ELSEVIER

Critical Reviews in Oncology/Hematology 90 (2014) 99–134

CRITICAL REVIEWS IN
Oncology
Hematology
Incorporating Geriatric Oncology

www.elsevier.com/locate/critrevonc

Occupational exposures in rare cancers: A critical review of the literature

B. Charbotel [a,b,c,*,1], B. Fervers [d,a,e,1], J.P. Droz [d,a,e]

[a] *Université de Lyon, F-69003 Lyon, France*
[b] *Université Lyon 1, UMRESTTE (Unité mixte IFSTTAR/UCBL), Domaine Rockefeller, 69373 Lyon, France*
[c] *Hospices Civils de Lyon, Service des Maladies Professionnelles, Centre Hospitalier Lyon Sud, 69495 Pierre Bénite, France*
[d] *Unité Cancer et Environnement, Centre Léon Bérard, 28 rue Laënnec, 69373 Lyon Cedex 08, France*
[e] *Université Lyon 1, Faculté Lyon Est, F-69008 Lyon, France*

Accepted 4 December 2013

Contents

* Corresponding author at: Université Lyon 1, UMRESTTE (Unité mixte IFSTTAR/UCBL), Domaine Rockefeller, 69373 Lyon, France. Tel.: +33 4 78772827.
E-mail address: barbara.charbotel@univ-lyon1.fr (B. Charbotel).
[1] These authors contributed equally to this work.

http://dx.doi.org/10.1016/j.critrevonc.2013.12.004

Abstract

The contribution of occupational exposures to rare cancers, which represent 22% of all cancers diagnosed annually in Europe, remains insufficiently considered. We conducted a comprehensive review of occupational risk factors in 67 rare cancers (annual incidence <6/100,000). An examination of relevant articles in PubMed (1960–2012) and the International Agency for Research on Cancer (IARC) monographs revealed that 26 cancer sites, such as mesothelioma, nasal, larynx, liver, ovarian cancer, bone sarcoma, and hematopoietic malignancies were consistently linked to occupational factors. Main exposures included asbestos, wood dust, metals/metalloids, formaldehyde, benzene, vinyl chloride, and radiation. There was inconsistent evidence regarding 22 rare malignancies. We did not identify relevant data for 19 rare cancers. Despite limitations of published evidence, our review provides useful information that can facilitate the identification of work-related factors that contribute to rare cancers. International collaborations, development of improved exposure assessment methods, and molecular approaches can improve future studies.

Keywords: Rare cancer; Occupational; Exposure; Epidemiology; IARC; Classification

1. Introduction

Despite their low occurrence, more than 500,000 rare cancers are diagnosed each year in Europe, corresponding to 22% of all cancers annually and one quarter of the total cancer prevalence [1]. Several rare malignancies are now well known to be caused by occupational factors, such as pleural mesothelioma in asbestos workers [2,3], nasopharyngeal carcinoma in wood workers [4] or liver angiosarcoma in vinyl chloride workers [5]. However, occupational exposures in many rare cancers remain under-reported due to lack of evidence and/or awareness by clinicians. Therefore, our article aimed to review the available evidence from epidemiology studies and IARC monographs to provide a synthesis of established and suspected occupational exposures associated with rare cancers.

2. Methods

We only considered here rare cancers, i.e., those with an incidence of <6 per 100,000 cases per year, according to RARECARE [1]. We searched PubMed from 1960 to December 2012, using search terms related to occupational exposure and specific cancer sites (Table 1). We included articles in English, French, German or Spanish. Overall, we identified 6820 articles. Possibly relevant articles were selected through assessment of titles and abstracts and through the reference lists of related articles. We further searched the International Agency for Research on Cancer (IARC) monographs (http://monographs.iarc.fr/). Studies were selected according to the following criteria: study design (cohort or case–control study); original study or meta-analyses; studies providing histology to differentiate between subtypes; studies providing information on association between cancer risk (effect size) and occupational exposure (i.e., included odds ratio [OR], relative risk [RR], standardized incidence ratio [SIR], or mortality rate ratio [SMR]). We selected in priority studies with detailed occupational exposure assessment, rather than studies based solely on job titles. When available, we preferentially selected meta-analyses. Only associations supported by consistent evidence from several studies were recorded (Tables 2 and 3). Also, when an association was consistently supported by numerous studies, the most recent data were retained. Finally, 187 articles and relevant data from IARC monographs were included (Tables 2 and 3).

3. Results

We included 67 rare cancers in our review. For 26 cancers (marked with an asterisk (*)), an association with occupational exposure was supported by IARC monographs and/or consistent evidence from epidemiology studies (Tables 2 and 3). We distinguished for each cancer site, occupational exposures classified by IARC with "*sufficient evidence*" for carcinogenicity (i.e. a causal relationship has been established between exposure to the agent and the given cancer type) or "*limited evidence*" for carcinogenicity (i.e. a positive association for which a causal interpretation is considered to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence) [6] as well as occupational exposures supported by consistent epidemiology evidence. For 22 rare malignancies (marked with two asterisks (**)), there was inconsistent or insufficient evidence (Table 3). Finally, we did not identify relevant occupational exposure studies for 19 cancers (Table 3). Some histology subtypes or rare cancers are covered by the IARC classification for a larger cancer entity. This is specified in the text and tables (Tables 2 and 3). Unless otherwise stated for a cancer site, no IARC classification exists.

3.1. Head and neck cancers

3.1.1. Epithelial tumors of the nasal cavity and sinuses*

IARC has classified several occupational exposures as carcinogens for nasal cavity and sinuses with sufficient evidence (isopropyl alcohol production, leather dust, nickel compounds, radium, wood dust) or limited evidence (carpentry and joinery, chromium (VI) compounds, formaldehyde, textile manufacturing). Epidemiological studies have consistently associated cancer of the nasal cavity with wood dust, leather dust, nickel and radium [4,7–10]. Epidemiological evidence remains insufficient for other agents including chromium, arsenic, formaldehyde and welding fumes [7,11–13]. Adenocarcinomas has been associated with wood dust, leather dust, and formaldehyde [7,13], whereas squamous cell carcinomas has been recently linked to arsenic and welding fumes [7].

3.1.2. Epithelial tumors of the nasopharynx*

IARC has classified wood dust and formaldehyde as carcinogenic for nasopharynx with sufficient evidence [14–17]. Also, nasopharyngeal carcinoma has been associated with chlorophenol exposure, mainly for machinists [18], as well as industrial heat [17].

3.1.3. Epithelial tumors of the major salivary glands**

Although few studies have focused on salivary gland cancer, occupational exposure to either ionizing radiation or formaldehyde have been linked to this cancer type [19].

3.1.4. Epithelial tumors of the hypopharynx*

Few studies have considered the hypopharynx separately from pharyngeal cancer. IARC has classified asbestos exposure as carcinogenic for pharynx with limited evidence. A twofold increase in hypopharyngeal cancer risk was identified for asbestos exposure [20]. Increased risk has also been reported for exposure to iron and steel [21].

Table 1
Search strategy and number of paper identified by cancer site.

Cancer type	Search strategy	Number of references identified
Search terms related to occupational exposure:	((occupation[Tiab] OR occupational[Tiab] OR work[Tiab] OR worker*[Tiab]) AND (exposure[Tiab] OR exposures[Tiab] OR exposed[Tiab]) AND (English[lang] OR French[lang] OR German[lang] OR Spanish[lang])).	
Search terms related to specific cancer sites:	Adapted from the National Cancer Institute: http://www.cancer.gov/cancertopics/litsearch	
Head and neck cancers		
Cancer of the nasal cavity and sinuses	(nose neoplasm*[Tiab] OR ((nose[Tiab] OR nasal[Tiab] OR sinonasal[Tiab] OR paranasal[Tiab] OR sinus[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	403
Naso-pharyngeal cancer (NPC)	(nasopharynx[Tiab] OR nasopharyngeal[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])	87
Cancers of the salivary gland	(salivary gland neoplasm*[Tiab]) OR ((salivary[Tiab] OR parotid[Tiab] OR sublingual[Tiab] OR submandibular[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignant[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	30
Hypo-pharyngeal cancer	(hypopharyngeal neoplasms[Tiab]) OR ((hypopharyn*[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	17
Laryngeal cancer	(laryngeal neoplasms[Tiab] OR ((laryngeal[Tiab] OR larynx[Tiab] OR glottis[Tiab] OR glottic[Tiab] OR subglottis[Tiab] OR subglottic[Tiab] OR supraglottis[Tiab] OR supraglottic[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	276
Pharyngeal cancer	pharyngeal neoplasms[Tiab]) OR ((pharyn*[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	118
Oropharyngeal cancer	oropharyngeal neoplasms[Tiab]) OR ((oropharyn*[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	25
Cancer of the oral cavity	(oral[Tiab] OR mouth[Tiab] OR lip[Tiab] OR gingiva[Tiab] OR gingival[Tiab] OR tongue[Tiab] OR palate[Tiab] OR palatal[Tiab] OR buccal[Tiab]) AND (cancer*[Tiab] OR	405
Cancer of the lip	carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])	
Gastrointestinal cancers		
Squamous cell carcinoma of the esophagus	(squamous cell[Tiab] AND (oesophag*[Tiab] OR esophagi*[Tiab])) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	8
Adenocarcinoma of the esophagus	(adenocarcinoma*[Tiab] AND (esophag*[Tiab] OR oesophag*[Tiab])) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	15
Carcinoma of the small intestine	((duodenal neoplasm*[Tiab] OR ileal neoplasm*[Tiab] OR jejunal neoplasm*[Tiab])) OR (((small[Tiab] AND (intestine*[Tiab] OR intestinal[Tiab] OR bowel*[Tiab])) OR (duodenal[Tiab] OR duodenum[Tiab] OR ileal[Tiab] OR ileum[Tiab] OR jejunal[Tiab] OR jejunum[Tiab])) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	51
Cancer of the anal canal	((anus neoplasms[Tiab]) OR ((anal[Tiab] OR anus[Tiab] OR perianal[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	7
Hepatocellular carcinoma of the liver and intrahepatic bile tract (IBT)	((liver neoplasm*[Tiab] OR cholangiocarcinoma[Tiab] OR hepatocellular carcinoma*[Tiab] OR hepatoblastoma*[Tiab] OR hepatoma*[Tiab]) OR ((liver*[Tiab] OR intrahepatic[Tiab] OR hepatic[Tiab] OR hepatocellular[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	853
Angiosarcoma	((liver[Tiab] OR hepatic[Tiab]) AND angiosarcoma[Tiab])	107
Epithelial tumors of gallbladder and extrahepatic biliary tract (EBT)	gallbladder[Tiab] OR extrahepatic[Tiab]	36
Thoracic cancers		
Epithelial tumor of the trachea	((trachea[Tiab]) OR tracheal[Tiab])) AND (((cancer*[Tiab]) OR tumor*[Tiab]) OR tumor*[Tiab])	41
Large cell lung carcinoma	(((large[Tiab]) AND cell[Tiab]) AND lung[Tiab]) AND ((carcinoma*[Tiab]) OR cancer*[Tiab])	60

Table 1 (*Continued*)

Cancer type	Search strategy	Number of references identified
Bronchiolo-alveolar lung carcinoma	((bronchiolo-alveolar[Tiab]) AND lung[Tiab]) AND (carcinoma*[Tiab] OR cancer*[Tiab])	2
Epithelial tumors of the thymus	AND (thymoma[Tiab] OR (thymus[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])))	34
Mesothelioma	(mesothelioma*[Tiab])	1007
Reproductive cancers		
Mammary Paget's Disease	((mammary[Tiab]) AND paget's[Tiab]) AND disease[Tiab])	0
Epithelial tumors of the male breast	(male[Tiab] AND (breast neoplasms[Tiab] OR dcis[Tiab] OR lcis[Tiab] OR ((breast*[Tiab] OR mammary[Tiab] OR nipple*[Tiab]) AND ((cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]) OR in situ[Tiab])))	95
Epithelial tumors of the cervix uteri	(cervix neoplasms[Tiab]) OR((cervix[Tiab] OR cervical[Tiab] OR exocervix[Tiab] OR exocervical[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	144
Ovarian cancer	(ovarian neoplasm*[Tiab]) OR ((ovarian[Tiab] OR ovary[Tiab] OR ovaries[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	170
Carcinoma of the vulva and vagina	(((vaginal neoplasm*[Tiab] OR vulvar neoplasm*[Tiab]) OR ((vagina [Tiab] OR vulva[Tiab] OR (bartholin[Tiab] AND gland[Tiab])) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))))	8
Urogenital cancers		
Testicular cancer	(testicular neoplasms[majr] AND human[mh] AND english[la]) OR ((testicular[Tiab] OR testis[Tiab] OR testicle*[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])	152
Extragonadal germ cell tumors	(neoplasms, germ cell and embryonal[MeSH Terms] OR (germcell[Tiab] OR germ cell[Tiab] OR dysgerminoma[Tiab] OR extragonadal[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	296
Penis carcinoma	(penile neoplasms[majr] AND human[mh]) [la]) OR ((penile[Tiab] OR penis[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	5
Squamous cell carcinoma of the kidney	squamous[Tiab] AND cell[Tiab] AND kidney AND (adenocarcinoma*[Tiab] OR carcinoma*[Tiab])	10
Non-transitional cell carcinoma of the urinary bladder	((bladder neoplasms[majr] OR (bladder[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])) AND (non-transitional[Tiab] OR squamous[Tiab] OR adenocarcinoma*[Tiab] OR sub-type[Tiab] OR histolog*[Tiab]))	54
Non-bladder urinary organs (renal pelvis, ureter, urethra)	((urologic neoplasms[majr] AND Carcinoma, Transitional Cell[majr]]) OR ((urologic[Tiab] OR urinary[Tiab] OR urothelial[Tiab] OR urethra*[Tiab] OR paraurethra*[Tiab] OR ureter[Tiab] OR (renal[Tiab] AND pelvis[Tiab])) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]) AND (TCC[Tiab] OR (transitional[Tiab] AND cell[Tiab]))))	76
Neuroectodermic and mesodermic tumors		
Malignant melanoma of mucosa	(melanoma[Tiab]) AND mucosa[Tiab])	2
Epithelial tumors of the eye and adnexa	(((cancer*[Tiab]) OR tumor*[Tiab]) OR tumor*[Tiab]) AND ((eye[Tiab]) OR adnexa[Tiab])	81
Malignant melanoma of the uvea	(melanoma*[Tiab] AND eye[Tiab])	29
Soft-tissue sarcoma	(soft-tissue sarcoma*[Tiab])	90
Bone sarcoma	(bone[Tiab]) AND sarcoma*[Tiab]	27
Glial tumors of the central nervous system	Glioma*[Tiab] OR (glial*[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab]))	113
Malignant meningioma	Meningioma*[Tiab] AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab])	26
Neuroendocrine tumors	Neuroendocrine[Tiab]) AND ((cancer*[Tiab] OR carcinoma*[Tiab] OR malignant OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab]))	9
Endocrine tumors		
Carcinoma of the pituitary gland	((pituitary neoplasms[Tiab] OR (pituitary[Tiab] AND (tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab] OR malignan*[Tiab]))	34

Table 1 (*Continued*)

Cancer type	Search strategy	Number of references identified
Carcinoma of the thyroid gland	thyroid neoplasm*[Tiab] OR (thyroid[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab])	196
Carcinoma of the parathyroid gland	((parathyroid neoplasms[Tiab] OR parathyroid AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab]))	10
Carcinoma of the adrenal gland	adrenal cortical carcinoma[Tiab] OR ((adrenocortical[Tiab] OR adrenal[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab]))	28
Lymphoid diseases		
Hodgkin disease	((hodgkin disease[majr] AND human[mh]) OR (((hodgkin[Tiab] OR hodgkins[Tiab]) NOT (non-hodgkin[ti] OR non-hodgkins[ti])) AND (lymphoma[Tiab] OR lymphomas[Tiab] OR disease[Tiab])))	35
Acute lymphoblastic leukemia	((lymphoblastic leukemia, acute[majr] AND human[mh]) OR (acute lymphoblastic leukemia[tiab] OR acute lymphocytic leukemia[tiab]))	63
Burkitt lymphoma	((b-cell lymphoma[majr] AND human[mh]) OR (Burkitt[Tiab] AND (lymphoma[Tiab] OR lymphomas[Tiab])))	22
Cutaneus T cell lymphoma/mycosis fungoides	((cutaneous t-cell lymphoma[majr] AND human[mh]]) OR CTCL[Tiab] OR (mycosis[Tiab] AND fungoides[ti]) OR ((cutaneous[Tiab] OR skin[Tiab]) AND t-cell[Tiab] AND (lymphoma[Tiab] OR lymphomas[Tiab])))	22
Other T cell lymphoma and NK cell neoplasms	((t-cell lymphoma[majr] AND human[mh]) OR ((t-cell[Tiab] OR (t[Tiab] AND lymphoblastic[Tiab]) OR NK-cell[Tiab] OR anaplastic large cell[Tiab] OR angioimmunoblastic[Tiab] OR sezary syndrome[Tiab] OR angiocentric[ti]) AND (lymphoma[Tiab] OR lymphomas[Tiab])))	53
Diffuse large B-cell lymphoma (DLBCL)	((b-cell lymphoma[majr] AND human[mh]) OR ((b-cell[Tiab] OR large-cell[Tiab] OR diffuse well-differentiated lymphocytic[Tiab] OR large-b-cell [Tiab]) AND (lymphoma[Tiab] OR lymphomas[Tiab])))	58
Follicular B lymphoma (FL)	((b-cell lymphoma[majr] AND human[mh]) OR (follicular[Tiab] AND (lymphoma[Tiab] OR lymphomas[Tiab])))	37
Hair cell leukemia	((hairy cell leukemia[majr] AND human[mh]]) OR (hairy[Tiab] AND cell[Tiab] AND (leukemia[Tiab] OR leukemias[Tiab] OR leukemia[Tiab] OR leukaemias[Tiab])) OR ((leukemic[Tiab] OR leukaemic[Tiab]))	87
Multiple myeloma (MM)	((plasmacytoma[majr] AND human[mh]) OR MGUS[Tiab] OR plasmacytoma[Tiab] OR plasmacytomas[Tiab] OR multiple myeloma[Tiab] OR multiple myelomas[Tiab] OR plasma cell neoplasm[Tiab] OR plasma cell neoplasms[Tiab])	205
Other non-Hodgkin, mature B cell lymphoma		
Chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL)	((lymphocytic leukemia, chronic[majr] AND human[mh]) OR CLL[Tiab] OR mu heavy chain disease[Tiab] OR mu-chain disease[Tiab] OR ((chronic[Tiab] OR b-cell[Tiab]) AND (lymphocytic[Tiab] OR lymphoid[Tiab] OR lymphogenous[Tiab] OR aleukemic[Tiab] OR aleukaemic[Tiab]) AND ((leukemia[Tiab] OR leukemias[Tiab] OR leukemia[Tiab] OR leukaemias[Tiab]) OR disorders[Tiab])))	136
Marginal zone lymphoma/MALT lymphoma	((b-cell lymphoma[majr] AND human[mh]) OR ((MALT[Tiab] OR marginal zone[Tiab]) AND (lymphoma[Tiab] OR lymphomas[Tiab])))	18
Mantle cell lymphoma	((b-cell lymphoma[majr] AND human[mh]) OR (mantle cell[Tiab] AND (lymphoma[Tiab] OR lymphomas[Tiab])))	17
Lymphoplasma-citic lymphoma/macroglobulinemia Waldestrom	((b-cell lymphoma[majr] AND human[mh]) OR ((lymphoplasmacytic[Tiab] OR waldenstrom[Tiab] OR macroglobulinemia[Tiab]) AND (lymphoma[Tiab] OR lymphomas[Tiab])))	21
Acute myeloid leukemia	((myeloid leukemia, acute[majr] AND human[mh]) OR (AML[ti] OR acute myeloid leukemia[ti] OR acute myelogenous leukemia[ti] OR acute myeloblastic leukemia[ti]))	167
Chronic myeloid leukemia	((myelogenous leukemia, chronic[majr] AND human[mh]) OR (CML[Tiab] OR (chronic[Tiab] AND (myeloid[Tiab] OR myelogenous[Tiab] OR granulocytic[Tiab]) AND (leukemia[Tiab] OR leukemias[Tiab] OR leukemia[Tiab] OR leukaemias[Tiab] OR disorders[Tiab]))))	90
Other myelodysplastic and myeloproliferative neoplasms	("myelodysplastic myeloproliferative diseases"[MeSH Major Topic] AND human[mh]) OR ((myelodysplastic [Tiab] OR myeloproliferative [Tiab]) AND (leukemia[Tiab] OR leukemias[Tiab] OR leukemia[Tiab] OR leukaemias[Tiab] OR disorders[Tiab]))	46
Histiocytic and dendritic cell neoplasms	(histiocytic[Tiab] OR dendritic[Tiab]) AND (neoplasm[Tiab] OR neoplasms[Tiab] OR sarcoma[Tiab] OR sarcomas[Tiab] OR lymphoma[Tiab] OR lymphomas[Tiab])	6
Childhood cancer	parental[Tiab] OR maternal[Tiab] OR paternal[Tiab] OR childhood[Tiab] OR offspring[Tiab] OR prenatal[Tiab]) AND (cancer*[Tiab] OR carcinoma*[Tiab] OR adenocarcinoma*[Tiab] OR malignan*[Tiab] OR tumor*[Tiab] OR tumor*[Tiab] OR neoplasm*[Tiab] OR leukemia[Tiab] OR lymphoma[Tiab])	500

Table 2
Associations supported by consistent evidence and relevant IARC classifications by cancer site.

