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Occupational exposures and haematological malignancies: overview on human recent data

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Abstract

Objective: Occupational causes of haematological malignancies are relatively uncommon, under-studied and under-identified. They are also often unrecognized by clinicians. This review summarizes the principal epidemiologic studies on this topic.
Methods: We analyzed the recent relevant human data found in the Medline, the Pascal and the BDSP databases.
Results: Benzene and ionizing radiation are the only agents conclusively demonstrated to be carcinogenic to the haematopoietic system. In particular, both exposures are strongly associated with acute myeloid leukaemia. Low doses of both may also be related to myeloid malignancies. Infectious agents and pesticides are also thought to induce lymphoproliferative cancers. Some studies show an association between haematological malignancies and low-frequency electromagnetic fields and organic solvents. All of these suspected occupational causes must be confirmed by further studies.
Conclusions: Better knowledge and understanding of occupational causes of haematological malignancies are necessary to improve their prevention and compensation.

Introduction

Historically, one of the earliest examples of a haematological malignancy thought to have an occupational cause is the blood cancer that probably killed Marie Curie: despite some diagnostic confusion, her cause of death was most likely due to occupational exposure to ionizing radiation exposure [1]. Although the known occupational causes of malignant blood diseases were once relatively rare, chemical and physical environmental hazards are thought to explain the increasing incidence of these diseases, especially lymphomas, over the past three decades [2]. Moreover, the risk of leukaemia attributable to occupational factors has been estimated at 10% in the USA [3, 4] and 5–10% in Europe [3, 4]: occupational causes would thus account for 111–224 incident cases of leukaemia per year in France, although only 20 leukaemia patients received disability compensation for an occupational disease in the year 2000. These figures indicate the extent to which the occupational causes of these diseases are under-diagnosed, in part because physicians often do not recognize their occupational factors.

Furthermore, many occupational causes of these diseases are simply unidentified. Benzene and ionizing radiation are the only agents conclusively proven to be carcinogenic to the haematopoietic system, although other occupational exposures are suspected of involvement in these diseases [1, 5, 6].

The purpose of this paper was to review the available epidemiologic data about occupational sources of haematological malignancies.

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Material and methods

We searched for relevant publications from 1992 through 2004 in three databases: the Medline database, the Pascal database (developed by the French national centre for scientific research, CNRS) and the BDSP database (developed by the French national school of public health, ENSP). We used the keywords leukaemia, lymphoma, Hodgkin's disease, multiple myeloma, and myelodysplastic syndromes, together with occupational, work, and occupational disease. To be exhaustive, we also checked cohort studies on major occupational exposures that have been linked to haematological malignancies. For this search, we used the following keywords 'occupational' AND 'cancer' AND ('benzene' or 'petrochemical' or 'gasoline' or 'nuclear industry' or 'ionizing radiation' or 'pesticide' or 'farmer' or 'butcher' or 'meatpacker' or 'slaughterhouse' or 'abattoir' or 'virus' or 'microbe' or 'organic solvent' or 'beautician' or 'hair dye' or 'diesel exhaust' or 'embalmer' or 'anatomist' or 'pathologist' or 'formaldehyde' or 'health worker' or 'antineoplastic'). We looked only at published data for humans and rejected studies with imprecise descriptions of exposure or diagnosis. Toxicological studies, particularly studies of biochemical pathways, were not included. Case reports were also excluded, but well-documented reviews were included, in part to ensure that our review took into account studies published before 1992.

We analyzed 153 papers: 71 cohort studies, 46 case–control studies, and 36 meta-analyses or critical reviews. They came from journals in the fields of occupational health (65%), epidemiology (12%), haematology and oncology (14%), and other fields (toxicology or general, for the remaining 9%).

We report the results according to strength of evidence and agent. Tables 1–10 summarize the principal results of selected studies according to disease.

