC in these countries. Furthermore, policymakers should evaluate the possibility of occupational carcinogens being related to cancer sites, as even minimal exposure to some of these agents’ increases cancer risk significantly (15).

Evidence Gaps and Implications for Future Surveys
Interviews and self-reporting of jobs and/or occupational exposures were used to acquire occupational information in case-control studies, and no effort was made to assign occupational exposures. Some of the case-control studies had problems in their design (eg, choice of controls, potential confounding, and power) that limited the interpretation of the results (14). According to this, it is recommended that in the future in developed and high-income countries, where the registration and quality of occupational carcinogens are higher than the registration and quality of information collected in developing countries, the emphasis be on conducting studies with stronger methodologies, including historical cohorts with higher sample sizes.

Strengths and Limitations
Overall, almost all of the studies included in this review were methodologically strong, and the few weaknesses of these studies did not affect the outcome evaluation of these studies. RRs, ORs, HRs, prevalence, and type of exposure were needed to estimate the burden attributed to cancers. The present study, which focused on 5 occupational malignancies and identified the key carcinogens linked to these cancers, appears to be a suitable reference for future studies estimating the burden of occupational cancers. The absence of carcinogenic exposures described in non-cancer research is this review’s major limitation. Furthermore, researches that were not published in studies indexed by the searched databases may have been overlooked. Some investigations were not sufficiently comprehensive to obtain all essential data. “Questionnaires evaluated occupational exposures,” for example, although the sort of exposure was not specified.

Conclusion
The findings of this study revealed that cancers caused by industrial chemical exposures place a significant financial burden on developed and developing countries alike.
Furthermore, occupational carcinogens of asbestos, benzene, crystalline silica, PAH, and DME were among the most common exposures associated with the 5 known occupational cancers (LC, BC, LaC, LeC, and PLC).

The present review also found that although the number of published studies related to occupational carcinogens is high, the majority of these researches have been performed in both high-income and low-income nations. The number of research has been quite low in areas where these exposures are significantly more common. In the future, more high-quality research should be undertaken in developing countries, with a focus on approved occupational cancers. In developed countries, where occupational exposures and malignancies are well documented and collected, historical cohort studies should be conducted.

**Ethics Approval**

The ethics committee of Kerman University of Medical Sciences (KUMS) approved this study with ID number IR.KMU.REC.1399.407.

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**Conflict of Interests**

The authors declare that they have no competing interests.

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