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Head and neck cancers			
Cancer of the nasal cavity and sinuses	Wood dust	**d'Errico et al.** [7]: Hospital based case control study (113 cases, 336 controls) OR for adenocarcinoma: 58.6 (23.74–144.8), adjusted for age and sex **Demers et al.** [4]: data from 12 case control studies (680 male cases, 2349 male controls, 250 female cases, and 787 female controls) Adenocarcinoma among men: OR = 3.1 (1.6–6.1) for moderate exposure and 45.5 (28.3–72.9) for high wood dust, adjusted for study and age Little evidence for squamous cell carcinoma	Group 1. Sufficient evidence for this specific site. (Vol. 100 C, 2012)
	Nickel	**Grimsrud et al.** [8]: Cohort study among workers employed at a nickel refinery (2 cases, 0.23 expected) SMR = 870 (105–3141)	Group 1. Sufficient evidence for this specific site. (Vol. 100 C, 2012)
	Leather dust	**d'Errico et al.** [7]: Hospital based case control study (113 cases, 336 controls) OR for adenocarcinoma: 26.6 (5.09–139.0) age and sex adjusted and 32.8 (5.96–181.1) when including wood dust exposure in the model	Group 1. Sufficient evidence for this specific site. (Vol. 100 C, 2012)
	Formaldehyde	**d'Errico et al.** [7]: Hospital based case control study (113 cases, 336 controls) OR for adenocarcinoma 9.5 (2.62–34.20) age and sex adjusted **Luce et al.** [13]: Pooled analysis of 12 studies (930 cases 3136 controls) from seven countries OR for adenocarcinoma in men: 3.0 (1.5–5.7) in the high level of exposure category, adjusted for age, exposure to wood dust and leather dust OR for adenocarcinoma in women 6.2 (2.0–19.7) in the high level of exposure category, adjusted for age	Group 1. Limited evidence for this specific site. (Vol. 100 F, 2012)
	Chromium	**Rosenman et al.** [11]: Cohort study among 3408 workers from facilities producing chromium compounds. 6 cases of sino-nasal cancers were observed. PMR = 6.85 (3.14–14.94) in white men. No adjustment. **Hernberg et al.** [12]: Case control study (167 cases and 167 controls) from Denmark, Finland or Sweden. OR = 2.7 (1.1–2.6). No adjustment.	Group 1. Limited evidence for this specific site. (Vol. 100 C, 2012)
	Arsenic	**d'Errico et al.** [7]: Hospital based case control study (113 cases, 336 controls) OR for squamous cell carcinoma: 5.2 (1.20–22.20) age and sex adjusted	Group 1. No evidence for this specific site. (Vol. 100 C, 2012)
	Welding fumes	**d'Errico et al.** [7]: OR for squamous cell carcinoma: 4.1 (1.66–10.13) age and sex adjusted	Group 2B. (Vol. 49, 1990)
Nasopharyngeal cancer (NPC)	Formaldehyde	**Bachand et al.** [14]: Meta-analysis on case control studies Overall OR = 1.22 (1.00–1.50), based on 6 case control studies Overall OR adjusted for smoking = 1.10 (0.8–1.51) based on 6 case control studies **Bosetti et al.** [15]: Meta-analysis on 12 cohort studies Meta-SMR = 1.33 (0.61–2.53)	Group 1. Sufficient evidence for this specific site. (Vol. 100 F, 2012)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Wood dust	**Hildesheim et al.** [16]: OR for wood dust any level: 1.7 (1.0–3.0) adjusted for age, sex, education, and ethnicity >10 years OR = 2.4 (1.1–5.0); *p* for trend: 0.02; adjusted for age, sex, education, ethnicity **Armstrong et al.** [17]: case control study 282 cases with squamous cell nasopharyngeal carcinoma, 282 controls OR = 2.36 (1.33–4.19) adjusted for diet and cigarette smoke	Group 1. Sufficient evidence for this specific site. (Vol. 100 C, 2012)
	Chlorophenol	**Mirabelli et al.** [18]: 92 nasopharyngeal carcinoma cases from cancer registries and 1909 controls obtained through random digit dialing OR = 1.94 (1.03–3.50) among those with medium or high intensity chlorophenol exposure, adjusted for registry, age group, Asian ethnicity, and smoking status	Group 2B. (Vol. 71, 1999)
	Industrial heat	**Armstrong et al.** [17]: OR = 2.21 (1.12–4.33) adjusted for diet and cigarette smoke	No IARC classification for this exposure.
Cancer of the salivary gland	Ionizing radiations	**Wilson et al.** [19]: Death certificate-based case control study on salivary gland cancer. African American (168 cases, 672 controls) and white (2237 cases, 8748 controls) cases from 24 states (1984–1989) matched to controls by age, sex, race, and region. Job exposure matrix OR = 1.7 (1.05–2.80) in white men exposed to mid-high probability and intensity adjusted for age, marital and social status	Group 1. Sufficient evidence for this specific site but not from occupational exposure contexts. (Vol. 100 D, 2012)
	Formaldehyde	**Wilson et al.** [19]: OR = 1.6 (1.30–2.00) in white men exposed to mid-high probability and intensity adjusted for age, marital and social status	Group 1. No evidence for this specific site. (Vol. 100 F, 2012)
Hypo-pharyngeal cancer	Asbestos	**Marchand et al.** [20]: Hospital-based case control (206 hypopharyngeal cancer cases/305 controls with other types of cancer Ever exposed OR = 1.80 (1.08–2.99) adjusted for age, smoking, and alcohol consumption Highest level of exposure OR = 2.14 (1.14–4.01) adjusted for age, smoking, and alcohol consumption	Group 1. Limited evidence for pharynx. (Vol. 100 C, 2012)
	Mild steel dust	**Shangina et al.** [21]: Hospital controls based case control study (350 cases/34 hypopharyngeal cancer and 728) OR = 3.04 (1.39–6.64) adjusted for age, country, tobacco smoking, and alcohol consumption Dose–response relationship with cumulative exposure (*p* for trend 0.006)	Iron and steel founding (occupational exposure during) classified Group 1. No evidence for this site. (Vol. 100 F, 2012)
	Iron compounds and fumes	**Shangina et al.** [21]: OR = 2.74 (1.29–5.84) adjusted for age, country, tobacco smoking, and alcohol consumption Dose-response relationship with exposure duration (*p* for trend 0.03)	
Laryngeal cancer	Asbestos	**Goodman et al.** [24]: Meta-analysis based on 69 asbestos-exposed occupational cohorts Meta-SMR = 133 (114–155) **IOM** [25]: Meta-analysis of 15 cohort studies Any exposure overall relative risk: 1.4 (1.19–1.64) High exposure overall relative risk: 2.02 (1.64–2.47)	Group 1. Sufficient evidence for this specific site. (Vol. 100 C, 2012)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Acid mists	**Steenland** [23]: Cohort of 1156 male steelworkers SIR = 2.2 (1.2–3.7) adjusted on tobacco smoking	Group 1. Sufficient evidence for this specific site. (Vol. 100 F, 2012)
	Silica dust	**Chen et al.** [26]: Meta-analysis *Case control studies* ($n = 6$): pooled OR = 1.39 (1.17–1.67) adjustment for smoking and alcohol consumption *Cohort studies silicosis cases* ($n = 5$): pooled SMR = 1.38 (0.79–1.96) *Silica dust exposed worker*: pooled SMR = 1.13 (0.82–1.45) based on six studies Pooled SIR = 1.50 (0.59–2.42) based on three studies	Group 1. No evidence for this specific site. (Vol. 100 C, 2012)
	Engine exhaust	**Paget-Bally et al.** [27]: Meta-analysis for agents with at least 10 available studies with homogenous exposure. 99 publications analyzed Meta-RR = 1.17 (1.05–1.30)	Engine exhaust, diesel Group 1. No evidence for this specific site. (Vol. 105, in prep, meeting in 2012)
	Polycyclic aromatic hydrocarbons (PAH)	**Paget-Bally et al.** [27]: Meta-analysis for agents with at least 10 available studies with homogenous exposure. 99 publications analyzed Meta-RR = 1.29 (1.10–1.52)	Some PAH-related exposures are Group 1 but none of them for this site of cancer. (Vol. 100 F, 2012)
	Chlorinated solvents	**Shangina et al.** [21]: Hospital based case control study (350 cases/316 laryngeal cancer cases and 728 controls) OR = 2.18 (1.03–4.61) adjusted for age, country, tobacco smoking, and alcohol consumption	No classification for this site.
	Passive smoking at work	**Lee et al.** [28]: Case control study 542 cases and 2197 controls who reported never using tobacco Duration of exposure > 15 years OR = 2.07 (1.04–4.11) adjusted for centers, age, sex, race/ethnicity, education, and alcohol drinking Duration of exposure >15 years, never alcohol users OR = 5.45 (1.69–17.52)	Second-hand smoke Group 1. Limited evidence for this site. (Vol. 100 E, 2012)
	Ionizing radiation	**Dupree et al.** [29]: Retrospective cohort mortality study of 995 white males employed at a uranium processing facility (5 larynx cancer cases) SMR = 447 (144–1043) no adjustment	Group 1. No evidence for this specific site. (Vol. 100 D, 2012)
	Textile dust	**Paget Bally et al.** [27]: Meta-analysis for agents with at least 10 available studies with homogenous exposure (99 publications analyzed) Meta-RR = 1.41 (1.09–1.83)	Textile manufacturing industry (work in) is Group 2B. (Vol. 48, 1990)
	Electromagnetic field low-frequency (ELF)	**Floderus et al.** [30]: Swedish cohort study on 1,596,959 men and 806,278 women In men exposed to medium level (0.084–0.115 μT) RR = 1.5 (1.2–1.9) adjusted for age In men exposed to high level (≥0.116 μT) RR = 1.6 (1.3–2.0) adjusted for age	Group 2B. (Vol. 80, 2002)
	Sulfur mustard	**Easton et al.** [31]: Cohort of 2498 men and 1032 women employed in a manufacture of mustard gas SMR = 273, $p < 0.001$ (no adjustment)	Group 1. Limited evidence for this site. (Vol. 100 F, 2012)
Pharyngeal cancer	Welding fumes	**Gustavsson et al.** [32]: Community based case referent study (545 cases and 641 referents) More than 8 years of exposure: OR = 2.3 (1.1–4.7) accounting for age, region, alcohol consumption and tobacco smoking	Welding fumes classified group 2B. No classification for this site. (Vol. 49, 1990)
	Sulfur mustard	**Easton et al.** [31]: Cohort of 2498 men and 1032 women employed in a manufacture of mustard gas SMR = 549, $p < 0.001$ (no adjustment)	Group 1. No evidence for this site. (Vol. 100 F, 2012)
Cancer of the oral cavity	Wood dust	**Smailyte et al.** [33]: cohort of woodworkers exposed to softwood dust (1080 men and 438 women) Oral cavity SIR in males: 2.83 (1.29–5.37)	Group 1. No evidence for this site. (Vol. 100 C, 2012)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Cancer of the lip	Outdoor work	**Kenborg et al.** [35]: Nationwide, population-based case control study. 3187 male cases of lip cancer ascertained from the Danish Cancer Registry. 9361 controls Outdoor work >10 years, OR = 1.67 (1.38–2.03) adjusted for social class and place of birth	Solar radiations are Group 1. Limited evidence for this site. (Vol. 100 D, 2012)
Gastrointestinal cancers			
Squamous cell carcinoma of the esophagus	Carbon black	**Parent et al.** [36]: population based, case-control study (99 esophageal cancers/63 squamous cell esophageal carcinomas/1066 controls) Any exposure OR = 3.4 (1.5–7.7) adjusted for age, respondent status, birthplace, educational level, alcohol consumption, carotene index, smoking Substantial exposure OR = 8.9 (1.2–64.3) adjusted for age, respondent status, birthplace, educational level, alcohol consumption, carotene index, smoking	Group 2B. (Vol. 93, 2010)
	Sulfuric acid	**Parent et al.** [36]: Any exposure OR = 2.8 (1.2–6.1) adjusted for age, respondent status, birthplace, educational level, alcohol consumption, carotene index, smoking	Group 1. No evidence for this site. (Vol. 100 F, 2012)
	Chrysotile asbestos	**Parent et al.** [36]: Any exposure OR = 2.0 (1.1–3.8) adjusted for age, respondent status, birthplace, educational level, alcohol consumption, carotene index, smoking	Group 1. No evidence for this site. (Vol. 100 C, 2012)
	Polycyclic aromatic hydrocarbons	**Gustavson et al.** [32]: Community based case referent study (545 cases and 641 referents) Low level OR = 2.01 (1.16–3.48) adjusted for region, age, alcohol consumption and smoking habits High level OR = 1.87 (1.11–3.16) adjusted for region, age, alcohol consumption and smoking habits	Some compounds are Group 1. No evidence for this site. (Vol. 100 F, 2012)
Adeno-carcinoma of the esophagus	Sulfur compounds	**Santibañez et al.** [37]: Hospital-based case control study, 147 squamous cell carcinoma and 38 adenocarcinoma of esophagus and 285 frequency matched controls High level of exposure (0.025 ppm) OR for adenocarcinoma = 3.12 (1.00–9.77) adjusted for age, province, educational level, alcohol drinking and tobacco smoking	Group 1. No evidence for this site. (Vol. 100 F, 2012)
	Lead	**Santibañez et al.** [37]: High level of exposure High (.0.237 mmol/l) OR for adenocarcinoma = 5.30 (1.39–20.22) adjusted for age, province, educational level, alcohol drinking and tobacco smoking	Group 2 B. (Vol. 23, Sup 7, 1987)
Cancer of the small intestine	Asbestos	**Clin et al.** [38]: Cohort of 2024 subjects occupationally exposed to asbestos (3 cases of small intestine cancer) SIR = 6.93 (1.39–20.25) among men with an exposure exceeding 80 fibers/mL × years (no adjustment)	Group 1. Limited evidence for colorectum. (Vol. 100 C, 2012)
	Semiautomatic arc welding (MIG/MAG)	**Kaerlev et al.** [39]: European case control study (79 cases, 579 colon cancer controls, and 2070 population controls) Small bowel adenocarcinoma OR = 5.0 (1.3–19.6) adjusted for country, year of birth, and sex	Welding fumes classified group 2B. No classification for this site. (Vol. 49, 1990)
	Organic solvents	**Kaerlev et al.** [40]: European case control study (84 cases and 2070 population controls) Small bowel carcinoid tumor OR = 2.0 (CI 1.0–4.2) adjusted for country, year of birth, and sex	No classification for this site.