Results

Demonstrated occupational causes

A causal relation with leukaemia, especially acute myeloid leukaemia (AML), has been demonstrated according to the Bradford Hill criteria (strength of association, consistency, specificity, temporality, dose-response relationship, coherence and analogy) for only two agents: benzene and ionizing radiation [5–7].

Ionizing radiation

Ionizing radiation has been classified as a certain carcinogenic hazard (group 1) by the International Agency for Research on Cancer (IARC) [5].

AML. The causal relation between ionizing radiation and haematological malignancies was first demonstrated during the follow-up of the Nagasaki and Hiroshima atomic-bomb survivors by their high incidence of AML [6, 7]. In the workplace, a retrospective cohort study of nuclear workers found an elevated mortality rate from leukaemia (Table 1), significant only for high exposure (cumulative dose higher than 200 mSv) and considering all haematological malignancies, with a rate ratio estimated at 15.65 (95% CI: 3.33–73.50) [8].

Low doses of ionizing radiation and leukaemia. The United Nations Scientific Committee on Effects of Atomic Radiations (UNSCEAR) reported in 2000 that levels of occupational radiation exposure today are approximately half of what they were in earlier decades. The average annual effective dose in the 1990s has been estimated at 1–5 mSv [7]. Some studies of nuclear and medical workers have examined in more detail the relation between exposure to low doses of ionizing radiation (<5 mSv/year) and leukaemia [8–13]. Pooling mortality data from seven cohort studies covering 96,673 nuclear workers, the IARC study group found a significant excess relative risk of leukaemia [excluding chronic lymphoid leukaemia (CLL)] associated with exposure to ionizing radiation (Table 1) [9]. The mean duration of exposure was 36 years and 80% of the study population had a cumulative exposure less than 50 mSv.

Other types of leukaemia and myelodysplastic syndromes. A cohort study of workers who participated in the United Kingdom's atmospheric weapons tests and experimental program found them to have a significantly increased relative risk of leukaemia (CLL excluded) [14]. The UNSCEAR report in 2000 also noted associations between exposure to ionizing radiation and leukaemia, except for T-cell leukaemia and CLL [7]. A case–control study showed an association between occupational exposure to ionizing radiation and myelodysplastic syndromes (Table 4) [15].

Malignant B-cell lymphoproliferative disorders. No epidemiological data support a causal relation between occupational exposure to ionizing radiation and non-Hodgkin lymphoma (NHL) or Hodgkin disease (Tables 6 and 7) [7, 16]. A case–control study showed an excess risk of multiple myeloma (MM) among older nuclear facility workers exposed to low levels of ionizing radiation: the odds ratio reached 5.15 in the group with thehighest exposure at the age of 45 and older (cumulative dose of 100 mSv or more) [17]. A cohort study

Table 1. Epidemiologic surveys of occupational leukaemia, all types, not specified