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Benzidine	**Brown et al.** [41]: Cohort of 997 individuals employed at a chemical production facility (4 cases of small intestine cancer) SIR = 18.4 (2.2–66.4) among workers with highest level of exposure (no adjustment)	Group 1. No classification for this site. (Vol. 100 F, 2012)
Hepatocellular carcinoma of the liver and intrahepatic bile tract (IBT)	Polychlorinated biphenyls (PCBs)	**Ahrens et al.** [45]: Case control study among men in six European countries (183 case, 1938 controls) Exposure to Oils with polychlorinated biphenyls OR = 2.8 (1.3–5.9) for carcinoma of the extrahepatic biliary tract (adjusted for age, country and gallstones) **Prince et al.** [46]: Cohort mortality study among 2572 workers highly exposed to polychlorinated biphenyls (PCBs) during the manufacture of electrical capacitors (11 deaths from biliary passage, liver and gall bladder cancer) SMR = 2.11 (1.05–3.77) (no adjustment)	Group 1. Limited evidence for this specific site. (Vol. 107, in prep, meeting in 2013)
	Vinyl chloride monomer	**Mastrangelo et al.** [47]: Case-referent study nested in a cohort of 1658 VCM workers Hepatocellular carcinoma OR = 1.71 (1.28–2.44) for each extra increase of 1000 ppm × years of VCM cumulative exposure (adjusted for alcohol and viral hepatitis infection) **Boffetta et al.** [48]: Meta-analysis (8 cohort studies). Two large studies considered liver cancers other than ASL separately ($n = 68$): Meta-SMR = 1.35 (1.04–1.77) (no adjustment)	Group 1. Sufficient evidence for this site of cancer. (Vol. 100 F, 2012)
	Toluene and xylene	**Porru et al.** [49]: Case control study (144 males with a liver cancer, 283 male controls) OR = 2.8 (1.0–7.6) for 20 or more years of exposure adjusted for age, residence, education, HBsAg and HCVAb positivity and heavy alcohol consumption	Toluene Group 3. Xylene Group 3. (Vol. 71, 1999)
	Trichloro-ethylene	**Wartenberg et al.** [50]: Review on trichloroethylene and cancer Liver cancer average risk: 1.9 (1.0–3.4) based on SIRs from 3 cohorts	Group 1. Limited evidence for this cancer site. (Vol. 106, in prep, meeting in 2012)
	Tetrachloro-ethylene	**Lynge et al.** [51]: Cohort of 10,600 laundry and dry-cleaning workers exposed to tetrachloroethylene and other solvents Liver cancer among women SIR = 3.4 (1.4–7.0)	Group 2A. No evidence for this site. (Vol. 106, in prep, meeting in 2012)
	Organic solvents	**Chen et al.** [52]: Meta-analysis of mortality among workers exposed to organic solvents from 55 studies Liver and biliary passages SMR = 119.7 (104.4–137.2)	
	Radon in underground miners	**Darby et al.** [53]: Collaborative analysis of 11 cohort studies Liver cancer SMR = 1.73 (1.29–2.28), no clear dose-exposure relation **Tomasek et al.** [54]: Cancer mortality in 4320 uranium miners in West Bohemia. 22 liver cancer cases. Liver O/E = 1.67 (1.04–2.52)	Group 1. No mention to this specific site. (Vol. 100 D, 2012)
	Plutonium	**Sokolnikov et al.** [55]: Cohort of 17,740 nuclear facility workers ERRs for liver cancer were 2.6 for males and 29 and for females	Group 1. Sufficient evidence for liver. (Vol. 100 D, 2012)
	Magnetic field exposure (ELF)	**Floderus et al.** [30]: Swedish cohort study including 1,596,959 men from national census and job exposure matrix based on measurements. Biliary passage and liver Medium exposure (0.084–0.115 μT) RR = 1.2 (1.1–1.4) age adjusted High exposure (≥116 μT) RR = 1.3 (1.2–1.5) age adjusted	Group 2B. (Vol. 80, 2002)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Hepatic angiosarcoma	Vinyl chloride monomer	**Kielhorn et al.** [5]: Review and Meta-Analysis (4 main cohort studies) The global SMR from European and north American studies for all liver cancer including angiosarcoma was 5.33 (4.23–6.82) reaching 280.0 among highest exposure group (no adjustment)	Group 1. Sufficient evidence for this site of cancer. (Vol. 100 F, 2012)
Epithelial tumors of gallbladder and extrahepatic biliary tract (EBT)	Radon in underground miners	**Tomasek et al.** [54]: Cancer mortality in 4320 uranium miners in West Bohemia. 12 EBT cases. Gallbladder and extrahepatic bile ducts O/E = 2.26 (1.16–3.94)	Group 1. No mention to this specific site. (Vol. 100 D, 2012)
Thoracic cancers			
Large cell lung carcinoma	Asbestos	**de Klerk et al.** [57]: Cohort study among workers from Wittenoom asbestos industry (2400 men and 149 women, 71 lung cancer cases, 8 LCell) OR = 2.1 (1.0–4.3) (Ioge) cumulative exposure (fibers.ml.years) adjusted for tobacco smoking	Group 1. Sufficient evidence for lung cancer. (Vol. 100 C, 2012)
	Diesel exhaust	**Villeneuve et al.** [58]: Case control study among men ≥40 years (1681 incident cases of lung cancer and 2053 population controls) OR across the three increasing tertiles of cumulative lifetime exposure: 1.06, 1.19, 1.68 ($p = 0.02$), adjusted for age, province, tobacco smoking, occupational exposure to silica and asbestos	Group 1. Sufficient evidence for lung. (Vol. 105, in prep, meeting in 2012)
Mesothelioma	Asbestos	**Harding et al.** [59]: Cohort of 98,912 asbestos workers (649 mesothelioma cases) SMR men: 13.3 (12.3–14.4) SMR women: 30.9 (18.3–48.8) **Loomis et al.** [60]: Cohort of 5770 asbestos textile workers (4 mesothelioma cases (not separately coded before 1999)) SMR = 10.92 (2.98–27.96) **Berman et al.** [2]: Meta-analysis from 11 cohort studies providing type of fibers SMR amphibole = 13.8 (3.5–26.3)	Group 1. Sufficient evidence for this site. (Vol. 100 C, 2012)
Reproductive cancers			
Epithelial tumors of the male breast	Alkylphenolic compounds	**Villeneuve et al.** [64]: European case control study (104 cases and 1901 controls) OR = 3.8 (1.5–9.5) among subjects exposed above the median, adjusted for age, country, alcohol consumption, body mass index and education	Not classified.
	Gasoline and combustion products	**Hansen et al.** [63]: Nationwide register based case control study on male breast cancer morbidity (230 cases and 12,880 control) OR = 2.5 (1.3–4.5) adjusted for birth year and socioeconomic status	Gasoline engine exhaust classified group 2B. (Vol. 105, in prep, meeting in 2012)
Epithelial tumors of the cervix uteri	Organic solvents	**Weiderpass et al.** [65]: Register linkage study in Finland among 413,877 female workers born (1906–1945), 1101 cervical cancer cases Aliphatic and alicyclic solvents RR = 1.3 (1.1–1.6) Aromatic-hydrocarbon solvents RR = 1.2 (1.1–1.4) Chlorinated-hydrocarbon solvents RR = 1.3 (1.0–1.7)	No classification for this site.
	Trichloroethylene	**Wartenberg et al.** [50]: Review on trichloroethylene and cancer (over 80 papers) SMR (from 4 mortality studies) average risk 1.7 (1.5–2.0) SIR (one cohort study) 2.4 (1.1–4.8)	Group 1. No evidence for this site. (Vol. 106, 2012)
	Metalworking fluids	**Betenia et al.** [66]: Cohort of 4374 female autoworkers followed from 1985–2004 SIR = 2.96 (2.11–4.02) based on 40 cases	No classification for this site.

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Ovarian cancer	Asbestos	**Camargo et al.** [67]: Meta-Analysis including 18 studies SMR 1.77 (1.37–2.28)	Group 1. Sufficient evidence for this site. (Vol. 100 C, 2012)
	Silica dust	**Wernli et al.** [68]: Nested case control study 261 incident ovarian cancer cases and 3121 control women RR = 5.6 (1.4–23.6) among women exposed 10 years or more, adjusted for age and reproductive category	Group 1. No mention to this specific site.
	Diesel exhaust	**Guo et al.** [69]: Register-linkage study in Finland among female workers born 1906–1945 RR = 3.7 (1.4–9.9) among highest exposure group ($\geq$10.0 mg/m^3-years) adjusted for mean number of children, body mass index, socioeconomic status, age and calendar period; *p* for trend 0.006	Group 1. No evidence for this specific site. (Vol. 105, in prep, meeting in 2012)
	Trichloroethylene	**Morgan et al.** [70]: Cohort of 20,508 aerospace workers (13 cases), 4733 with occupational exposure to trichloroethylene (8 cases) High cumulative exposure RR = 7.1 (2.1–23.5), no adjustment	Group 1. No evidence for this site. (Vol. 106, in prep, meeting in 2012)
Urogenital cancers			
Non-bladder urinary organs (renal pelvis, ureter, urethra)	Polycyclic aromatic hydrocarbons (PAH)	**Jensen et al.** [75]: Hospital-based case control study (96 cases (renal pelvis, ureter) 294 controls) Coke, coal OR = 4.0 (1.2–13.6); Asphalt, tar OR = 5.5 (1.6–19.6), adjusted for sex and lifetime tobacco consumption	Group 1. No classification for this site. Coal-tar pitch: limited evidence for bladder cancer, no classification for this site. (Vol. 100 F, 2012)
Neuroectodermic and mesodermic tumors			
Epithelial tumors of the eye and adnexa	Ultraviolet radiation	**Vajdic et al.** [76]: Population-based case control study in Australia (290 cases, 893 controls) Highest category of GSR-weighted (MJ/m2) occupational outdoor hours during decade years OR = 2.1 (1.2–3.7) adjusted for age, region of birth, eye color, ability to tan and squinting as a child **Håkansson et al.** [77]: Cohort study on sunlight exposure and cancer incidence in the Swedish construction industry including 323,860 men (35 eye melanoma cases) Ocular melanoma in the highest exposed group RR = 3.4 (1.1–10.5) adjusted for age, smoking, and magnetic field exposure	UV-emitting tanning devices classified group 1 with sufficient evidence for this site. (Vol. 100 D, 2012) Welding classified Group 1 with sufficient evidence for eye. (Vol. 100 D, 2012)
Malignant melanoma of the uvea		**Guénel et al.** [78]: French part of a European Case control study (50 cases, 479 controls) Ocular melanoma among men welders age adjusted OR = 7.3 (2.6–20.1) and dose-response relationship with job duration	
Soft-tissue sarcoma	PolyChloroPhenol	**Hoppin et al.** [79]: Population-based case control study among men (295 cases, 1908 controls) Odds ratios were adjusted for age, registry, race, medical radiations and exposure to herbicides High-intensity exposure OR = 1.79 (1.10–2.88). Duration-response trend (*p* for trend < 0.0001) 10 years of substantial exposure or more, OR = 7.78 (2.46–24.65)	Group 2B. Limited evidence for this site. (Vol. 71 and 100 F, 2012)
	2,3,7,8-Tetrachloro-dibenzo-p-dioxin	**Fingerhut et al.** [80]: Cohort mortality study among 5172 workers at 12 plants in the United States that produced chemicals contaminated with TCDD SMR = 922 (190–2695) **Collins et al.** [81]: Cohort study of 1615 workers exposed to dioxins in trichlorophenol production SMR = 4.1 (1.1–10.5)	Group 1. Limited evidence for STS. (Vol.100 F, 2012)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Bone sarcoma	Ionizing radiation	**Carnes et al.** [9]: cohort studies of 820 women working as radium watch-dial painters (46 bone sarcomas) Dose response relationships for bone sarcoma mortality: $p < 0.001$	Group 1. Sufficient evidence for this site. (Vol. 100 D, 2012)
	Pesticides	**Merletti et al.** [83]: Multicenter case control study in 7 European countries. 96 bone sarcoma cases compared to 2632 controls OR adjusted for age, country, gender and number of job periods: 2.61 (1.49–4.57)	
Malignant meningioma	Lead (inorganic)	**Rajaraman et al.** [95]: Case control study of 197 meningioma cancers and 799 non-cancer controls In subject with ALAD2 variant allele and exposed to 100 μg/m^3-year, OR = 12.8 (1.4–120.8) **Navas-Acien et al.** [96]: Historical cohort of all Swedish men and women RR = 2.36 (1.12–4.96) in male workers with possible exposure to lead (adjusted for age, Period, Geographical Category and Town Size and exposure to other chemicals)	Group 2 A. (Vol. 87, 2006)
Endocrine tumors			
Carcinoma of the thyroid gland	Ionizing radiations	**Zielinski et al.** [98]: Cohort study of the Canadian national dose registry of radiation workers SIR = 1.74 (1.40–2.10) **Zabel et al.** [100]: Among workers exposed more than 5 years prior to 1950: RR = 3.04 (1.01–10.78)	Group 1. Sufficient evidence for thyroid cancer in Atomic-bomb survivors, medical patients. (vol. 100 D, 2012)
	Organic solvents	**Lope et al.** [101]: Cohort of 2,992,166 Swedish workers employed in the 1970 census (2599 TC) Probable exposure to organic solvents among women, RR = 1.91 (1.05–3.45) after adjustment for exposure to ionizing radiations	
Lymphoid diseases			
Hodgkin disease	Pesticides	**Karunanayake et al.** [104]: Population-based, case control study (316 Hodgkin Lymphoma and 1506 controls). Adjustment for age, province of residence, medical and family history, use of other pesticides, correlated with chlorpyrifos use. Exposure to insecticide chlorpyrifos OR = 1.19 (1.03–1.37) All other pesticides showed not significant association or no association **Orsi et al.** [102]: Hospital based case control study (491 cases (87 HD); 456 controls); Analysis adjusted for age, socioeconomic category Fungicides OR 4.5 = (1.6–12.2); organochlorine OR = 4.7 (1.1–20.8) Organophosphate OR = 3.0 (1.0–9.4); Pyrethrin OR = 3.6 (1.2–11.2) Phenoline herbicide OR = 4.3 (1.1–17.2); Picoline OR = 9.4 (2.0–43.1) Amide OR 3.8 = (1.1–12.7); Urea herbicide OR = 10.8 (2.4–48.1) **Khuder et al.** [103]: Meta-analysis of 30 peer-reviewed studies. Occupational exposure as farmer: combined RR = 1.25 (1.11–1.42); 13 case–control studies: combined RR = 1.53 (1.18–1.98); 7 cohort studies combined RR = 1.08 (0.97–1.20); 10 mortality and morbidity studies: combined RR = 1.18 (1.02–1.36)	No classification for this site.

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Wood dust	**Briggs et al.** [105]: Population-based case control study (343 Hodgkin disease and 1910 controls) OR = 4.6 (1.6–13.3), adjusted for age and race	Group 1. No classification for this site. (Vol. 100 C, 2012)
Acute lymphoblastic leukemia	Benzene	**Vlaanderen et al.** [107]: MA of 44 studies (17 studies for ALL with 47 ALL cases). Meta-RR = 1.44 (95%CI 1.03–2.02)	Group 1. Limited evidence for ALL. (Vol. 100 F, 2012)
Diffuse large B-cell lymphoma (DLBCL)	Solvents	**Cocco et al.** [113]: Multicenter case-control study (2348 lymphoma cases (251 FL)/2462 controls). Adjustment for age, gender, education and center. All solvents: OR = 1.0 (0.8–1.2), *p* trend NS **Wang et al.** [117]: population-based case–control study in women (601 NHL cases (189 DLBCL)/717 controls). Adjustment for age, gender, education and center. Organic solvents, medium-high intensity OR = 2.1 (1.4– 3.3), *p* trend < 0.01 Chlorinated solvents, medium-high intensity OR = 2.2 (1.4, 3.4), *p* trend < 0.01 **Miligli et al.** [120]: Population-based multicenter case-control study (1428 NHL (308 DLBCL)/1530 controls); adjustment for sex, age, education, and area Any solvent OR = 1.5 (1.0–2.1); Xylene OR = 2.3(1.2–4.4); Toluene OR = 2.4 (1.3–4.6)	Limited evidence for Benzene Group 1. (Vol. 100 F, 2012)
	Trichloroethylene (TCE)	**Purdue et al.** [119]: population-based case–control study (1189 cases (293 follicular lymphoma, 366 DLBCL), 982 controls). Adjustment for age, sex, race, education level and area TCE > 150 ppm–hr/week OR = 1.11 (1.01–1.23), p trend = 0.03 TCE Cumulative exposure (estimated ppm–hr) > 112,320 OR = 1.07 (0.94–1.22), *p* trend NS **Seidler et al.** [115]: population-based case–control study (710 malignant lymphoma (158 DLCBL)/710 controls) adjustment for smoking (in pack years) and alcohol consumption TCE >35 ppm*yrs.: OR = 2.6 (0.7–3.0), *p* trend 0.03	Group 1. Limited evidence for lymphoma. (Vol. 106, in prep, meeting in 2012)
	Ethylene oxide	**Kiran et al.** [136]: case–control study (2347 lymphoma cases (530 DLCBL) and 2463 controls), adjustment for age, sex, and participating center. Ever exposure to ethylene oxide OR = 1.3 (0.6–2.9) Exposed > 5% of working hours OR = 6.4 (1.8–23.0); *p* trend 0.063	Group 1. Limited evidence for NHL. (Vol. 100 F, 2012)
Follicular B lymphoma (FL)	Solvents	**Cocco et al.** [113]: Multicenter case–control study (2348 lymphoma cases (251 FL)/2462 controls). Adjustment for age, gender, education and center. All solvents: OR = 1.3 (1.0–1.7), *p* trend < 0.001 Benzene, toluene and xylene combined: OR = 1.7 (1.2–2.5), *p* trend < 0.0001 **Wang et al.** [117]: population-based case-control study in women (601 NHL cases (136 FL)/717 controls) adjustment for age, family history of hematopoietic cancers, alcohol, and race Organic solvents, medium-high intensity OR = 1.3 (0.7–2.1), *p* trend NS **Seidler et al.** [115]: population-based case–control study (710 malignant lymphoma (92 FL)/710 controls) adjustment for smoking (in pack years) and alcohol consumption Chlorinated hydrocarbons >47.3 ppm*yrs.: OR = 3.9 (1.3–12.1), *p* trend = 0.04	Limited evidence for Benzene Group 1. (Vol. 100 F, 2012) Xylenes: Group 3. Toluene: Group 3. Dichloromethane: Group 2B. 1,2-Dicloroéthane Group 2B. (Vol. 71, 1999)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Trichloroethylene (TCE)	**Purdue et al.** [119]: population-based case–control study (1189 cases (293 follicular lymphoma, 293 FL), 982 controls). Adjustment for age, sex, race, education level and area TCE > 150 ppm–hr/week OR = 3.7 (1.2–11.7), *p* trend = 0.005 TCE Cumulative exposure (estimated ppm–hr) > 112,320 OR = 1.17 (1.04–1.32), *p* trend = 0.01	Group 1. Limited evidence for lymphoma. (Vol. 106, in prep, meeting in 2012)
Hair cell leukemia	Pesticides	**Orsi et al.** [102]: Hospital based case control study (491 cases (27 HCL); 456 controls); Analysis adjusted for age, socioeconomic category. Occupational exposure to organochlorine insecticides OR = 4.9 (1.1–21.2); Herbicides (Picoline OR = 4.1 (1.1–15.5), Triazine OR = 5.1 (1.4–19.3). **Clavel et al.** [122]: Hospital based case control study (226 HCL, 425 controls). Possible or definite occupational pesticides exposure OR = 1.5 (1.0–2.3). Definite organochlorines exposure OR = 2.1 (1.2–3.7); Definite organophosphorus exposure OR = 2.6 (1.1–57) Definite herbicide exposure OR = 2.0 (1.1–3.5); definite exposure to Triazine OR = 2.4 (1.2–4.8) Definite organic fungicides exposure OR = 2.9 (1.5–5.3); Duration of definite exposure to organic fungicides OR = 2.6 (0.9–7.5) and OR = 3.5 (1.4–8.3), for <10 years and >10 years of exposure respectively. After adjustment for smoking: OR = 7.5 (0.9–61.5) for exposure to organophosphorus insecticides, OR = 2.8 (1.4–5.6) for forage growing in non-smokers. **Nordström et al.** [123]: Population-based case-control study (121 male HCL; 484 controls) Exposure to herbicides OR = 2.9 (1.4–5.9), insecticides OR = 2.0 (1.1–3.5), fungicides OR = 3.8 (1.4–9.9), impregnating agents OR = 2.4 (1.3–4.6). After adjustment for age: herbicides OR = 1.8 (0.7–4.6), insecticides OR = 0.7 (0.3–3.7), fungicides OR = 2.1 (0.6–6.5), impregnating agents OR = 2.0 (1.0–3.9).	Group 2A: occupational exposures to non-arsenical insecticides (spraying and application) (Vol. 53, 1991) Several compounds classified 2B or 3 (Vol. 5 sup 7, 1987; Vol. 30, sup 7, 1987; Vol. 41, sup 7, 1987; Vol. 53, 1991) No classification for this site.
Multiple myeloma (MM)	Pesticides	**Perrotta et al.** [129]: Systematic review of 55 case control studies (1970–2007) Working as a farmer pooled OR = 1.39 (1.18–1.65) Working on a farm >10 years pooled OR = 1.87 (1.15–3.16) Ever occupational pesticides exposure OR = 1.47 (1.11–1.94) Herbicides exposure pooled OR = 2.19 (1.30–2.95) **Pahwa et al.** [130]: Population-based case control study (342 Multiple Myelomas and 1506 controls). Analysis adjusted for age, province of residence, medical and family history of MM, exposure to chemicals other than pesticides Carbamate insecticides OR = 1.81 (1.05–5.35) Fungicide captan OR = 2.35 (1.03–5.35) **Orsi et al.** [102]: Hospital based case control study (244 NHL (56 MM) and 456 controls); Analysis adjusted for age, socioeconomic category Occupational pesticides use OR = 3.5 (1.6–7.7); Insecticides OR = 2.8 (1.2–6.5); Fungicides OR = 3.2 (1.4–7.2); Herbicides OR = 2.9 (1.3–6.5)	

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
		Lope et al. [128]: Historical cohort (1971–1989; 2992,166 workers; linkage with National Cancer and Death Registries). Multiple Myeloma 3127 men and 1282 women. Analysis adjusted for age, period and geographical area Male occupation in agriculture, forestry, and fishing RR = 1.18 (1.07–1.30) Male exposure to peaks of pesticides RR = 1.2 (1.07–1.34)	Group 2A: (occupational exposures to non-arsenical insecticides (spraying and application) (Vol. 53, 1991) Several compounds classified B or 3 (Vol. 5, sup 7, 1987; Vol. 30, sup 7, 1987; Vol. 41, sup 7, 1987; Vol. 53, 1991)
		Pukkala et al. [73]: Historical cohort with record linkage in 5 Nordic countries (15 Million people; MM 22,106 men, 19,508 females) Male farmers SIR = 1.07 (1.03–1.11); female farmers SIR = 1.14 (1.05–1.24)	No classification for this site.
	Benzene	**Vlaanderen et al.** [107]: Meta-analysis of 44 studies (26 studies corresponding to 284 MM) Meta-RR = 1.12 (0.98–1.27) **Infante et al.** [125]: MA of 8 benzene cohorts (22 observed MM deaths) SMR = 1.61 (1.01–2.44); pooled weighted RR = 2.13 (1.31–3.46) **Sonoda et al.** [127]: Meta-analysis (8 case-control studies) Meta OR = 0.74 (0.60–0.90)	Group 1.Limited evidence for MM. (Vol. 100 F, 2012)
	Ethylene oxide		Group 1. Limited evidence for MM. (Vol. 100 F, 2012)
	Engine exhaust	**Sonoda et al.** [127]: Meta-analysis (7 case-control studies) OR = 1.34 (1.14–1.57)	Diesel engine exhaust Group 1. Gasoline engine exhaust Group 2B. (Vol. 105, 2012) No classification for this site.
Chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL)	Benzene	**Schnatter et al.** [135]: pooled analysis of 3 cohort studies (370 cases (80 CLL); 1587 controls) OR = 1.49(0.81–2.75) **Vlaanderen et al.** [107]: Meta-analysis of 44 studies (18 studies for CLL with 111 CLL cases) meta-RR 1.14 (0.78–1.67) **Khalade et al.** [134]: Meta-analysis of 15 (10 studies for CLL) Summary effect-size OR 1.31 (1.09–1.57)	Group 1. Limited evidence for this site. (Vol. 100 F, 2012)
	Solvents	**Cocco et al.** [113]: Multicenter case-control study (2348 lymphoma cases (323 CLL)/2462 controls). Adjustment for age, gender, education and center; Bonferroni correction for multiple testing All solvents OR = 1.5 (1.1–1.9) p trend 0.000004; Benzene, Toluene and Xylene combined OR = 1.4 (1.0–1.9); p trend 0.000048 **Purdue et al.** [119]: population-based case–control study (1189 cases (141 CLL/SLL), 982 controls). Adjustment for age, sex, race, education level and area OR = 2.7 (1.2–5.8) p trend not significant. **Seidler et al.** [115]: population-based case-control study (710 malignant lymphoma 104 CLL/710 controls) adjustment for smoking (in pack years) and alcohol consumption, no positive association with solvents found **Miligli et al.** [120]: Population-based multicenter case-control study (1428 NHL 285 CLL and small lymphocytic lymphoma)/1530 controls); adjustment for sex, age, education, and area Dichloromethane OR = 3.2 (1.0–10.1); Toluene OR = 2.0 (1.0–4.0)	No IARC classification for this site. Xylenes: Group 3. Toluene: Group 3. Dichloromethane: Group 2B. (Vol. 71, 1999)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
	Ethylene oxide	**Kiran et al.** [136]: case–control study (2347 lymphoma cases (406 CLL) and 2463 controls), adjustment for age, sex, and participating center. Ever exposure to ethylene oxide OR = 2.0 (0.8–4.7) Exposed >5% of working hours OR = 6.2 (1.3–29.3), p trend 0.028	Group 1. Limited evidence for CLL. (Vol. 100 F, 2012)
	1,3-Butadiene	**Sielken et al.** [138]: cohort study 16,585 workers (12,819 workers exposed). 81 leukemia mortalities, 71 among exposed workers. Slope per cumulative ppm-years, $\beta = 0.000417$, $p = 0.000027$	Group 1. Sufficient evidence for haematolymphatic organs. No specific classification for this subtype. (Vol. 100 F, 2012)
Marginal zone lymphoma/MALT lymphoma	Solvents	**Seidler et al.** [115]: population-based case–control study (710 malignant lymphoma (38 marginal zone lymphoma)/710 controls) adjustment for smoking (in pack years) and alcohol consumption High occupational exposure to chlorinated hydrocarbons OR = 7.0 (1.8–26.3)	No IARC classification.
Acute myeloid leukemia	Benzene	**Vlaanderen et al.** [124]: Meta-analysis of (21 studies with 217 AML cases) Meta RR 1.68(1.35–2.10) **Khalade et al.** [134]: Meta-analysis of 15 studies (9 studies for AML) Summary effect-size 1.38 (1.15–1.64)	Group 1. Sufficient evidence for this site. (Vol 100F, 2012)
	Pesticides	**Van Maele Fabry et al.** [146]: Meta-analysis of 17 cohort and 16 case-control studies Meta RR = 1.55 (1.02–2.34)	Group 1: Arsenic (Vol. 100 C, 2012) Group 2A: Occupational exposures to non-arsenical insecticides (spraying and application) (Vol. 53, 1991) Captafol (Vol. 53, 1991) Several compounds classified 2B or 3 (Vol. 5, sup 7, 1987; Vol. 30, sup 7, 1987; Vol. 41, sup 7, 1987; Vol. 53, 1991) No classification for this site.
	Formaldehyde	**Zhang et al.** [132]: Meta-analyses of 21 studies (4 studies with myeloid leukemia subtype specific data) Meta RR for myeloid leukemia = 2.47 (1.42–4.27)	Group 1. Sufficient evidence for leukemia. No specific classification for this subtype. (Vol. 100 F, 2012)
	Radiation	**Daniels et al.** [145]: nested case-control study in nuclear worker cohort (369 leukemia deaths (150 AML) in 105,245 US nuclear workers) AML in the 6–14-years window (ERR per 100 mGy = 7.0 (0.079–32.0); adjusted for sex, race and hire year	Group 1. Sufficient evidence for leukemia. No specific classification for this subtype. (Vol. 100 D, 2012)
Chronic myeloid leukemia	Formaldehyde	**Zhang et al.** [132]: Meta-analyses of 21 studies (4 studies with myeloid leukemia subtype specific data) Meta RR for myeloid leukemia = 2.47 (1.42–4.27)	Group 1. Sufficient evidence for myeloid leukemia. (Vol. 100 F, 2012)
	Radiation	**Daniels et al.** [145]: nested case–control study in nuclear worker cohort (369 leukemia deaths (52 CML) in 105,245 US nuclear workers) CML (ERR per 100 mGy = 0.29 (<0 to 1.8); adjusted for sex, race and hire year	Group 1. Sufficient evidence for leukemia. (Vol. 100 D, 2012)
Other myelo-dysplastic and myelo-proliferative neoplasms	Benzene	**Schnatter et al.** [135]: pooled analysis of 3 cohort studies (370 cases (29 myelodysplastic syndromes (MDS) and 30 myeloproliferative disorders (MPD), 1587 controls) Cumulative exposure >2.93 ppm–years high and medium certainty diagnoses MDS OR = 4.33 (1.31–14.3); MPD OR = 1.79(0.68–4.74)	Group 1. No classification for this site. (Vol. 100F, 2012)

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Childhood leukemia	Parental occupational pesticides exposure	**Vinson et al.** [164]: Meta-analysis of 40 studies: 3 cohort studies: OR = 0.95 (0.81–1.11); 37 case control studies: Paternal occupational exposure OR = 1.37 (1.23–1.52); Paternal exposure during prenatal period (occupation and environmental) OR = 1.32 (1.20–1.46); Maternal exposure during prenatal period (occupation and environmental) OR = 1.48 (1.26–1.75); Parental exposure to insecticides OR = 1.17 (1.03–1.33); Parental exposure to herbicides OR = 1.26 (1.14–1.39) **Van Maele-Fabry et al.** [162]: Meta-analysis of 25 studies: Maternal occupational exposure meta RR = 1.62 (1.22–2.16); Paternal occupational exposure meta RR = 1.14 (0.76–1.69) **Wigle et al.** [163]: Meta-analysis of 30 studies: Maternal occupational exposure (16 studies) OR = 2.09 (1.51–2.88) Paternal occupational exposure (30 studies) OR = 1.09 (0.88–1.34)	Group 1: Arsenic (Vol. 100 C, 2012) Group 2A: application of non-arsenical pesticides. Captafol (Vol. 53, 1991), Several compounds classified 2B or 3 (Vol. 5, sup 7, 1987; Vol. 30, sup 7, 1987; Vol. 41, sup 7, 1987; Vol. 53, 1991) No classification for this site.
	Painting	**Schuz et al.** [157]: Case control study (1138 ALL/2962 controls): Maternal pre-conceptional occupational exposure: OR = 1.6 (1.1–2.4) and during pregnancy: OR = 2.0 (1.2–3.3). Adjusted for socioeconomic status and degree of urbanization **Shu et al.** [155]: Case control study including 1842 ALL and 1986 controls. Maternal occupational exposure pre-conceptional OR = 1.6 (1.1–2.3) and during pregnancy OR = 1.7 (1.2–2.3). Adjusted for maternal education, race, and family income **Buckley et al.** [154]: Case control study with 204 AML/ANLL) Maternal exposure OR = 2.2 (0.9–5.4), prolonged spray paints exposure OR = 3.0 (trend, $p = 0.03$). Adjusted for other professional exposure **Lowengart et al.** [158]: registry based case control study (123 acute leukemia cases, 123 controls) paternal exposure after delivery to spray paint OR = 2.2 (0.96–4.39). No information on adjustment variables **Van Steensel-Moll et al.** [153]: Registry based case control study (519 ALL, 507 controls) Maternal occupational exposure during pregnancy RR = 2.4 (1.2–4.6)	Group 1. Limited evidence for this site. (Vol. 100 F, 2012)
	Solvents	**Castro-Jimmenez et al.** [165]: Case control study (85 ALL and 85 individually matched neighborhood controls) Parental occupational exposure to hydrocarbons during 24 months before conception: mother only OR = 6.33 (1.41–28.31), both parents OR = 13.47 (3.31–54.71); adjustment for maternal age at child's birth, parental pre-conception smoking status, and maternal socioeconomic status during index pregnancy **Infante-Rivard et al.** [167]: Population-based case–control study (790 childhood ALL, 790 healthy controls) Maternal occupational exposure to solvents before and during pregnancy, adjusted for maternal age and sex. OR = 1.11 (0.88–1.40); mononuclear aromatic hydrocarbons OR = 1.64 (1.12–2.41)	

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
		Mc Kinney et al. [159]: Registry-based case control study (40,647 female workers, 47 childhood cancers); Childhood leukemia RR = 3.83 (1.17–12.55); all childhood malignancies RR = 2.26 (1.12–4.54) peri-conceptional maternal occupational exposure **Schuz et al.** [157]: Case control study (1138 ALL/2962 controls) Maternal occupational exposure pre-conceptional: OR = 1.2 (0.9–1.7) and during pregnancy: OR = 1.3 (0.8–1.9). Adjusted for socioeconomic status and degree of urbanization **Smulevitch et al.** [168]: Population-based case control study including 593 childhood cancers and 1181 controls Maternal occupational exposure OR = 3.1 (1.5–6.3) adjusted for parental alcohol consumption **Shu et al.** [155]: Case control study 1842 ALL and 1986 controls) Maternal occupational exposure pre-conception OR = 1.8 (1.3–2.5); during pregnancy OR = 1.6 (1.1–2.3) adjusted for maternal education, race, and family income **Buckley et al.** [154]: Case control study including 204 AML/ANLL Paternal exposure OR = 2.0 (1.2–3.8), adjusted for other professional exposures **Lowengart et al.** [158]: Registry based case control study (123 acute leukemia cases and 123 controls) Paternal exposure after delivery to chlorinated solvents OR = 3.5 (1.1–14.6). No information on adjustment variables	No IARC classification for this site. Xylenes: Group 3. Toluene: Group 3. Dichloromethane: Group 2B. 1,2-Dicloroéthane Group 2B. (Vol. 71, 1999)
	Wood work	**Feychting et al.** [160]: Birth cohort (235,635 children, 161 leukemias), registry linkage study Paternal pre-conceptional occupation in wood work RR = 2.18 (1.26–3.78). Adjusted for maternal age, socio-economic status, census year and gender	Group 1. No IARC classification for this site. (Vol. 100 C, 2012)
Childhood lymphoma	Pesticides	**Vinson et al.** [164]: Meta-analysis of 40 studies Paternal prenatal exposure (9 studies) OR = 1.37 (1.16–1.61) Maternal prenatal pesticides exposure (5 studies) OR = 1.53 (1.22–1.91) Ever exposure mother (6 studies) OR = 1.90 (1.14–3.17) Parental exposure to insecticides (11 studies) OR = 1.46 (1.20–1.78) Parental exposure to herbicides (4 studies) OR = 1.31 (1.02–1.67) Parental exposure to fungicides (3 studies) OR = 1.45 (1.06–1.99)	No IARC classification for this site.
Ewing sarcoma	Pesticides	**Vinson et al.** [164]: Meta-analysis Paternal occupational exposure (3 case control studies) OR = 2.34 (1.33–4.12); Maternal occupational exposure non-significant increased risk **Valery et al.** [186]: Meta-analysis of 5 case control studies with 199 cases and 1451 controls Paternal farm work OR = 2.3 (1.3–4.1) and maternal farm work OR = 3.9 (1.6–9.9) during pre-conception and gestation period After adjustment for farm residency OR = 1.4 (0.3–6.2) and OR = 1.7 (0.7–4.0) respectively	No IARC classification for this site.

Table 2 (*Continued*)

Cancer type	Occupational exposure	Risk level (95% confidence intervals)	IARC Classification
Childhood brain tumor	Pesticides	**Vinson et al.** [164]: Meta-analysis of 40 studies Paternal occupational exposure (11 case control studies) OR = 1.40 (1.20–1.62); Maternal occupational exposure non-significant Paternal prenatal exposure (9 studies/OR) OR = 1.49 (1.23–1.79); Maternal prenatal exposure non-significant Parental exposure to herbicides (16 studies/OR) OR = 1.31(1.08–1.60) Parental exposure to insecticides (24 studies/OR) OR = 1.18 (1.06–1.33) Parental exposure to fungicides (15 studies/OR) OR = 1.32 (1.06–1.65) **Kristensen et al.** [175]: Registry based cohort study including 323,292 children and 1275 cancer Parents farm holders, risk adjusted for year of birth and calendar year Risk of brain tumors ($n = 41$) RR = 1.71 (1.11–2.63) Risk of non-astrocytic neuroepithelial tumors ($n = 22$) RR = 3.37 (1.63–6.94) **Feychting et al.** [160]: Registry linkage study (235 635 children, 162 nervous system tumors) paternal pre-conceptional occupational exposure to pesticides RR = 2.36 (1.27–4.39), adjusted for maternal age, socio-economic status, census year and gender	No IARC classification for this site.
	Painting	**Feychting et al.** [160]: Registry linkage study (235 635 children, 162 nervous system tumors) paternal pre-conceptional work as painter RR = 3.65 (1.71–7.80) Adjusted for maternal age, socio-economic status, census year and gender	Group 1. No classification for this site.
	Polycyclic aromatic hydrocarbons (PAH)	**Cordier et al.** [179]: Case control study including 1218 cases and 2223 controls); adjusted for maternal age, sex of child, mother's level of education Paternal pre-conceptional occupational exposure to PAH All childhood brain tumors OR = 1.3 (1.1–1.6) Astroglial tumors OR = 1.4 (1.1–1.7) Astroglial tumors after exclusion of smoking fathers OR = 1.7 (1.3–2.3) Maternal occupational exposure OR = 1.11 (0.88–1.40 **Johnson et al.** [178]: Case control study with 499 childhood death from intracranial or spinal cord tumors and 998 controls Parental occupation with high PAH exposure: printers and graphics arts workers OR = 4.5 (1.4–14.7); chemical and petroleum workers levels: OR = 3.0 (1.1–8.5)	Some PAH-related exposures are Group 1 but none of them for this site of cancer. (Vol. 100 F, 2012)
	Diesel exhaust	**Peters et al.** [180]: Case control study including 306 cases and 950 controls Maternal exposure before child's birth OR = 2.03 (1.09–3.81) and paternal exposure around the time of the child's conception OR = 1.62 (1.12–2.34)	Group 1. No classification for this site. (Vol. 105, in prep, meeting in 2012)
Neuro-blastoma	Parental exposure to pesticides	**Vinson et al.** [164]: Meta-analysis OR = 1.70 (1.14–2.51)	No IARC classification for this site.

*3.1.5. Epithelial tumors of the larynx**

IARC has classified asbestos and acid mists as carcinogenic for larynx (sufficient evidence) as well as the rubber industry (limited evidence) [10,22,23]. The association with asbestos is supported by numerous studies and a meta-analysis [24,25]. Furthermore, meta-analyses have been conducted for several other occupational risks. Thus, Chen et al. reported evidence for an association with silica dust, although only the pooled risk from case–control studies was statistically significant [26]. Moreover, Paget-Bailly et al. identified significant increased meta-relative risks associated with polycyclic aromatic hydrocarbons, engine exhaust, and textile dust [27]. A relationship with wood dust exposure has also been suggested, but a meta-analysis did not confirm this finding [27]. Similarly, although laryngeal cancer was suggested to be linked to chlorinated solvent exposure, this could

Table 3
Summary of the available evidence by cancer site.