References	Study design	Exposure or job	Risk estimation	CI 95%
Guenel 2002 [23]	Cohort	Benzene	OR = 1.2[a,b]	1.0–1.5
Seniori 2003 [27]	Cohort	Benzene (>200 ppm years)	SMR = 5.1[a,b]	1.4–13.0
Rinsky 2002 [26]	Cohort	Benzene (between 40 and 200 ppm years)	SMR = 3.21[b]	0.86–8.89
Rinsky 2002 [26]	Cohort	Benzene (between 200 and 400 ppm years)	SMR = 5.55[b]	0.62–24.08
Rinsky 2002 [26]	Cohort	Benzene (>400 ppm years)	SMR = 23.96[a,b]	4.82–78.51
Wong 1999 [33]	Nested case–control	Gasoline (3–5% benzene)	SMR = 0.80	0.56–1.07
Huebner 2000 [52]	Cohort	Petrochemical industry	SIR = 1.29	0.75–2.07
Wong 2001 [28]	Cohort	Petrochemical industry	SMR = 1.37	0.96–1.88
Sathiakumar 1995 [30]	Case–control	Petrochemical industry	OR = 2.0[b]	0.97–4.2
Gun 2004 [144]	Cohort	Petrochemical industry	SIR = 1.39	0.91–2.02
Hunting 1995 [25]	Cohort	Vehicle mechanics (benzene exposure)	SMR = 9.26[a]	1.12–33.43
Wong 1999 [33]	Nested case–control	Gasoline (3–5% benzene)	SMR = 0.80	0.56–1.07
Blair 1998 [46]	Cohort	Trichloroethylene	RR = 0.6	0.3–1.2
Gustavsson 1999 [145]	Cohort	Chemical industry	SIR = 2.24	0.46–6.54
Steenland 2004 [118]	Cohort	Ethylene oxide	SMR = 0.99	0.71–1.36
Coggon 2004 [117]	Cohort	Ethylene oxide (chemical and hospital)	SMR = 1.08	0.35–2.51
Eisen 2001 [146]	Cohort	Automobile industry (metalworking fluids)	SMR = 1.34[a]	1.14–1.58
Divine 2001 [107]	Cohort	Butadiene	SMR = 1.29	0.77–2.04
Santos-Burgoa 1992 [111]	Case–control	Butadiene	OR = 9.36[a]	2.05–22.9
Santos-Burgoa 1992 [111]	Case–control	Styrene	OR = 3.13	0.84–11.2
Hauptmann 2003 [125]	Cohort	Formaldehyde (peak levels >4 ppm)	RR = 3.46[a,c]	1.27–9.43
Coggon 2003 [127]	Cohort	Formaldehyde	SMR = 0.91	0.62–1.29
IARC 1994 [9]	Meta-analysis	Ionizing radiation (low doses)	Meta ERR = 2.2 (by Sv)[a]	0.1–5.7[f]
Ritz 1999 [8]	Cohort	Ionizing radiation	RR = 1.60	0.95–2.52
Sont 2001 [16]	Cohort	Ionizing radiation	ERR = 5.4 (by Sv)[a]	0.2–20.0[f]
Muirhead 2003 [14]	Cohort	Ionizing radiation	RR = 1.83[a,d]	1.15–2.93[f]
Kheifets 1999 [61]	Meta-analysis	Electromagnetic fields	RR = 1.09 (by 10 μT years)	0.98–1.21
Harrington 2001 [62]	Cohort	Electromagnetic fields	SMR = 0.84	0.69–1.01
Hakansson 2002 [63]	Cohort	Electromagnetic fields (0.25–0.53 μT), men	RR = 0.80	0.5–1.3
Hakansson 2002 [63]	Cohort	Electromagnetic fields (0.25–0.53 μT), women	RR = 2.0	0.8–4.6
Morgan 2000 [64]	Cohort	Radiofrequency exposure >5 Years	RR = 1.05	0.46–2.07
Savitz 2000 [147]	Nested case–control	Electromagnetic fields between 4.75–12.2 μT/Year	RR = 1.44	0.53–3.91
Baker 1999 [80]	Meta-analysis	Teachers	RR = 1.77[a,e]	1.22–2.47
Blair 2001 [74]	Case–control	Health workers	OR = 1.8	0.9–3.6
Bertazzi 2001 [101]	Cohort	Dioxins (TCDD)	RR = 3.8[a]	1.2–12.5
Fleming 1999 [148]	Cohort	Agriculture	OR = 1.29	0.78–2.02
Blair 2001 [74]	Case–control	Agriculture >10 years exposure	OR = 2.1[a]	1.0–4.5

[a] Statistically significant with p-level <0.05.
[b] Dose-effect (or time-effect) relation.
[c] RR related to myeloid leukaemia.
[d] RR related to leukaemia without chronic lymphoid leukaemia.
ppm = parts per million; μT = microtesla; ERR = Excess relative risk (significant when ERR do not include 0); RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; CI95% = Confidence interval at 95%.
[e] Confidence interval was at 90%.