Cancer type	Occupational exposure (Group 1 carcinogen) with sufficient evidence supported by IARC classification	Occupational exposure with limited evidence supported by IARC classification or evidence from meta-analysis ([a])	Comments
Head and neck cancers			
Cancer of the nasal cavity and sinuses	Wood dust, Nickel, Leather dust, Isopropyl alcohol production, radium	Formaldehyde, Chromium, carpentry and joinery, textile manufacturing	
Naso-pharyngeal cancer (NPC)	Formaldehyde, Wood Dust		
Cancers of the salivary gland			Insufficient evidence[c]
Hypo-pharyngeal cancer		Asbestos	
Laryngeal cancer	Asbestos, Acid mists	Rubber industry Passive smoking at work, Sulfur mustard, Engine exhaust[a], Silica dust[a], Polycyclic aromatic hydrocarbons[a], Textile dust[a]	
Pharyngeal/Oropharyngeal cancer			Insufficient evidence
Cancer of the oral cavity			Insufficient evidence
Cancer of the lip		Outdoor work (solar radiations)	
Gastrointestinal cancers			
Squamous cell carcinoma of the esophagus			Insufficient evidence; Covered by the IARC classification for esophagus cancer[b]
Adenocarcinoma of the esophagus			Insufficient evidence; Covered by the IARC classification for esophagus cancer[b]
Carcinoma of the small intestine			Insufficient evidence
Cancer of the anal canal			No relevant data identified
Hepatocellular carcinoma of the liver and intrahepatic bile tract (IBT)	Vinyl chloride monomer, Plutonium, Thorium, Aflatoxin, Hepatitis B, Hepatitis C	Polychlorinated biphenyls, Trichloroethylene, Arsenic Radon in underground miners[a]	
Hepatic angiosarcoma*	Vinyl chloride monomer		
Epithelial tumors of gallbladder and extrahepatic biliary tract (EBT)			Insufficient evidence
Thoracic cancers			
Epithelial tumor of the trachea			No relevant data identified
Large cell lung carcinoma	Asbestos, Diesel exhaust		Covered by the IARC classification for lung cancer[b]
Bronchiolo-alveolar lung carcinoma			No relevant data identified
Epithelial tumors of the thymus			No relevant data identified
Mesothelioma	Asbestos, Erionite, Painting		
Reproductive cancers			
Mammary Paget's Disease			No relevant data identified
Epithelial tumors of the male breast			Insufficient evidence; Covered by the IARC classification for breast cancer[b]
Epithelial tumors of the cervix uteri			Insufficient evidence
Ovarian cancer	Asbestos		
Tumors of the vulva and vagina			No relevant data identified

Table 3 (*Continued*)

Cancer type	Occupational exposure (Group 1 carcinogen) with sufficient evidence supported by IARC classification	Occupational exposure with limited evidence supported by IARC classification or evidence from meta-analysis ([a])	Comments
Urogenital cancers			
Testicular cancer			Insufficient evidence
Extragonadal germ cell tumors			No relevant data identified
Penis carcinoma			No relevant data identified
Squamous cell carcinoma of the kidney			No relevant data identified; Covered by the IARC classification for kidney cancer[b]
Non-transitional cell carcinoma of the urinary bladder			No relevant data identified; Covered by the IARC classification for bladder cancer[b]
Non-bladder urinary organs (renal pelvis, ureter, urethra)	Aristochloric acid	PAH	Transitional cell carcinoma of non-bladder urinary organs are covered by IARC classification for bladder cancer[b]
Neuroectodermic and mesodermic tumors			
Malignant melanoma of mucosa			No relevant data identified
Epithelial tumors of the eye and adnexa	Welding	Solar radiation	
Malignant melanoma of uvea	Welding	Solar radiation	
Soft-tissue sarcoma		Polychlorophenol, 2,3,7,8-tetrachloro-dibenzo-p-dioxin	
Bone sarcoma	Ionizing radiation		
Glial tumors of the central nervous system			Insufficient evidence
Malignant meningioma			Insufficient evidence
Neuroendocrine tumors			No relevant data identified
Endocrine tumors			
Carcinoma of the pituitary gland			No relevant data identified
Carcinoma of the thyroid gland			Insufficient evidence
Carcinoma of the parathyroid gland			No relevant data identified
Carcinoma of the adrenal gland			No relevant data identified
Lymphoid diseases			
Hodgkin disease			Insufficient evidence
Acute lymphoblastic leukemia		Benzene	
Burkitt lymphoma			No relevant data identified;
Cutaneus T cell lymphoma/mycosis fungoides			Insufficient evidence
Other T cell lymphoma and NK cell neoplasms			Insufficient evidence; Covered by the IARC classification for lymphoma[b]
Diffuse large B-cell lymphoma (DLBCL)		Benzene, Trichloroethylene	Covered by the IARC classification for lymphoma[b]
Follicular B lymphoma (FL)		Benzene, Trichloroethylene	Covered by the IARC classification for lymphoma[b]
Hair cell leukemia			Insufficient evidence; Covered by the IARC classification for lymphoma[b]
Multiple myeloma (MM)		Benzene, Ethylene oxide Pesticides[a]	
Other non-Hodgkin, mature B cell lymphoma			
Chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL)		Benzene, Ethylene oxide	Insufficient evidence for SLL

Table 3 (*Continued*)

Cancer type	Occupational exposure (Group 1 carcinogen) with sufficient evidence supported by IARC classification	Occupational exposure with limited evidence supported by IARC classification or evidence from meta-analysis ([a])	Comments
Marginal zone lymphoma/MALT lymphoma			Insufficient evidence; Covered by the IARC classification for lymphoma[b]
Mantle cell lymphoma			No relevant data identified; Covered by the IARC classification for lymphoma[b]
Lymphoplasma-citic lymphoma/macroglobulinemia Waldestrom			No relevant data identified
Acute myeloid leukemia	Benzene, Formaldehyde		
Chronic myeloid leukemia	Formaldehyde, Radiation		
Other myelodysplastic and myeloproliferative neoplasms			Insufficient evidence
Histiocytic and dendritic cell neoplasms			No relevant data identified
Childhood cancer			
Childhood leukemia		Parental exposure to paintings Parental pesticides exposure[a]	
Childhood lymphoma		Parental pesticides exposure[a]	
Childhood brain tumor		Parental pesticides exposure[a]	
Neuroblastoma			Insufficient evidence
Ewing sarcoma		Parental pesticides exposure[a]	
Wilms' tumor			Insufficient evidence
Other childhood cancer			Insufficient evidence

[a] Supported by meta-analyses.
[b] http://monographs.iarc.fr/ENG/Classification/index.php (accessed October 2013).
[c] Insufficient evidence for association with occupational exposure.

not be verified in a review of the literature [21,27]. Machining fluids have also been associated with laryngeal cancer, but results are inconsistent [27]. Finally, several carcinogens that are known to affect other sites have been reported to increase the risk of laryngeal cancer, including second hand smoke and ionizing radiation [28,29], whereas other suggested occupational factors might be seen as more anecdotal (e.g., extremely low frequency magnetic fields) [30].

3.1.6. Epithelial tumors of the oropharynx**

Few studies have focused on oropharyngeal cancers separately. When considering pharyngeal cancers, some occupational factors have been identified in the literature (i.e., sulfur mustard [31] and welding fumes [32]).

3.1.7. Epithelial tumors of the oral** cavity and lip*

IARC classified solar radiations as carcinogenic for lip (limited evidence). Wood dust has been suggested as a risk factor for cancer of the oral cavity [33]. Additionally, there is an increased risk of lip cancer associated with outdoor occupations (e.g., construction workers, seafarers, farmers) [34] and outdoor occupational activities [35].

3.2. Gastrointestinal cancers

3.2.1. Squamous cell carcinoma and adenocarcinoma of the esophagus**

IARC classified dry cleaning and rubber industry production as carcinogenic for esophagus (no distinction of histology subtype) with limited evidence [10].

For squamous cell carcinoma, associations with several agents have been suggested, including carbon black, sulfuric acid, asbestos, and polycyclic aromatic hydrocarbons (PAHs) [32,36]. On the other hand, adenocarcinoma has been linked to sulfur compounds and lead [37].

3.2.2. Carcinoma of the small intestine**

IARC has classified asbestos as carcinogenic for colon and rectum with limited evidence [10]. Nevertheless, a cohort study found a significant increase in the risk of small intestine cancer among men highly exposed to asbestos [38]. Additionally, an increased risk of small bowel adenocarcinoma has been found among workers exposed to semiautomatic arc welding [39]. However, no significant association was observed with other categories of welding or with welding activity in general. An elevated risk of small bowel carcinoid tumors was also reported for organic solvent-exposed

workers [40]. More recently, an increased risk for cancer of the small intestine has been suggested among workers exposed to benzidine [41].

3.2.3. Epithelial tumors of the anal canal

No relevant data identified.

3.2.4. Hepatocellular carcinoma of the liver and intrahepatic bile tract (IBT)*

IARC has classified several occupational exposures as carcinogens for liver and bile duct with sufficient evidence (aflatoxin, hepatitis B and C, plutonium, thorium-232 and its decay products, vinyl chloride) or limited evidence (arsenic, polychlorinated biphenyls, and trichloroethylene).

Aflatoxin is known to induce liver cancer, mainly via food contamination. However, workplace exposure to aflatoxin was also suggested to increase the risk of liver cancer in workers in the animal-feed processing industry [42] and other occupations [43,44].

Several other occupational exposures have been linked to liver cancer, in particular vinyl chloride monomer, organic solvents and polychlorinated biphenyls [45–52]. In fact, vinyl chloride monomer has been known to induce hepatic angiosarcoma for decades, and epidemiological studies have identified the association of this type of exposure with hepatocellular carcinoma [48]. An increased risk of liver cancer has been observed in workers exposed to radon [53,54] as well as in plutonium workers [55]. Magnetic fields were also associated with cancer at this site [30].

*3.2.4.1. Hepatic angiosarcoma**. IARC has concluded that there is a causal relationship between hepatic angiosarcoma and vinyl chloride exposure (sufficient evidence). The strong association is supported by a positive dose–response relationship [5,56].

3.2.5. Epithelial tumors of gallbladder and extrahepatic biliary tract (EBT)**

IARC has classified thorium-232 and its decay products as carcinogenic for this site with sufficient evidence. Radon exposure has been reported to increase the risk of gallbladder, and extra hepatic bile ducts [54].

3.3. Thoracic cancers

3.3.1. Epithelial tumor of the trachea

No relevant data identified.

3.3.2. Large cell lung carcinoma*

IARC has classified numerous occupational exposures as carcinogens for lung with sufficient evidence (aluminum production, arsenic and inorganic arsenic compounds, asbestos, beryllium and compounds, bis(chloromethyl)ether; chloromethyl, methyl ether, cadmium and compounds, chromium(VI) compounds, coal, coal gasification, coal-tar pitch, coke production, diesel engine exhaust, hematite mining, iron and steel founding, nickel compounds, painting, plutonium, radon-222 and its decay products, rubber production industry, crystalline silica dust, soot, sulfur mustard, secondhand tobacco smoke, X-radiation, gamma-radiation) or limited evidence (strong inorganic acid mists, manufacture of glass, bitumens, exposure to oxidized and hard bitumens, carbon electrode manufacture, alpha-chlorinated toluenes and benzoyl chloride, cobalt metal with tungsten carbide, creosotes, occupational exposures in spraying and application of insecticides, printing processes, 2,3,7,8-tetrachlorodibenzopara-dioxin, welding fumes) [10].

Few reports have distinguished cases based on histological subtype, and only two studies on large cell lung carcinoma were identified, which showed association with two common lung carcinogens: asbestos and diesel exhaust [57,58].

3.3.3. Bronchioloalveolar carcinoma of the lung

No relevant data identified.

3.3.4. Epithelial tumors of the thymus

No relevant data identified.

3.3.5. Mesothelioma*

Asbestos, erionite and painting are IARC group 1 carcinogens for mesothelioma with sufficient evidence [10]. Hundreds of studies have investigated the link between asbestos exposure and mesothelioma [3,59,60]. The first series of asbestos-associated pleural mesothelioma cases was reported in 1960 [61]. The risk for mesothelioma seems to be higher for some categories of fibers (amphiboles) [2].

3.4. Reproductive cancers

3.4.1. Mammary Paget's disease of the breast

No relevant data identified.

3.4.2. Epithelial tumors of the male breast**

IARC has concluded that shift work involving circadian disruption is associated with female breast cancer (limited evidence). Some occupations have been found to be at increased risk for male breast cancer, and exposure to electromagnetic fields [EMFs], polycyclic aromatic hydrocarbons, herbicides, pesticides, and organic solvents have been suggested to increase risk. Nevertheless, most studies focusing on specific exposures failed to find significant increases in associated risk [62]. However, an increase in risk was associated with gasoline motor exhaust [63]. More recently, male breast cancer was suggested to be associated with alkyl phenolic compounds that are endocrine disrupting chemicals [64].

3.4.3. Epithelial tumors of the cervix uteri**

Organic solvents (particularly trichloroethylene) are suggested to be the principal occupational exposure associated with cervical cancers [50,65]. Increased risk has also been

reported for exposure to metalworking fluids [66]. However, epidemiological studies that reported on this association did not adjust for well-known risk factors, including HPV. In addition, histological subtypes have not been well documented, and results have been inconsistent.

3.4.4. Adenocarcinoma of the ovary*

Ovarian cancer is one of the four cancers considered by the IARC to show sufficient evidence for association with asbestos exposure [67]. In addition, several other occupational exposures were shown to significantly increase the risk for ovarian cancer, including silica dust, diesel exhaust, and organic solvents; however, evidence is sparse [68–70]. Also, reports do not distinguish between histological subtypes of ovarian cancer.

3.4.5. Epithelial tumors of the vulva and vagina

No relevant data identified.

3.5. Urogenital cancers

3.5.1. Testicular cancer**

Several occupational exposures have been suggested to be associated with TGCT, such as EMF exposure, polychlorinated biphenyls, and pesticides. In addition, several occupations have been linked to TGCT, including agricultural workers, firemen, policemen, military personnel, and industrial workers (paper, plastic or metal). However, available evidence has not permitted the identification of strong and consistent occupational risk factors for TGCT [71]. Since testicular cancer occurs mainly in young adults, the potential impact of occupational exposure of mothers to endocrine disruptors during intra-uterine development has also been suggested [71,72]. However, data supporting this notion are sparse and inconsistent.

3.5.2. Extragonadal germ cell tumors

No relevant data identified.

3.5.3. Epithelial tumors of the penis

No relevant data identified.

3.5.4. Squamous cell carcinoma of the kidney

IARC has classified trichloroethylene as Group 1 carcinogen with sufficient evidence for kidney [10]. IARC concluded that there is a positive association (limited evidence) with arsenic and inorganic arsenic compounds, cadmium and cadmium compounds, and printing processes [10]. We did not identify any studies on this histology sub-type.

3.5.5. Non-transitional cell carcinoma of the urinary bladder

IARC has classified numerous occupational exposures as carcinogens (Group 1) for bladder (no distinction of histology subtype) with sufficient evidence (aluminum production, 4-aminobiphenyl, arsenic and inorganic arsenic compounds, auramine production, benzidine, magenta production, 2-naphthylamine, painting, rubber production industry, ortho-toluidine, X-radiation, gamma-radiation) and limited evidence (4-chloro-ortho-toluidine, coal-tar pitch, diesel engine exhaust, dry cleaning, occupational exposure of hairdressers and barbers, printing processes, soot, textile manufacturing, tetrachloroethylene). Occupational risks for bladder cancer have been mainly evaluated in regard to transitional cell carcinoma; we did not identify any studies investigating occupational exposures in other histology subtypes (i.e., adenocarcinoma, squamous cell carcinoma).

3.5.6. Non-bladder urinary organs (renal pelvis, ureter, and urethra)*

IARC has classified aristochloric acids as carcinogenic for ureter and renal pelvis (sufficient evidence). Occupational risks for renal pelvis cancer (>90% transitional cell carcinoma) are those established for bladder cancer [73–75]. Associations have also been observed with exposure to coke, coal, asphalt, tar products, and certain refining industries (mineral oil, chemical, and petroleum) [75].

3.6. Neuroectodermic and mesodermic tumors

3.6.1. Malignant melanoma of the mucosa

No relevant data identified.

3.6.2. Epithelial tumors of the eye and adnexa*

IARC has classified welding with sufficient evidence and solar radiation with limited evidence for eye.

No occupational exposure study could be identified for this type of cancer, except for melanoma.

3.6.3. Malignant melanoma of the uvea*

IARC has classified welding with sufficient evidence and solar radiation with limited evidence for eye. Occupational exposure to ultraviolet radiation has been described to increase eye melanoma in workers exposed during outdoor occupational activities and welders [76–78].

3.6.4. Soft-tissue sarcoma (STS)*

IARC classified 2,3,7,8-tetrachlorodibenzopara-dioxin (Group 1) and polychlorophenols and their sodium salts (Group 2B) as carcinogenic for STS (limited evidence). We did not identify other occupational risk factors for this cancer site [79–81].

3.6.5. Bone sarcoma*

IARC has classified radium (Group 1) with sufficient evidence for association with bone sarcoma. An increased risk was shown mainly in radium-dial painters [9]. Also, epidemiological evidence supports an increased risk of bone cancer in plutonium workers [22]. Moreover, epidemiological studies indicated that bone sarcoma is associated with exposure to medical X- or gamma-radiation. However, large cohorts on nuclear workers failed to display increased risk for bone

sarcoma, mainly due to small doses of exposure [82]. Furthermore, an increased risk was reported for workers who had been exposed to pesticides at work [83].

3.6.6. Glial tumors of the central nervous system**

IARC has classified radiofrequency electromagnetic field (including from wireless phones) as carcinogenic for the brain and central nervous system (Group 2B) [84], but our review did not identify data supporting association with occupational EMF exposure. Furthermore, low frequency EMF exposure has been suggested to increase the risk of glioma, but results have been inconsistent [85–88]. Glioma was suggested to be associated with farming activities and exposure to pesticides, but these findings have not been confirmed [89–93]. In addition, an increased risk of glioma was recently suggested in workers exposed to carbon tetrachloride [94].

3.6.7. Malignant meningioma**

An increased risk of meningioma in workers exposed to lead has been described [95–97]. Also, an association between meningioma and EMF exposure was reported with inconsistent results [86–88], and evidence supporting meningioma risk and pesticides is conflicting [91,93].

3.7. Neuroendocrine tumors

No relevant data identified.

3.8. Endocrine tumors

3.8.1. Carcinomas of the pituitary gland

No relevant data identified.

3.8.2. Carcinomas of the thyroid gland**

According to IARC, there is sufficient evidence that ionizing radiation causes thyroid cancer, but not for occupational exposures (except under conditions of exposure during nuclear accidents) [10]. However, some epidemiological studies have suggested an excess risk among subjects occupationally exposed to ionizing radiation [98–100]. An increased risk of thyroid cancer has also been reported in women exposed to organic solvents [101].

3.8.3. Carcinomas of the parathyroid gland

No relevant data identified.

3.8.4. Carcinomas of the adrenal gland

No relevant data identified.

3.9. Lymphoid diseases

IARC has concluded that there is a causal relationship between lymphoma and several carcinogens with sufficient evidence (rubber industry, 1,3-butadiene) or limited evidence (benzene, tetrachlorodibenzo-para-dioxin, polychlorinated biphenyls, ethylene oxide, trichloroethylene). Below (Sections 3.9.2–3.9.6), we only report on subtype-specific associations.

3.9.1. Hodgkin disease (HD)**

No strong association has been found with specific occupational exposures. Several studies and meta-analyses have suggested an association with pesticide exposure [102–104]. Also, studies have indicated an increased risk of HD associated with wood dust exposure or wood-related occupations [105,106]; however, data are inconsistent and evidence supporting a causal role of occupational wood dust exposure is lacking according to IARC. Meta-analyses showed no increased risk associated with occupational benzene or other solvent exposure [107]. Finally, elevated risk for HD, particularly nodular sclerosis, has been reported for subjects exposed to immunologically active agents [108].

3.9.2. Acute lymphoblastic leukemia (ALL)*

According to IARC the relation of ALL and occupational benzene exposure is supported by limited evidence. The association is supported by meta-analyses evidence [107]. ALL is one of the main radiogenic leukemia subtypes, but epidemiological data on occupational radiation exposure is insufficient. Associations with occupational EMF exposure has been suggested [109], but evidence is also insufficient.

3.9.3. Burkitt leukemia/lymphoma

No relevant data identified.

3.9.4. Cutaneous T cell lymphoma/mycosis fungoides**

Studies on occupational exposures that might be associated with this subtype are rare. However, associations have been suggested for occupational hydrocarbon and sun exposure [110–112].

3.9.5. Other T cell lymphomas and NK cell neoplasms**

Studies regarding other T cell lymphomas and occupational exposures are sparse. Data from the European Epilymph study did not support association with solvent exposure [113].

3.9.6. Diffuse large B-cell lymphoma (DLCBL)* and follicular B lymphoma (FL)*

Occupational benzene exposure was found to be positively associated with DLBCL and FL in several studies, although the relationship was not significant in all studies [114–118]. Overall, a positive association with solvents has been reported [113,115,117,119,120], although this appears to be more consistent for FL than for DLBCL. Also, an association of DLBCL with formaldehyde exposure was suggested [117]. For FL, a significant association with combined exposure to benzene, toluene, and xylene has been reported [113]. Furthermore, FL has been linked to occupational pesticide exposure [102,121].

*3.9.7. Hairy cell leukemia (HCL)***

No strong association has been shown with occupational exposures. A positive association was suggested for farming [102,122,123]. So far, the evidence linking organic solvents and exhaust fumes to HCL has been insufficient.

*3.9.8. Multiple myeloma (MM)**

According to IARC, a positive association of MM with benzene exposure exists (limited evidence), supported by epidemiology studies [124–127]. Furthermore, IARC concluded that there is a positive association with ethylene oxide exposure (limited evidence). A consistent, but modest, increased risk of multiple myeloma has been suggested for occupational pesticides exposure [73,102,128–130]. Residents and/or workers of sheep farms have shown an increased risk of MM, indicating that certain animal viruses may be involved. Moreover, a meta-analysis has suggested an association with engine exhaust [127]. An inconsistent association has been shown with chlorinated solvents [113,115,131] and for work in the rubber industry [126]. Finally, associations with occupational exposure to TCDD, formaldehyde, and radiation have been reported [126,132,133].

3.9.9. Other non-Hodgkin, mature B cell lymphoma

3.9.9.1. Chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL)***. IARC concluded that there is a positive association (limited evidence) with benzene and ethylene oxide exposure [126]. Three meta-analyses have reported on elevated CLL risk associated with occupational exposure to benzene, but the findings were not significant in two of them [107,134,135]. The association with ethylene oxide exposure is supported by some epidemiological findings [136], Moreover, a positive association with other solvents has been reported for CLL and SLL [113,120,137], but overall the results are inconsistent [117,119,135]. Furthermore, an increase in CLL risk has been reported for occupational 1,3-butadiene exposure [138]. A positive association was identified with occupational exposure to several classes of pesticides, but evidence also remains insufficient for these findings [102,139,140]. Recent studies have suggested an association with low doses of external ionizing radiation, but again the evidence was inconsistent [133,141–143]. Finally, a meta-analysis reported a non-significant increase in risk associated with EMF exposure [109].

*3.9.9.2. Marginal zone lymphoma/MALT lymphoma***. Evidence for association of these types of lymphomas with occupational exposure is sparse; however, an association with chlorinated solvents has been suggested [115]. Occupational animal exposure has also been linked with ocular adnexal marginal zone B-cell lymphoma, but the evidence remains insufficient [144].

3.9.9.3. Mantle cell lymphoma/centrocytic lymphoma. No relevant data identified.

3.9.9.4. Lymphoplasmacytic lymphoma/macroglobulinaemia Waldenstrom. No relevant data identified.

*3.10. Acute myeloid leukemia (AML)**

IARC concluded that there is sufficient evidence for a causal relationship between AML and benzene exposure [124,126,134]. Moreover, IARC concluded that there is sufficient evidence for a causal link between leukemia and formaldehyde exposure [126]. Evidence from meta-analyses support the associations of AML with both, benzene and formaldehyde [124,126,132,134]. Furthermore, a positive association was found with 1,3-butadiene exposure, with a significant dose–response relationship [126], results not confirmed by a recent study [138]. AML is a radiogenic subtype [22], and an increased risk has been shown in a cohort of US nuclear workers [145]. Meta-analyses have indicated an increased risk associated with occupational exposure to pesticides [146]. Furthermore, an increased risk among workers exposed to diesel exhaust has been suggested [147], but the findings are inconsistent. Additionally, some studies have suggested a positive association with EMF exposure, but evidence for this remains insufficient [109].

3.11. Myeloproliferative neoplasms

*3.11.1. Chronic myeloid leukemia**

The IARC concluded that there was sufficient evidence to support a causal relationship between formaldehyde exposure and myeloproliferative neoplasms [126]. Evidence from meta-analyses supports this association [132]. Moreover, according to IARC, there is sufficient evidence that ionizing radiation causes leukemia. CML is a radiogenic subtype and positive associations have been observed [22]. Numerous epidemiologic studies and meta-analyses have investigated the association of CML with benzene exposure, reporting inconsistent results [124,134,135,148,149]. The most recent meta-analyses provides solid arguments for increasing relative risks with increasing exposure assessment quality [124]. Also, associations have been suggested for occupational exposure to pesticides [146]. Data regarding association with 1,3-butadiene are inconsistent [138,150].

*3.11.2. Other myeloproliferative and myelodysplastic neoplasms***

Although data linking occupational factors to other myeloproliferative and myelodysplastic neoplasms is sparse, associations with benzene, pesticides, and radiation have been suggested [135,151,152].

3.12. Histiocytic and dendritic cell neoplasms

No relevant data identified.

3.13. Childhood cancer

3.13.1. Childhood leukemia (CL)*

IARC concluded that there is a positive association between maternal exposure to painting and childhood leukemia (limited evidence). In numerous case–control studies, a positive association was reported between maternal exposure to painting (including pre-conception and during pregnancy) and AML (myelocytic, myelogenous, or non-lymphoblastic leukemia) or ALL (lymphoblastic or lymphoid leukemia) in offspring [153–157]. In contrast, the association with paternal exposure to paints has been inconsistent for both ALL and AML [153–155,157–161]. Also, three meta-analyses [162–164] have reported an increased risk of CL associated with parental exposure to pesticides; however, the leukemia subtype, exposure, and periods considered differed among the studies. Moreover, an association between CL and parental exposure to benzene and solvents has been suggested [155,158,159,165–169]. However, evidence on solvent exposure before conception has been inconsistent. While prenatal exposure to ionizing radiation, mainly through maternal medical exams, has been shown to cause CL [126], studies regarding maternal or paternal occupational radiation exposure (pre-conception or during pregnancy) and CL risk have shown inconsistent results [168,170,171]. Furthermore, data related to parental occupational EMF exposure have been inconsistent, and overall suggested a lack of association, in coherence with IARC monographs [161,168,172–174]. Finally, other factors that are suggested to be linked to CL include exposure to motor exhaust fumes, plastic materials, or woodwork (father). Nevertheless, it is impossible to draw any firm conclusions from these data.

3.13.2. Lymphoma*

A meta-analysis has suggested increased risk associated with parental exposure to pesticides [164]. Also, an increased risk of non-Hodgkin's lymphoma was suggested with maternal exposure to oil products, unspecified chemicals [168], and ionizing radiation [170].

3.13.3. Childhood brain tumors (CBT)*

A meta-analysis and two large scale registry-based cohort studies found a significantly increased risk of CBT for paternal [160,164] or parental [175] occupational pesticide exposure. Moreover, differences according to histological subtype were found (i.e., a positive association was reported for astrocytoma, whereas no association was found with primitive neuroectodermal tumors [PNET]) [176,177]. Maternal pesticide exposure was inconsistently found. Evidence also suggested an association between CBT and parental occupational exposure to polycyclic aromatic hydrocarbons (PAH) [178,179], paint [160], and diesel exhaust [180]. Furthermore, some parental occupations were associated with an increased risk of CBT, including mechanics or drivers [181], chemical industry employees [182,183], and electrical workers [182].

3.13.4. Neuroblastoma**

A meta-analysis suggested increased risk associated with parental occupational pesticide exposure [164], but the evidence in support of this is insufficient. Data related to other parental occupational exposures are sparse and inconsistent, so conclusions cannot be drawn [184,185].

3.13.5. Ewing sarcoma*

Meta-analyses reported a significant increase in risk of Ewing sarcoma associated with paternal occupational pesticide exposure or farm work [164,186]; however, the data regarding maternal exposure to these factors were divergent. Evidence related to Ewing sarcoma and other parental occupational exposures is sparse and inconsistent [187].

3.13.6. Wilms' tumor**

Current meta-analyses do not support an association of Wilms' tumor with parental occupational exposure to pesticides [164]. Evidence related to Wilms' tumor and other parental occupational exposures is sparse and inconsistent [188].

3.13.7. Other childhood cancer**

Parental occupational exposure in other childhood cancers has been poorly studied. According to IARC a causal relation (sufficient evidence) exists between solid childhood cancer and prenatal radiation exposure, in Japanese A-bomb survivors and through medical exams. However, data on parental occupational radiation exposure are insufficient.

4. Discussion

Improving prevention and clinical management of rare cancers are important goals in cancer control. To our knowledge, this is the first comprehensive review on established and suspected occupational exposures associated with rare cancers. The term "rare cancer" recovers a heterogeneous group of malignancies, i.e., cancers of rare occurrence, rare histology subtypes and rare localizations of common tumors. Although the RARECARE rarity threshold (<6/100,000) might be considered too high, it has the advantage to be the subject of an international consensus [1], and provided a practical basis for selecting cancers for our review. The common histologic subtypes of lung and bladder cancer, the malignancies most often linked to occupational etiologies, were beyond the scope of this review because of their frequencies.

For 40% of the rare cancers included in our review, a positive association with exposure to occupational carcinogens is supported by IARC monographs and/or consistent evidence from epidemiology studies. However, for many others, the evidence is lacking or remains sparse and inconsistent.

The low incidence of rare cancers often constitutes a barrier to conducting adequately powered epidemiological studies. This could partly explain the inconsistent results reported [111]. Also, in many studies, not designed to investigate histologic subtypes, subtype-specific risk estimates are associated with large 95% confidence intervals and should be interpreted with caution [7,41,78,111]. Furthermore, positive associations might be due to chance when multiple testing is performed [189]. Although for some occupations the job title can be used as surrogate for occupational exposure (e.g., welders and welding fumes), the reliability of the exposure assessment might be limited for others (e.g., farmers). Also, heterogeneity of exposure definitions and exposure data collected might further explain inconsistencies across studies [190,191]. Furthermore, evolution of industrial practices could explain why some previously identified associations were not confirmed in more recent studies. Well-conducted meta-analyses can provide more solid evidence, in particular when individual studies are inconsistent or insufficiently powered, such as for parental pesticide exposure and risk of childhood leukemia [162–164]. This is why our review considered in priority evidence from meta-analyses.

Lack of subtype-specific histopathology details led to the exclusion of many studies. Also, advances in genetic and molecular biology have led to improvements in tumor subtyping (e.g., hematopoietic malignancies and sarcomas) and the evolution of classifications over time [192–194]. For example, the definition of NHL has evolved over the last decades and now includes previously distinct disorders (e.g., CLL, MM), analyzed separately in past epidemiological studies [193,194]. Furthermore, certain genetic polymorphisms of enzymes involved in xenobiotic metabolism may lead to augmented risk for some individuals [95,195]. However, such contributing factors were rarely considered in the reviewed studies.

In addition to epidemiology studies, IARC classification of human carcinogens takes into account animal and in vitro studies [196]. Thus, animal studies have confirmed that inhalation of asbestos fibers induces lung cancer and malignant mesothelioma, and several direct and indirect mechanisms have been proposed [197]. Likewise, animal studies and mechanistic evidence have shown that the genotoxic effects of benzene at the level of the pluripotent hematopoietic stem cells result in chromosomal changes. These are consistent with alterations seen in hematopoietic malignancies and circulating lymphocytes of benzene-exposed workers [198,199].

Our study presents several limitations. Given the objectives of our review, after careful review of the evidence for the 48 cancer sites for which relevant data were identified, we recorded occupational exposures supported by IARC classifications, meta-analyses or consistent evidence from several epidemiology studies. For selected articles, we retrieved the most relevant data and provided adjusted risks when available. Also, we reported occupational exposures inconsistently associated with a given cancer site or supported by insufficient evidence. However given the wide range of cancers included in our review, it would have been impossible to record all studies with limited or negative results. Furthermore, the majority of publications in our review were identified through PubMed. While we can be confident that our combined search strategy enabled us to identify the relevant studies for a given cancer site, we did not perform a cross search for occupational exposures identified. Therefore we cannot exclude that some studies focusing on multiple cancer sites with negative results for a given cancer site, may have been ignored.

In conclusion, the main rare cancer sites with strong and consistent evidence were nasal, larynx, liver and ovarian cancer, mesothelioma, bone sarcoma, transitional cell carcinoma of non-bladder urinary organs and hematopoietic malignancies. The main exposures for these cancers were asbestos, wood dust, metals/metalloids, formaldehyde, benzene, vinyl chloride, and radiation. This review provides useful information to clinicians to pay greater attention to work-related exposures in patients with rare cancers. Five percent of cancers are of occupational origin [200], but occupational cancers remain largely under-reported with important spatial disparities [201,202]. The risk of not seeking compensation is significantly higher for women and the elderly [203]. Furthermore, care for people with occupational cancers represents an unnecessary financial burden and productivity loss [204]. Due to the latency between exposure and cancer development, work-related cancers often appear after retirement, making it important for clinicians to investigate the former occupations and exposures of elderly patients. Moreover, patients of the socio-professional categories that are most at risk frequently display considerable horizontal occupational mobility, resulting in multiple job-related exposures that they might overlook. In many countries, patients with certain cancers matching medical and exposure conditions can ask for compensation. In the European Union, the recognition of work-related cancers is based on a list of registered occupational diseases [205]. Although several rare cancers (i.e., mesothelioma, sinonasal cancer) with well-established work-related origins are registered on various national lists as occupationally related diseases, these classifications remain heterogeneous among countries.

Future research should focus on cancer sites such as NHL or the upper aerodigestive tract. In this regard, national and international collaborative studies should allow for more adequately powered epidemiological studies. Moreover, the accurate assessment of exposure remains a challenge [191]; therefore, more sophisticated methods for assessing exposure as well as genetic and molecular biology techniques for evaluating gene–environment interactions will be important for future epidemiologic research. For instance, we are currently conducting a case–control study on TGCT to investigate the association between TGCT risk and life-time exposure using combined methodologies, including job exposure matrices, geographical information system technology, analysis of

suspected genetic polymorphisms, and socio-economic risk factors.

Conflict of interest statement

The authors declare that there is no conflict of interest.

Role of the funding source

This review received no grant from any funding agency in the public, commercial or not-for-profit sectors.

Reviewers

Derek Raghavan, Carolinas Health Care System, Levine Cancer Institute, Carolina, United States.

Gemma Gatta, Evaluative Epidemiology Unit, Department of Preventive and Predictive Medicine, Fondazione IRCCS Istituto Nazionale dei Tumori, Via Venezian 1, I-20133 Milan, Italy.

References

[1] Gatta G, van der Zwan JM, Casali PGET-AL>. Rare cancers are not so rare: the rare cancer burden in Europe. Eur J Cancer 2011;47:2493–511.
[2] Berman DW, Crump KS. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. Crit Rev Toxicol 2008;38(Suppl 1):49–73.
[3] Bourdes V, Boffetta P, Pisani P. Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. Eur J Epidemiol 2000;16:411–7.
[4] Demers PA, Kogevinas M, Boffetta P, et al. Wood dust and sino-nasal cancer: pooled reanalysis of twelve case-control studies. Am J Ind Med 1995;28:151–66.
[5] Kielhorn J, Melber C, Wahnschaffe U, Aitio A, Mangelsdorf I. Vinyl chloride: still a cause for concern. Environ Health Perspect 2000;108:579–88.
[6] IARC. IARC Monographs on the evaluation of carcinogenic risks to humans. PREAMBLE. Lyon, France: World Health Organisation Agency for Research on Cancer; 2006.
[7] d'Errico A, Pasian S, Baratti A, et al. A case-control study on occupational risk factors for sino-nasal cancer. Occup Environ Med 2009;66:448–55.
[8] Grimsrud TK, Peto J. Persisting risk of nickel related lung cancer and nasal cancer among Clydach refiners. Occup Environ Med 2006;63:365–6.
[9] Carnes BA, Groer PG, Kotek TJ. Radium dial workers: issues concerning dose response and modeling. Radiat Res 1997;147:707–14.
[10] IARC. List of Classifications by cancer sites with sufficient or limited evidence in humans, Volumes 1 to 108*; 2013 http://monographs.iarc.fr/ENG/Classification/Table 4.pdf
[11] Rosenman KD, Stanbury M. Risk of lung cancer among former chromium smelter workers. Am J Ind Med 1996;29:491–500.
[12] Hernberg S, Westerholm P, Schultz-Larsen K, et al. Nasal and sinonasal cancer. Connection with occupational exposures in Denmark, Finland and Sweden. Scand J Work Environ Health 1983;9:315–26.
[13] Luce D, Leclerc A, Begin D, et al. Sinonasal cancer and occupational exposures: a pooled analysis of 12 case-control studies. Cancer Causes Control 2002;13:147–57.
[14] Bachand AM, Mundt KA, Mundt DJ, Montgomery RR. Epidemiological studies of formaldehyde exposure and risk of leukemia and nasopharyngeal cancer: a meta-analysis. Crit Rev Toxicol 2008;40:85–100.
[15] Bosetti C, McLaughlin JK, Tarone RE, Pira E, La Vecchia C. Formaldehyde and cancer risk: a quantitative review of cohort studies through 2006. Ann Oncol 2008;19:29–43.
[16] Hildesheim A, Dosemeci M, Chan CC, et al. Occupational exposure to wood, formaldehyde, and solvents and risk of nasopharyngeal carcinoma. Cancer Epidemiol Biomarkers Prev 2001;10:1145–53.
[17] Armstrong RW, Imrey PB, Lye MS, Armstrong MJ, Yu MC, Sani S. Nasopharyngeal carcinoma in Malaysian Chinese: occupational exposures to particles, formaldehyde and heat. Int J Epidemiol 2000;29:991–8.
[18] Mirabelli MC, Hoppin JA, Tolbert PE, Herrick RF, Gnepp DR, Brann EA. Occupational exposure to chlorophenol and the risk of nasal and nasopharyngeal cancers among U.S. men aged 30 to 60. Am J Ind Med 2000;37:532–41.
[19] Wilson RT, Moore LE, Dosemeci M. Occupational exposures and salivary gland cancer mortality among African American and white workers in the United States. J Occup Environ Med 2004;46:287–97.
[20] Marchand JL, Luce D, Leclerc A, et al. Laryngeal and hypopharyngeal cancer and occupational exposure to asbestos and man-made vitreous fibers: results of a case-control study. Am J Ind Med 2000;37:581–9.
[21] Shangina O, Brennan P, Szeszenia-Dabrowska N, et al. Occupational exposure and laryngeal and hypopharyngeal cancer risk in central and eastern Europe. Am J Epidemiol 2006;164:367–75.
[22] El Ghissassi F, Baan R, Straif K, et al. A review of human carcinogens—part D: radiation. Lancet Oncol 2009;10:751–2.
[23] Steenland K. Laryngeal cancer incidence among workers exposed to acid mists (United States). Cancer Causes Control 1997;8:34–8.
[24] Goodman M, Morgan RW, Ray R, Malloy CD, Zhao K. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. Cancer Causes Control 1999;10:453–65.
[25] IOM, Effects. IoMUCoASH. Asbestos: Selected Cancers; 2006.
[26] Chen M, Tse LA. Laryngeal cancer and silica dust exposure: a systematic review and meta-analysis. Am J Ind Med 2012;55:669–76.
[27] Paget-Bailly S, Cyr D, Luce D. Occupational exposures and cancer of the larynx-systematic review and meta-analysis. J Occup Environ Med 2012;54:71–84.
[28] Lee YC, Boffetta P, Sturgis EM, et al. Involuntary smoking and head and neck cancer risk: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium. Cancer Epidemiol Biomarkers Prev 2008;17:1974–81.
[29] Dupree EA, Cragle DL, McLain RW, Crawford-Brown DJ, Teta MJ. Mortality among workers at a uranium processing facility, the Linde Air Products Company Ceramics Plant, 1943-1949. Scand J Work Environ Health 1987;13:100–7.
[30] Floderus B, Stenlund C, Persson T. Occupational magnetic field exposure and site-specific cancer incidence: a Swedish cohort study. Cancer Causes Control 1999;10:323–32.
[31] Easton DF, Peto J, Doll R. Cancers of the respiratory tract in mustard gas workers. Br J Ind Med 1988;45:652–9.
[32] Gustavsson P, Jakobsson R, Johansson H, Lewin F, Norell S, Rutkvist LE. Occupational exposures and squamous cell carcinoma of the oral cavity, pharynx, larynx, and oesophagus: a case-control study in Sweden. Occup Environ Med 1998;55:393–400.
[33] Smailyte G. Cancer incidence among workers exposed to softwood dust in Lithuania. Occup Environ Med 2012;69:449–51.