[18] also showed an association between MM mortality and combined exposure to ionizing radiation and chemicals, but the number of deaths (n = 2) from this disease was too low to justify a definitive conclusion (Table 10). The UNSCEAR report considered the available data and concluded in 2000 that no association has yet been demonstrated between ionizing radiation and MM [7].

Benzene

Occupational benzene exposure has also been demonstrated to cause haematological malignancies [19].

AML. There is strong evidence that high daily exposure to benzene (more than 10 ppm) is associated with AML, and both dose-response and time-response relations have been demonstrated (Table 2) [19]. For example,

Table 2. Epidemiologic surveys of occupational acute myeloid leukaemia

References	Study design	Exposure or job	Risk estimation	CI 95%
Wong 1995 [20]	Cohort	Benzene	SMR = 5.03[a,b]	1.81–10.97
Wong 1995 [20]	Cohort	Benzene <200 ppm years	SMR = 0.91	0.02–5.11
Wong 1995 [20]	Cohort	Benzene 200–400 ppm years[c]	SMR = 27.21[a,b]	3.29–98.24
Wong 1995 [20]	Cohort	Benzene >400 ppm years[c]	SMR = 98.37[a,b]	20.28–287.65
Hayes 1997 [21]	Cohort	Benzene <10 ppm	RR = 3.2[a,b]	1.0–10.3
Hayes 1997 [21]	Cohort	Benzene <25 ppm	RR = 7.1[a,b]	2.1–23.7
Bloemen 2004 [149]	Cohort	Benzene	SMR = 1.1	0.30–2.83
Jakobsson 1993 [22]	Mortality	Gasoline (3–5% benzene)	SMR = 3.60[a]	1.70–6.60
Huebner 1997 [36]	Cohort	Petrochemical industry	SMR = 1.00	0.56–1.65
Wong 1999 [33]	Nested case–control	Gasoline (3–5% benzene)	SMR = 1.17	0.69–1.85
Sathiakumar 1995 [30]	Case–control	Petrochemical industry	OR = 2.8[a]	1.1–7.3
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 1.29	0.78–1.99
Brown 2002 [57]	Cohort	Male workers in the paint and varnish industry	SIR = 2.2[a]	1.0–4.2
Pinkerton 2004 [126]	Cohort	Formaldehyde	SMR = 1.34	0.61–2.54
Massoudi 1997 [55]	Case–control	Chemical industry	OR = 1.09	0.40–2.96
Albin 2000 [39]	Case–control	Organic solvent (except benzene)	OR = 2.7[a,b]	1.0–7.3
Lazarov 2000 [37]	Case–control	Organic solvent	OR = 2.52[a]	1.45–4.39
Hunter 1993 [38]	Mortality	Chemical laboratory	SMR = 1.92[a]	1.12–3.08
Kheifets 1997 [59]	Meta-Analysis	Electromagnetic fields	Meta-RR = 1.40[a]	1.16–1.69
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 2.25	0.79–6.46
Willett 2003 [65]	Case–control	Electromagnetic fields	OR = 0.91	0.69–1.18
Mele 1994 [72]	Case–control	Child-care workers and teachers	OR = 3.3[a]	1.2–9.0
Bethwaite 2001 [71]	Case–control	Butchers	OR = 2.6[a,b]	1.1–6.5
Cocco 1997 [78]	Mortality	Organochlorine (DDT)	PMR = 1.89	0.38–5.52

[a] Statistically significant with p-level < 0.05.
[b] Dose-effect (or time-effect) relation
ppm = parts per million; Meta-RR = Meta Risk ratio; RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Table 3. Epidemiologic surveys of suspected occupational chronic myeloid leukaemia