[34] Nordby KC, Andersen A, Kristensen P. Incidence of lip cancer in the male Norwegian agricultural population. Cancer Causes Control 2004;15:619–26.
[35] Kenborg L, Jorgensen AD, Budtz-Jorgensen E, Knudsen LE, Hansen J. Occupational exposure to the sun and risk of skin and lip cancer among male wage earners in Denmark: a population-based case-control study. Cancer Causes Control 2010;21:1347–55.
[36] Parent ME, Siemiatycki J, Fritschi L. Workplace exposures and oesophageal cancer. Occup Environ Med 2000;57:325–34.
[37] Santibanez M, Vioque J, Alguacil J, Barber X, Garcia de la Hera M, Kauppinen T. Occupational exposures and risk of oesophageal cancer by histological type: a case-control study in eastern Spain. Occup Environ Med 2008;65:774–81.
[38] Clin B, Morlais F, Dubois B, et al. Occupational asbestos exposure and digestive cancers – a cohort study. Aliment Pharmacol Ther 2009;30:364–74.
[39] Kaerlev L, Teglbjaerg PS, Sabroe S, et al. Occupation and small bowel adenocarcinoma: a European case-control study. Occup Environ Med 2000;57:760–6.
[40] Kaerlev L, Teglbjaerg PS, Sabroe S, et al. Occupational risk factors for small bowel carcinoid tumor: a European population-based case-control study. J Occup Environ Med 2002;44:516–22.
[41] Brown SC, Alberts R, Schoenberg M. Cancer incidence and mortality among workers exposed to benzidine. Am J Ind Med 2011;54:300–6.
[42] Autrup JL, Schmidt J, Autrup H. Exposure to aflatoxin B1 in animal-feed production plant workers. Environ Health Perspect 1993;99:195–7.
[43] Kauppinen T, Riala R, Seitsamo J, Hernberg S. Primary liver cancer and occupational exposure. Scand J Work Environ Health 1992;18:18–25.
[44] Alavanja MC, Malker H, Hayes RB. Occupational cancer risk associated with the storage and bulk handling of agricultural foodstuff. J Toxicol Environ Health 1987;22:247–54.
[45] Ahrens W, Mambetova C, Bourdon-Raverdy N, et al. Occupational exposure to endocrine-disrupting compounds and biliary tract cancer among men. Scand J Work Environ Health 2007;33:387–96.
[46] Prince MM, Hein MJ, Ruder AM, Waters MA, Laber PA, Whelan EA. Update: cohort mortality study of workers highly exposed to polychlorinated biphenyls (PCBs) during the manufacture of electrical capacitors, 1940-1998. Environ Health 2006;5:13.
[47] Mastrangelo G, Fedeli U, Fadda E, et al. Increased risk of hepatocellular carcinoma and liver cirrhosis in vinyl chloride workers: synergistic effect of occupational exposure with alcohol intake. Environ Health Perspect 2004;112:1188–92.
[48] Boffetta P, Matisane L, Mundt KA, Dell LD. Meta-analysis of studies of occupational exposure to vinyl chloride in relation to cancer mortality. Scand J Work Environ Health 2003;29:220–9.
[49] Porru S, Placidi D, Carta A, et al. Primary liver cancer and occupation in men: a case-control study in a high-incidence area in Northern Italy. Int J Cancer 2001;94:878–83.
[50] Wartenberg D, Reyner D, Scott CS. Trichloroethylene and cancer: epidemiologic evidence. Environ Health Perspect 2000;108(Suppl 2):161–76.
[51] Lynge E, Thygesen L. Primary liver cancer among women in laundry and dry-cleaning work in Denmark. Scand J Work Environ Health 1990;16:108–12.
[52] Chen R, Seaton A. A meta-analysis of mortality among workers exposed to organic solvents. Occup Med (Lond) 1996;46:337–44.
[53] Darby SC, Whitley E, Howe GR, et al. Radon and cancers other than lung cancer in underground miners: a collaborative analysis of 11 studies. J Natl Cancer Inst 1995;87:378–84.
[54] Tomasek L, Darby SC, Swerdlow AJ, Placek V, Kunz E. Radon exposure and cancers other than lung cancer among uranium miners in West Bohemia. Lancet 1993;341:919–23.
[55] Sokolnikov ME, Gilbert ES, Preston DL, et al. Lung, liver and bone cancer mortality in Mayak workers. Int J Cancer 2008;123:905–11.
[56] Elliott P, Kleinschmidt I. Angiosarcoma of the liver in Great Britain in proximity to vinyl chloride sites. Occup Environ Med 1997;54:14–8.
[57] de Klerk NH, Musk AW, Eccles JL, Hansen J, Hobbs MS. Exposure to crocidolite and the incidence of different histological types of lung cancer. Occup Environ Med 1996;53:157–9.
[58] Villeneuve PJ, Parent ME, Sahni V, Johnson KC. Occupational exposure to diesel and gasoline emissions and lung cancer in Canadian men. Environ Res 2011;111:727–35.
[59] Harding AH, Darnton AJ. Asbestosis and mesothelioma among British asbestos workers (1971-2005). Am J Ind Med 2010;53:1070–80.
[60] Loomis D, Dement JM, Wolf SH, Richardson DB. Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers. Occup Environ Med 2009;66:535–42.
[61] McDonald JC, McDonald AD. The epidemiology of mesothelioma in historical context. Eur Respir J 1996;9:1932–42.
[62] Cocco P, Figgs L, Dosemeci M, Hayes R, Linet MS, Hsing AW. Case-control study of occupational exposures and male breast cancer. Occup Environ Med 1998;55:599–604.
[63] Hansen J. Elevated risk for male breast cancer after occupational exposure to gasoline and vehicular combustion products. Am J Ind Med 2000;37:349–52.
[64] Villeneuve S, Cyr D, Lynge E, et al. Occupation and occupational exposure to endocrine disrupting chemicals in male breast cancer: a case-control study in Europe. Occup Environ Med 2010;67:837–44.
[65] Weiderpass E, Pukkala E, Vasama-Neuvonen K, et al. Occupational exposures and cancers of the endometrium and cervix uteri in Finland. Am J Ind Med 2001;39:572–80.
[66] Betenia N, Costello S, Eisen EA. Risk of cervical cancer among female autoworkers exposed to metalworking fluids. Scand J Work Environ Health 2012;38:78–83.
[67] Camargo MC, Stayner LT, Straif K, et al. Occupational exposure to asbestos and ovarian cancer: a meta-analysis. Environ Health Perspect 2011;119:1211–7.
[68] Wernli KJ, Ray RM, Gao DL, et al. Occupational exposures and ovarian cancer in textile workers. Epidemiology 2008;19:244–50.
[69] Guo J, Kauppinen T, Kyyronen P, Heikkila P, Lindbohm ML, Pukkala E. Risk of esophageal, ovarian, testicular, kidney and bladder cancers and leukemia among Finnish workers exposed to diesel or gasoline engine exhaust. Int J Cancer 2004;111:286–92.
[70] Morgan RW, Kelsh MA, Zhao K, Heringer S. Mortality of aerospace workers exposed to trichloroethylene. Epidemiology 1998;9:424–31.
[71] McGlynn KA, Trabert B. Adolescent and adult risk factors for testicular cancer. Nat Rev Urol 2012;9:339–49.
[72] Skakkebaek NE, Rajpert-De Meyts E, Main KM. Testicular dysgenesis syndrome: an increasingly common developmental disorder with environmental aspects. Hum Reprod 2001;16:972–8.
[73] Pukkala E, Martinsen JI, Lynge E, et al. Occupation and cancer - follow-up of 15 million people in five Nordic countries. Acta Oncol 2009;48:646–790.
[74] Wilson RT, Donahue M, Gridley G, Adami J, El Ghormli L, Dosemeci M. Shared occupational risks for transitional cell cancer of the bladder and renal pelvis among men and women in Sweden. Am J Ind Med 2008;51:83–99.
[75] Jensen OM, Knudsen JB, McLaughlin JK, Sorensen BL. The Copenhagen case-control study of renal pelvis and ureter cancer: role of smoking and occupational exposures. Int J Cancer 1988;41:557–61.
[76] Vajdic CM, Kricker A, Giblin M, et al. Sun exposure predicts risk of ocular melanoma in Australia. Int J Cancer 2002;101:175–82.
[77] Hakansson N, Floderus B, Gustavsson P, Feychting M, Hallin N. Occupational sunlight exposure and cancer incidence among Swedish construction workers. Epidemiology 2001;12:552–7.
[78] Guenel P, Laforest L, Cyr D, et al. Occupational risk factors, ultraviolet radiation, and ocular melanoma: a case-control study in France. Cancer Causes Control 2001;12:451–9.

[79] Hoppin JA, Tolbert PE, Herrick RF, et al. Occupational chlorophenol exposure and soft tissue sarcoma risk among men aged 30-60 years. Am J Epidemiol 1998;148:693–703.
[80] Fingerhut MA, Halperin WE, Marlow DA, et al. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. N Engl J Med 1991;324:212–8.
[81] Collins JJ, Bodner K, Aylward LL, Wilken M, Bodnar CM. Mortality rates among trichlorophenol workers with exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. Am J Epidemiol 2009;170:501–6.
[82] Cardis E, Vrijheid M, Blettner M, et al. The 15-Country Collaborative Study of Cancer Risk among Radiation Workers in the Nuclear Industry: estimates of radiation-related cancer risks. Radiat Res 2007;167:396–416.
[83] Merletti F, Richiardi L, Bertoni F, et al. Occupational factors and risk of adult bone sarcomas: a multicentric case-control study in Europe. Int J Cancer 2006;118:721–7.
[84] Baan R, Grosse Y, Lauby-Secretan B, et al. Carcinogenicity of radiofrequency electromagnetic fields. Lancet Oncol 2011;12:624–6.
[85] Speers MA, Dobbins JG, Miller VS. Occupational exposures and brain cancer mortality: a preliminary study of east Texas residents. Am J Ind Med 1988;13:629–38.
[86] Navas-Acien A, Pollan M, Gustavsson P, Floderus B, Plato N, Dosemeci M. Interactive effect of chemical substances and occupational electromagnetic field exposure on the risk of gliomas and meningiomas in Swedish men. Cancer Epidemiol Biomarkers Prev 2002;11:1678–83.
[87] Baldi I, Coureau G, Jaffre A, et al. Occupational and residential exposure to electromagnetic fields and risk of brain tumors in adults: a case-control study in Gironde, France. Int J Cancer 2011;129:1477–84.
[88] Coble JB, Dosemeci M, Stewart PA, et al. Occupational exposure to magnetic fields and the risk of brain tumors. Neuro Oncol 2009;11:242–9.
[89] Musicco M, Sant M, Molinari S, Filippini G, Gatta G, Berrino F. A case-control study of brain gliomas and occupational exposure to chemical carcinogens: the risk to farmers. Am J Epidemiol 1988;128:778–85.
[90] Rodvall Y, Ahlbom A, Spannare B, Nise G. Glioma and occupational exposure in Sweden, a case-control study. Occup Environ Med 1996;53:526–32.
[91] Samanic CM, De Roos AJ, Stewart PA, Rajaraman P, Waters MA, Inskip PD. Occupational exposure to pesticides and risk of adult brain tumors. Am J Epidemiol 2008;167:976–85.
[92] Yiin JH, Ruder AM, Stewart PA, et al. The Upper Midwest Health Study: a case-control study of pesticide applicators and risk of glioma. Environ Health 2012;11:39.
[93] Provost D, Cantagrel A, Lebailly P, et al. Brain tumours and exposure to pesticides: a case-control study in southwestern France. Occup Environ Med 2007;64:509–14.
[94] Neta G, Stewart PA, Rajaraman P, et al. Occupational exposure to chlorinated solvents and risks of glioma and meningioma in adults. Occup Environ Med 2012;69:793–801.
[95] Rajaraman P, Stewart PA, Samet JM, et al. Lead, genetic susceptibility, and risk of adult brain tumors. Cancer Epidemiol Biomarkers Prev 2006;15:2514–20.
[96] Navas-Acien A, Pollan M, Gustavsson P, Plato N. Occupation, exposure to chemicals and risk of gliomas and meningiomas in Sweden. Am J Ind Med 2002;42:214–27.
[97] Cocco P, Heineman EF, Dosemeci M. Occupational risk factors for cancer of the central nervous system (CNS) among US women. Am J Ind Med 1999;36:70–4.
[98] Zielinski JM, Garner MJ, Band PR, et al. Health outcomes of low-dose ionizing radiation exposure among medical workers: a cohort study of the Canadian national dose registry of radiation workers. Int J Occup Med Environ Health 2009;22:149–56.
[99] Lope V, Perez-Gomez B, Aragones N, et al. Occupational exposure to ionizing radiation and electromagnetic fields in relation to the risk of thyroid cancer in Sweden. Scand J Work Environ Health 2006;32:276–84.
[100] Zabel EW, Alexander BH, Mongin SJ, et al. Thyroid cancer and employment as a radiologic technologist. Int J Cancer 2006;119:1940–5.
[101] Lope V, Perez-Gomez B, Aragones N, et al. Occupational exposure to chemicals and risk of thyroid cancer in Sweden. Int Arch Occup Environ Health 2009;82:267–74.
[102] Orsi L, Delabre L, Monnereau A, et al. Occupational exposure to pesticides and lymphoid neoplasms among men: results of a French case-control study. Occup Environ Med 2009;66:291–8.
[103] Khuder SA, Mutgi AB, Schaub EA, Tano BD. Meta-analysis of Hodgkin's disease among farmers. Scand J Work Environ Health 1999;25:436–41.
[104] Karunanayake CP, Spinelli JJ, McLaughlin JR, Dosman JA, Pahwa P, McDuffie HH. Hodgkin lymphoma and pesticides exposure in men: a Canadian case-control study. J Agromedicine 2012;17:30–9.
[105] Briggs NC, Levine RS, Hall HI, Cosby O, Brann EA, Hennekens CH. Occupational risk factors for selected cancers among African American and White men in the United States. Am J Public Health 2003;93:1748–52.
[106] McCunney RJ. Hodgkin's disease, work, and the environment. A review. J Occup Environ Med 1999;41:36–46.
[107] Vlaanderen J, Lan Q, Kromhout H, Rothman N, Vermeulen R. Occupational benzene exposure and the risk of lymphoma subtypes: a meta-analysis of cohort studies incorporating three study quality dimensions. Environ Health Perspect 2011;119:159–67.
[108] Kogevinas M, Zock JP, Alvaro T, et al. Occupational exposure to immunologically active agents and risk for lymphoma. Cancer Epidemiol Biomarkers Prev 2004;13:1814–8.
[109] Wang H, Murat Y, Nomura S, et al. A meta-analysis of epidemiological studies on the relationship between occupational electromagnetic field exposure and the risk of adult leukemia. Environ Health Prev Med 2000;5:43–6.
[110] Morales-Suarez-Varela MM, Olsen J, Johansen P, et al. Occupational risk factors for mycosis fungoides: a European multicenter case-control study. J Occup Environ Med 2004;46:205–11.
[111] Morales-Suarez-Varela MM, Olsen J, Johansen P, et al. Occupational exposures and mycosis fungoides. A European multicentre case-control study (Europe). Cancer Causes Control 2005;16:1253–9.
[112] Morales-Suarez-Varela MM, Olsen J, Johansen P, et al. Occupational sun exposure and mycosis fungoides: a European multicenter case-control study. J Occup Environ Med 2006;48:390–3.
[113] Cocco P, t'Mannetje A, Fadda D, et al. Occupational exposure to solvents and risk of lymphoma subtypes: results from the Epilymph case-control study. Occup Environ Med 2010;67:341–7.
[114] Kane EV, Newton R. Benzene and the risk of non-Hodgkin lymphoma: a review and meta-analysis of the literature. Cancer Epidemiol 2010;34:7–12.
[115] Seidler A, Mohner M, Berger J, et al. Solvent exposure and malignant lymphoma: a population-based case-control study in Germany. J Occup Med Toxicol 2007;2:2.
[116] Wong O, Harris F, Armstrong TW, Hua F. A hospital-based case-control study of non-Hodgkin lymphoid neoplasms in Shanghai: analysis of environmental and occupational risk factors by subtypes of the WHO classification. Chem Biol Interact 2010;184:129–46.
[117] Wang R, Zhang Y, Lan Q, et al. Occupational exposure to solvents and risk of non-Hodgkin lymphoma in Connecticut women. Am J Epidemiol 2009;169:176–85.
[118] Kato I, Koenig KL, Watanabe-Meserve H, et al. Personal and occupational exposure to organic solvents and risk of non-Hodgkin's lymphoma (NHL) in women (United States). Cancer Causes Control 2005;16:1215–24.
[119] Purdue MP, Bakke B, Stewart P, et al. A case-control study of occupational exposure to trichloroethylene and non-Hodgkin lymphoma. Environ Health Perspect 2011;119:232–8.