References	Study design	Exposure or job	Risk estimation	CI 95%
Wong 2001 [28]	Cohort	Petrochemical industry	SMR = 1.31	0.43–3.07
Huebner 1997 [36]	Cohort	Petrochemical industry	SMR = 1.02	0.44–2.00
Guenel 2002 [23]	Cohort	Benzene (high exposure group)	OR = 1.2	0.1–11.4
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 1.05	0.54–1.83
Lewis 2000 [150]	Cohort	Petrochemical industry	SMR = 3.51[a]	1.68–6.45
Pinkerton 2004 [126]	Cohort	Formaldehyde	SMR = 1.39	0.38–3.56
Kheifets 1997 [59]	Meta-Analysis	Electromagnetic fields	Meta-RR = 1.24	0.98–1.57
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 0.61	0.18–2.05
Mele 1994 [72]	Case–control	Child-care workers and teachers	OR = 7.8[a]	2.3–26.3

[a] Statistically significant with p-level < 0.05.
Meta-RR = Meta Risk ratio; SMR = Standardized mortality ratio; OR = Odds ratio; CI95% = Confidence interval at 95%.

two large petrochemical industry cohort studies showed an association between cumulative benzene exposure and mortality from AML [20, 21].

Low doses of benzene and leukaemia. Although the association between high benzene exposure and leukaemia is now well documented, long periods of

Table 4. Epidemiologic surveys of suspected occupational myelodysplastic syndromes

References	Study design	Exposure or job	Risk estimation	CI 95%
West 1995 [15]	Case–control	Ionizing radiation	OR = 2.05[a]	1.16–2.52
West 1995 [15]	Case–control	Halogenated organics	OR = 1.57	0.97–2.57
Rigolin 1998 [40]	Case–control	Organic solvent	OR = 7.11[a]	2.42–20.88
Rigolin 1998 [40]	Case–control	Pesticides	OR = 2.12[a]	1.26–3.59

[a] Statistically significant with p-level < 0.05.
OR = Odds ratio; CI95% = Confidence interval at 95%.

Table 5. Epidemiologic surveys of suspected occupational hairy-cell leukaemia

References	Study design	Exposure or job	Risk estimation	CI 95%
Clavel 1996 [106]	Case–control	Pesticides	OR = 1.7[a]	1.0–2.6
Clavel 1996 [106]	Case–control	Organophosphorus insecticides	OR = 7.6	0.9–61.6
Clavel 1995 [105]	Case–control	Agriculture employment (men)	OR = 1.7[a]	1.1–2.4
Clavel 1995 [105]	Case–control	Agriculture employment (women)	OR = 2.7[a]	1.1–6.7
Nordstrom 1998 [104]	Case–control	Fungicides	OR = 3.8[a]	1.4–9.9
Nordstrom 1998 [104]	Case–control	Herbicides	OR = 2.9[a]	1.4–5.9
Nordstrom 1998 [104]	Case–control	Insecticides	OR = 2.0[a]	1.1–3.5
Nordstrom 1998 [104]	Case–control	Solvents	OR = 1.5	0.99–2.3

[a] Statistically significant with p-level < 0.05.
OR = Odds ratio; CI95% = Confidence interval at 95%.

low-dose exposure have also been related to leukaemia [22–25]: a cohort study of almost 75,000 workers suggests that low benzene exposure (average levels below 10 ppm) is associated with acute non-lymphocytic leukaemia (and related myelodysplastic syndromes) (Table 2) [21]. Results from a case–control study nested in a cohort of gas and electrical workers confirm an association between low benzene exposure and leukaemia (Table 1) [23]. However, no distinction of cell type was made, and some studies do not support this relation (Tables 1 and 2) [20, 26, 27].

Other types of leukaemia. Cohort studies of workers in the petroleum, gas and electricity industries have not shown any significant excess risk of other types of leukaemia, and in particular of chronic myeloid leukaemia (Table 3) [23, 28, 29]. Epidemiologic studies of occupational diseases, however, frequently combine acute and chronic myeloid leukaemia, and this failure to differentiate them may account in part for the absence of evidence [30].