[120] Miligi L, Costantini AS, Benvenuti A, et al. Occupational exposure to solvents and the risk of lymphomas. Epidemiology 2006;17:552–61.
[121] Fritschi L, Benke G, Hughes AM, et al. Occupational exposure to pesticides and risk of non-Hodgkin's lymphoma. Am J Epidemiol 2005;162:849–57.
[122] Clavel J, Hemon D, Mandereau L, Delemotte B, Severin F, Flandrin G. Farming, pesticide use and hairy-cell leukemia. Scand J Work Environ Health 1996;22:285–93.
[123] Nordstrom M, Hardell L, Magnuson A, Hagberg H, Rask-Andersen A. Occupational exposures, animal exposure and smoking as risk factors for hairy cell leukaemia evaluated in a case-control study. Br J Cancer 1998;77:2048–52.
[124] Vlaanderen J, Lan Q, Kromhout H, Rothman N, Vermeulen R. Occupational benzene exposure and the risk of chronic myeloid leukemia: a meta-analysis of cohort studies incorporating study quality dimensions. Am J Ind Med 2012;55:779–85.
[125] Infante PF. Benzene exposure and multiple myeloma: a detailed meta-analysis of benzene cohort studies. Ann N Y Acad Sci 2006;1076:90–109.
[126] Baan R, Grosse Y, Straif K, et al. A review of human carcinogens–Part F: chemical agents and related occupations. Lancet Oncol 2009;10:1143–4.
[127] Sonoda T, Nagata Y, Mori M, Ishida T, Imai K. Meta-analysis of multiple myeloma and benzene exposure. J Epidemiol 2001;11:249–54.
[128] Lope V, Perez-Gomez B, Aragones N, et al. Occupation, exposure to chemicals, sensitizing agents, and risk of multiple myeloma in Sweden. Cancer Epidemiol Biomarkers Prev 2008;17:3123–7.
[129] Perrotta C, Staines A, Cocco P. Multiple myeloma and farming. A systematic review of 30 years of research. Where next? J Occup Med Toxicol 2008;3:27.
[130] Pahwa P, Karunanayake CP, Dosman JA, Spinelli JJ, McDuffie HH, McLaughlin JR. Multiple myeloma and exposure to pesticides: a Canadian case-control study. J Agromedicine 2012;17:40–50.
[131] Gold LS, Stewart PA, Milliken K, et al. The relationship between multiple myeloma and occupational exposure to six chlorinated solvents. Occup Environ Med 2011;68:391–9.
[132] Zhang L, Steinmaus C, Eastmond DA, Xin XK, Smith MT. Formaldehyde exposure and leukemia: a new meta-analysis and potential mechanisms. Mutat Res 2009;681:150–68.
[133] Gluzman DF, Sklyarenko LM, Nadgornaya VA, Zavelevich MP. Mature B-cell neoplasms in Chernobyl clean-up workers of 1986-1987: summary of cytomorphological and immunocytochemical study in 25 years after Chernobyl accident. Exp Oncol 2011;33:47–51.
[134] Khalade A, Jaakkola MS, Pukkala E, Jaakkola JJ. Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis. Environ Health 2010;9:31.
[135] Schnatter AR, Glass DC, Tang G, Irons RD, Rushton L. Myelodysplastic syndrome and benzene exposure among petroleum workers: an international pooled analysis. J Natl Cancer Inst 2012;104:1724–37.
[136] Kiran S, Cocco P, Mannetje A, et al. Occupational exposure to ethylene oxide and risk of lymphoma. Epidemiology 2010;21:905–10.
[137] Tatham L, Tolbert P, Kjeldsberg C. Occupational risk factors for subgroups of non-Hodgkin's lymphoma. Epidemiology 1997;8:551–8.
[138] Sielken Jr RL, Valdez-Flores C. Butadiene cancer exposure-response modeling: based on workers in the styrene-butadiene-rubber industry: total leukemia, acute myelogenous leukemia, chronic lymphocytic leukemia, and chronic myelogenous leukemia. Regul Toxicol Pharmacol 2011;60:332–41.
[139] Nanni O, Amadori D, Lugaresi C, et al. Chronic lymphocytic leukaemias and non-Hodgkin's lymphomas by histological type in farming-animal breeding workers: a population case-control study based on a priori exposure matrices. Occup Environ Med 1996;53:652–7.
[140] Cocco P, Satta G, Dubois S, et al. Lymphoma risk and occupational exposure to pesticides: results of the Epilymph study. Occup Environ Med 2013;70:91–8.
[141] Schubauer-Berigan MK, Daniels RD, Fleming DA, et al. Chronic lymphocytic leukaemia and radiation: findings among workers at five US nuclear facilities and a review of the recent literature. Br J Haematol 2007;139:799–808.
[142] Kesminiene A, Evrard AS, Ivanov VK, et al. Risk of hematological malignancies among Chernobyl liquidators. Radiat Res 2008;170:721–35.
[143] Vrijheid M, Cardis E, Ashmore P, et al. Ionizing radiation and risk of chronic lymphocytic leukemia in the 15-country study of nuclear industry workers. Radiat Res 2008;170:661–5.
[144] Dolcetti R, Serraino D, Dognini G, et al. Exposure to animals and increased risk of marginal zone B-cell lymphomas of the ocular adnexae. Br J Cancer 2012;106:966–9.
[145] Daniels RD, Bertke S, Waters KM, Schubauer-Berigan MK. Risk of leukaemia mortality from exposure to ionising radiation in US nuclear workers: a pooled case-control study. Occup Environ Med 2013;70:41–8.
[146] Van Maele-Fabry G, Duhayon S, Lison D. A systematic review of myeloid leukemias and occupational pesticide exposure. Cancer Causes Control 2007;18:457–78.
[147] Boffetta P. Risk of acute myeloid leukemia after exposure to diesel exhaust: a review of the epidemiologic evidence. J Occup Environ Med 2004;46:1076–83.
[148] Lamm SH, Engel A, Joshi KP, Byrd 3rd DM, Chen R. Chronic myelogenous leukemia and benzene exposure: a systematic review and meta-analysis of the case-control literature. Chem Biol Interact 2009;182:93–7.
[149] Mehlman MA. Dangerous and cancer-causing properties of products and chemicals in the oil refining and petrochemical industries. Part XXX: Causal relationship between chronic myelogenous leukemia and benzene-containing solvents. Ann N Y Acad Sci 2006;1076:110–9.
[150] Delzell E, Sathiakumar N, Graff J, Macaluso M, Maldonado G, Matthews R. An updated study of mortality among North American synthetic rubber industry workers. Res Rep Health Eff Inst 2006;132:1–63, discussion 65-74.
[151] Kokouva M, Bitsolas N, Hadjigeorgiou GM, Rachiotis G, Papadoulis N, Hadjichristodoulou C. Pesticide exposure and lymphohaematopoietic cancers: a case-control study in an agricultural region (Larissa, Thessaly, Greece). BMC Public Health 2011;11:5.
[152] West RR, Stafford DA, Farrow A, Jacobs A. Occupational and environmental exposures and myelodysplasia: a case-control study. Leuk Res 1995;19:127–39.
[153] van Steensel-Moll HA, Valkenburg HA, van Zanen GE. Childhood leukemia and parental occupation. A register-based case-control study. Am J Epidemiol 1985;121:216–24.
[154] Buckley JD, Robison LL, Swotinsky R, et al. Occupational exposures of parents of children with acute nonlymphocytic leukemia: a report from the Childrens Cancer Study Group. Cancer Res 1989;49:4030–7.
[155] Shu XO, Stewart P, Wen WQ, et al. Parental occupational exposure to hydrocarbons and risk of acute lymphocytic leukemia in offspring. Cancer Epidemiol Biomarkers Prev 1999;8:783–91.
[156] Shu XO, Perentesis JP, Wen W, et al. Parental exposure to medications and hydrocarbons and ras mutations in children with acute lymphoblastic leukemia: a report from the Children's Oncology Group. Cancer Epidemiol Biomarkers Prev 2004;13:1230–5.
[157] Schuz J, Kaletsch U, Meinert R, Kaatsch P, Michaelis J. Risk of childhood leukemia and parental self-reported occupational exposure to chemicals, dusts, and fumes: results from pooled analyses of German population-based case-control studies. Cancer Epidemiol Biomarkers Prev 2000;9:835–8.
[158] Lowengart RA, Peters JM, Cicioni C, et al. Childhood leukemia and parents' occupational and home exposures. J Natl Cancer Inst 1987;79:39–46.
[159] McKinney PA, Raji OY, van Tongeren M, Feltbower RG. The UK Childhood Cancer Study: maternal occupational exposures

and childhood leukaemia and lymphoma. Radiat Prot Dosimetry 2008;132:232–40.
[160] Feychting M, Plato N, Nise G, Ahlbom A. Paternal occupational exposures and childhood cancer. Environ Health Perspect 2001;109:193–6.
[161] Reid A, Glass DC, Bailey HD, et al. Risk of childhood acute lymphoblastic leukaemia following parental occupational exposure to extremely low frequency electromagnetic fields. Br J Cancer 2011;105:1409–13.
[162] Van Maele-Fabry G, Lantin AC, Hoet P, Lison D. Childhood leukaemia and parental occupational exposure to pesticides: a systematic review and meta-analysis. Cancer Causes Control 2010;21:787–809.
[163] Wigle DT, Turner MC, Krewski D. A systematic review and meta-analysis of childhood leukemia and parental occupational pesticide exposure. Environ Health Perspect 2009;117:1505–13.
[164] Vinson F, Merhi M, Baldi I, Raynal H, Gamet-Payrastre L. Exposure to pesticides and risk of childhood cancer: a meta-analysis of recent epidemiological studies. Occup Environ Med 2011;68:694–702.
[165] Castro-Jimenez MA, Orozco-Vargas LC. Parental exposure to carcinogens and risk for childhood acute lymphoblastic leukemia, Colombia, 2000-2005. Prev Chronic Dis 2011;8:A106.
[166] Sung TI, Wang JD, Chen PC. Increased risk of cancer in the offspring of female electronics workers. Reprod Toxicol 2008;25:115–9.
[167] Infante-Rivard C, Siemiatycki J, Lakhani R, Nadon L. Maternal exposure to occupational solvents and childhood leukemia. Environ Health Perspect 2005;113:787–92.
[168] Smulevich VB, Solionova LG, Belyakova SV. Parental occupation and other factors and cancer risk in children: II. Occupational factors. Int J Cancer 1999;83:718–22.
[169] Colt JS, Blair A. Parental occupational exposures and risk of childhood cancer. Environ Health Perspect 1998;106(Suppl 3):909–25.
[170] Meinert R, Kaletsch U, Kaatsch P, Schuz J, Michaelis J. Associations between childhood cancer and ionizing radiation: results of a population-based case-control study in Germany. Cancer Epidemiol Biomarkers Prev 1999;8:793–9.
[171] Johnson KJ, Alexander BH, Doody MM, et al. Childhood cancer in the offspring born in 1921-1984 to US radiologic technologists. Br J Cancer 2008;99:545–50.
[172] Hug K, Grize L, Seidler A, Kaatsch P, Schuz J. Parental occupational exposure to extremely low frequency magnetic fields and childhood cancer: a German case-control study. Am J Epidemiol 2010;171:27–35.
[173] Feychting M, Floderus B, Ahlbom A. Parental occupational exposure to magnetic fields and childhood cancer (Sweden). Cancer Causes Control 2000;11:151–6.
[174] Pearce MS, Hammal DM, Dorak MT, McNally RJ, Parker L. Paternal occupational exposure to electro-magnetic fields as a risk factor for cancer in children and young adults: a case-control study from the North of England. Pediatr Blood Cancer 2007;49:280–6.
[175] Kristensen P, Andersen A, Irgens LM, Bye AS, Sundheim L. Cancer in offspring of parents engaged in agricultural activities in Norway: incidence and risk factors in the farm environment. Int J Cancer 1996;65:39–50.
[176] Shim YK, Mlynarek SP, van Wijngaarden E. Parental exposure to pesticides and childhood brain cancer: U.S. Atlantic coast childhood brain cancer study. Environ Health Perspect 2009;117:1002–6.
[177] van Wijngaarden E, Stewart PA, Olshan AF, Savitz DA, Bunin GR. Parental occupational exposure to pesticides and childhood brain cancer. Am J Epidemiol 2003;157:989–97.
[178] Johnson CC, Annegers JF, Frankowski RF, Spitz MR, Buffler PA. Childhood nervous system tumors—an evaluation of the association with paternal occupational exposure to hydrocarbons. Am J Epidemiol 1987;126:605–13.
[179] Cordier S, Monfort C, Filippini G, et al. Parental exposure to polycyclic aromatic hydrocarbons and the risk of childhood brain tumors: The SEARCH International Childhood Brain Tumor Study. Am J Epidemiol 2004;159:1109–16.
[180] Peters S, Glass DC, Reid A, et al. Parental occupational exposure to engine exhausts and childhood brain tumors. Int J Cancer 2012;2012:27972.
[181] Cordier S, Mandereau L, Preston-Martin S, et al. Parental occupations and childhood brain tumors: results of an international case-control study. Cancer Causes Control 2001;12:865–74.
[182] McKean-Cowdin R, Preston-Martin S, Pogoda JM, Holly EA, Mueller BA, Davis RL. Parental occupation and childhood brain tumors: astroglial and primitive neuroectodermal tumors. J Occup Environ Med 1998;40:332–40.
[183] Savitz DA, Chen JH. Parental occupation and childhood cancer: review of epidemiologic studies. Environ Health Perspect 1990;88:325–37.
[184] De Roos AJ, Olshan AF, Teschke K, et al. Parental occupational exposures to chemicals and incidence of neuroblastoma in offspring. Am J Epidemiol 2001;154:106–14.
[185] Kerr MA, Nasca PC, Mundt KA, Michalek AM, Baptiste MS, Mahoney MC. Parental occupational exposures and risk of neuroblastoma: a case-control study (United States). Cancer Causes Control 2000;11:635–43.
[186] Valery PC, Williams G, Sleigh AC, Holly EA, Kreiger N, Bain C. Parental occupation and Ewing's sarcoma: pooled and meta-analysis. Int J Cancer 2005;115:799–806.
[187] Moore LE, Gold L, Stewart PA, Gridley G, Prince JR, Zahm SH. Parental occupational exposures and Ewing's sarcoma. Int J Cancer 2005;114:472–8.
[188] Fear NT, Vincent TJ, King JC, MacCarthy A, Bunch KJ, Murphy MF. Wilms tumour and paternal occupation: an analysis of data from the National Registry of Childhood Tumours. Pediatr Blood Cancer 2009;53:28–32.
[189] MacCarthy A, Bunch KJ, Fear NT, King JC, Vincent TJ, Murphy MF. Paternal occupation and neuroblastoma: a case-control study based on cancer registry data for Great Britain 1962–1999. Br J Cancer 2010;102:615–9.
[190] Kopec JA, Esdaile JM. Bias in case-control studies. A review. J Epidemiol Community Health 1990;44:179–86.
[191] Bhatti P, Stewart PA, Linet MS, Blair A, Inskip PD, Rajaraman P. Comparison of occupational exposure assessment methods in a case-control study of lead, genetic susceptibility and risk of adult brain tumours. Occup Environ Med 2011;68:4–9.
[192] Tschoep K, Kohlmann A, Schlemmer M, Haferlach T, Issels RD. Gene expression profiling in sarcomas. Crit Rev Oncol Hematol 2007;63:111–24.
[193] Clarke CA, Undurraga DM, Harasty PJ, Glaser SL, Morton LM, Holly EA. Changes in cancer registry coding for lymphoma subtypes: reliability over time and relevance for surveillance and study. Cancer Epidemiol Biomarkers Prev 2006;15:630–8.
[194] Morton LM, Turner JJ, Cerhan JR, et al. Proposed classification of lymphoid neoplasms for epidemiologic research from the Pathology Working Group of the International Lymphoma Epidemiology Consortium (InterLymph). Blood 2007;110:695–708.
[195] Hung RJ, Boffetta P, Brennan P, et al. GST, NAT, SULT1A1, CYP1B1 genetic polymorphisms, interactions with environmental exposures and bladder cancer risk in a high-risk population. Int J Cancer 2004;110:598–604.
[196] Cogliano VJ, Baan R, Straif K, et al. Preventable exposures associated with human cancers. J Natl Cancer Inst 2011;103:1827–39.
[197] Kane AB. Animal models of malignant mesothelioma. Inhal Toxicol 2006;18:1001–4.
[198] Goldstein BD. Benzene as a cause of lymphoproliferative disorders. Chem Biol Interact 2010;184:147–50.
[199] Zhang L, Eastmond DA, Smith MT. The nature of chromosomal aberrations detected in humans exposed to benzene. Crit Rev Toxicol 2002;32:1–42.

[200] Rushton L, Hutchings S, Brown T. The burden of cancer at work: estimation as the first step to prevention. Occup Environ Med 2008;65:789–800.
[201] Garcia Gomez M, Urbaneja Arrue F, Markowitz S, Castaneda Lopez R, Menduina PL. Occupational diseases compensated in the Basque Country (Spain) from 1990 to 2008. Am J Ind Med 2013;56:326–34.
[202] Cellier C, Charbotel B, Carretier J, et al. Identification of occupational exposures among patients with lung cancer. Bull Cancer 2013;100:661–70.
[203] Marinaccio A, Scarselli A, Merler E, Iavicoli S. Mesothelioma incidence surveillance systems and claims for workers' compensation. Epidemiological evidence and prospects for an integrated framework. BMC Public Health 2012;12:314.
[204] Binazzi A, Scarselli A, Marinaccio A. The burden of mortality with costs in productivity loss from occupational cancer in Italy. Am J Ind Med 2013;2013:22224.
[205] European-Commission. Commission Recommendation 2003/670/EC of 19 September 2003 concerning the European schedule of occupational diseases: Official Journal L 238. 25/09/2003; 2003:28-34.

Biographies

Barbara Charbotel, MD, PhD, is an occupational physician specialized in occupational diseases and epidemiology. She previously published several papers on occupational cancers and participated to the IARC monograph 106 on chlorinated solvents.

Béatrice Fervers, MD, PhD, associate professor, is a medical oncologist and holds a PhD in public health. She is the head of the Cancer and Environment Unit of the Léon Bérard Cancer Centre (Lyon, France) and has previously coordinated a national program in evidence-based knowledge translation in oncology.

Jean-Pierre Droz, MD, PhD, emeritus Professor of Medical Oncology at the Lyon-Est Medical School, Lyon University and former Chairman of the Department of Medical Oncology of the Centre Léon Bérard. His clinical research focuses on germ-cell tumors and prostate cancer, new drug development and geriatric oncology. The three authors have contributed substantially to the conception and design of this review, acquisition and interpretation of data, drafting the article or revising it critically for important intellectual content, and to the final approval of the version submitted.