Malignant B-cell lymphoproliferative disorders. Although limited data suggest that benzene exposure may be associated with NHL (Table 6), there is not enough evidence today to support a causal association [19, 31, 32]. Similarly, there is no evidence to support a causal association (Table 10) between occupational benzene exposure and MM [20, 26, 29, 33, 34].

Suspected occupational causes

Aromatic hydrocarbons and organic solvents

Myeloid leukaemia and myelodysplastic syndromes. Aromatic hydrocarbons such as xylene and toluene, which are used as substitutes for benzene, are suspected to be related to the onset of AML (Table 2). An increased risk of death from AML was observed in different cohorts of petrochemical workers exposed to benzene and other aromatic hydrocarbons [29, 30]. A case–control study also found an association between self-reported exposure to toluene and acute leukaemia, with a dose-response relation [35]. Nevertheless, a cohort study of 80,000 petrochemical workers exposed to hydrocarbons found no increased risk of AML [36].

Several studies report an association between exposure to organic solvents, such as aromatic hydrocarbons, and myeloid leukaemia, principally AML (Tables 2 and 3). A case–control study of 98 cases found a positive association between organic solvent exposure and AML [37], and a mortality study of professional chemists observed a significant excess of deaths from this cause [38]. Only one study, however, considered solvents independently of benzene exposure (Table 2) [39].

Several case–control studies have shown a positive association between organic solvent exposure and myelodysplastic syndromes (Table 4) [15, 40, 41], but no specific agents were identified.

Table 6. Epidemiologic surveys of occupational non-Hodgkin lymphoma

References	Study design	Exposure or job	Risk estimation	CI 95%
Lagorio 1994 [151]	Cohort	Benzene (3–5%)	SMR = 1.58	0.43–4.08
Hayes 1997 [21]	Cohort	Benzene >10 years	RR = 4.2[a]	1.1–15.9
Wong 1999 [33]	Nested case–control	Benzene (3–5%)	SMR = 0.42	0.25–0.65
Fabbro-Peray 2001 [31]	Case–control	Benzene >810 days	OR = 4.6[a]	1.1–19.2
Collingwood 1996 [152]	Cohort	Petrochemical industry	SMR = 1.32	0.74–2.17
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 0.88	0.69–1.11
Huebner 2000 [52]	Cohort	Petrochemical industry	SIR = 1.06	0.67–1.61
Sathiakumar 1998 [108]	Cohort	Butadiene and styrene	SMR = 0.91	0.61–1.32
Divine 2001 [107]	Cohort	Butadiene	SMR = 1.48	0.89–2.31
Blair 1998 [46]	Cohort	Trichloroethylene	RR = 2	0.9–4.6
Axelson 1994 [50]	Cohort	Trichloroethylene	SIR = 1.56	0.51–3.64
Hansen 2001 [51]	Cohort	Trichloroethylene	SIR = 3.5[a]	1.5–6.9
Raaschou-Nielsen 2003 [49]	Cohort	Trichloroethylene (high exposure)	SIR = 1.5[a]	1.2–2.0
Boice 1999 [56]	Cohort	Trichloroethylene	SMR = 1.19	0.65–1.99
Anttila 1995 [47]	Cohort	Halogenated hydrocarbons	SIR = 2.13[a]	1.06–3.80
Rafnsson 2001 [153]	Cohort	Typesetters	SIR = 4.46[a]	1.63–9.70
Hansen 1994 [132]	Cohort	Pharmacy dispenser (long term)	SIR = 3.7[a]	1.2–8.9
Massoudi 1997 [55]	Case–control	Chemical industry	OR = 3.11[a]	1.10–8.82
Hardell 1994 [91]	Case–control	Solvents	OR = 2.4[a]	1.4–3.9
Blair 1993 [42]	Case–control	Typesetters >10 years	OR = 2.5[a,b]	1.1–5.7
Mao 2000 [154]	Case–control	Benzidine	OR = 1.9[a]	1.1–3.4
Mao 2000 [154]	Case–control	Lubricating oils	OR = 1.3[a]	1.0–1.5
Rego 2002 [45]	Case–control	Organic solvents	OR = 1.67	0.97–2.87
Rego 2002 [45]	Case–control	Organic solvents + domestic pesticides	OR = 2.24[a]	1.01–3.97
Tatham 1997 [44]	Case–control	Solvents	OR = 1.60[a,c]	1.10–2.20
Demers 1998 [155]	Meta-Analysis	Wood-workers	SMR = 1.08	0.81–1.39
Sont 2001 [16]	Cohort	Ionizing radiation	Risk excess (by Sv) = 6.6	0.0–28.3[d]
Schroeder 1997 [68]	Cohort	Electromagnetic fields >20 years	RR = 1.40	0.80–2.30
Morgan 2000 [64]	Cohort	Radiofrequency exposure >5 years	RR = 0.64	0.32–1.15
Cano 2001 [66]	Cohort	Communication and transport (except pilots and postmen) (Electromagnetic field)	RR = 2.43 to 3.43[a]	$p < 0.05$
Villeneuve 2000 [60]	Nested case–control	Electromagnetic fields >40 V/m	OR = 3.57[a,b]	1.30–9.80
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 1.22	0.77–1.94
Fabbro-Peray 2001 [31]	Case–control	Radio operator	OR = 3.1[a]	1.4–6.6
Baker 1999 [80]	Meta-Analysis	Teachers	RR = 1.36[a]	1.13–1.62
Blair 1992 [73]	Meta-Analysis	Agriculture	Meta-RR = 1.05	0.98–1.12
Khuder 1998 [83]	Meta-Analysis	Agriculture	Meta-OR = 1.10[a]	1.03–1.19
Baris 1998 [89]	Meta-Analysis	DDT exposure (pesticides)	OR = 1.2[a]	1.0–1.6
Gambini 1997 [96]	Cohort	Rice growers >20 years of exposure	SMR = 3.38	0.92–8.65
Burns 2001 [156]	Cohort	2.4-D (herbicide)	SMR = 1.00	0.21–2.92
Kelleher 1998 [77]	Cohort	Farmers	SIR = 1.69[a]	1.24–2.66
Thorn 2000 [92]	Cohort	Herbicides (phenoxyacetic acids)	SIR = 1.92[b]	0.03–10.7
Zahm 1997 [93]	Cohort	Pesticides	SMR = 1.14[b]	0.31–2.91
Zahm 1997 [93]	Cohort	Pesticides >3 years	SMR = 7.11[a]	1.78–28.42
Cano 2001 [66]	Cohort	Agriculture	RR = 0.96	0.88–1.04
MacLennan 2003 [157]	Cohort	Triazine herbicides	SMR = 3.72[a]	1.01–9.52
Rusiecki 2004 [100]	Cohort	Triazine herbicides	RR = 1.61	0.62–4.16
Acquavella 2004 [98]	Cohort	Alachlor herbicides, high exposure	SIR = 2.07	0.43–6.04
Bertazzi 2001 [101]	Cohort	Dioxins (TCDD)	RR = 2.8[a]	1.1–7.0
Metayer 1998 [88]	Nested case–control	Abattoir workers (viruses)	OR = 12[a]	1.1–130.6
Amadori 1995 [79]	Case–control	Breeders (agriculture)	OR = 2.22[a]	1.16–4.26
Hardell 1994 [91]	Case–control	Herbicides (phenoxyacetic acids)	OR = 5.5[a]	2.7–11
Hardell 1994 [91]	Case–control	Chlorophenols	OR = 4.8[a]	2.7–8.8
Fabbro-Peray 2001 [31]	Case–control	Agriculture	OR = 1.5[a]	1.0–2.1
Mao 2000 [154]	Case–control	Herbicides and pesticides	OR = 1.3[a]	1.0–1.6
De Roos 2003 [95]	Case–control	Triazine and Alachlor herbicides	OR = 2.1[a]	1.1–3.9

Table 6. (Continued)

References	Study design	Exposure or job	Risk estimation	CI 95%
De Roos 2003 [95]	Case–control	Triazine herbicides and Diazinon insecticides	OR = 3.9[a]	1.7–8.8
Kato 2004 [97]	Case–control	Pesticides 10–18 years (women)	OR = 2.72[a]	1.37–5.40
Zheng 2001 [90]	Case–control	Carbamate (herbicides and insecticides)	OR = 1.5[a]	1.1–2.0
Garabedian 1999 [158]	Case–control	Chlorophenol >8 years	OR = 1.51	0.88–2.59
Fritschi 2002 [82]	Case–control	Workers with animals	OR = 1.8[a]	1.1–2.9
Lee 2002 [76]	Mortality	Breeders	PMR = 1.17[a]	1.06–1.30
Cerhan 1998 [85]	Mortality	Agriculture	PMR = 1.09	0.96–1.23

[a] Statistically significant with p-level < 0.05.
[b] Dose-effect (or time-effect) relation.
[c] OR related to small cell diffuse lymphoma.
[d] Confidence interval at 90%.
μT = microtesla; Meta-RR = Meta risk ratio; Meta-OR = Meta odds ratio; RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Table 7. Epidemiologic surveys of suspected occupational Hodgkin disease

References	Study design	Exposure or job	Risk estimation	CI 95%
Wong 2001 [28]	Cohort	Petrochemical industry	SMR = 0.61	0.13–1.77
Wong 1999 [33]	Nested case–control	Benzene (3–5%)	SMR = 0.48	0.18–1.05
Sathiakumar 1998 [108]	Cohort	Butadiene and styrene	SMR = 0.95	0.41–1.87
Blair 1998 [46]	Cohort	Trichloroethylene	RR = 1.4	0.2–12
Axelson 1994 [50]	Cohort	Trichloroethylene	SIR = 1.07	0.03–5.95
Hunter 1993 [38]	Mortality	Chemistry	SMR = 0.51[a]	0.218–0.996
Sont 2001 [16]	Cohort	Ionizing radiation	Risk excess (by Sv) = 64.8	0.0–591.3[b]
Morgan 2000 [64]	Cohort	Radiofrequency exposure >5 years	RR = 1.14	0.31–3.10
Schroeder 1997 [68]	Cohort	Electromagnetic fields >20 years	RR = 1.1	0.3–4.4
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 1.33	0.65–2.70
Khuder 1999 [84]	Meta-Analysis	Agriculture	RR = 1.25[a]	1.11–1.42
Blair 1992 [73]	Meta-Analysis	Agriculture	Meta-RR = 1.16[a]	1.03–1.29
Pukkala 1997 [159]	Cohort	Farmers (without animals)	SIR = 1.74[a]	1.12–2.59
Swaen 1992 [102]	Cohort	Pesticides	SMR = 3.34	0.04–18.61
Bertazzi 2001 [101]	Cohort	Dioxins (TCDD)	RR = 4.9[a]	1.5–16.4
Metayer 1998 [88]	Nested case–control	Abattoir workers (viruses)	OR = 12[a]	1.1–130.6
Pahwa 2003 [160]	Case–control	Farmers	OR = 0.95	0.61–1.48
Cerhan 1998 [85]	Mortality	Agriculture	PMR = 1.62[a]	1.04–2.54
Khuder 1998 [83]	Meta-Analysis	Agriculture	RR = 1.25[a]	1.11–1.42
Blair 1992 [73]	Meta-Analysis	Agriculture	Meta-RR = 1.16[a]	1.03–1.29
Pukkala 1997 [159]	Cohort	Farmers (without animals)	SIR = 1.74[a]	1.12–2.59
Swaen 1992 [102]	Cohort	Pesticides	SMR = 3.34	0.04–18.61
Bertazzi 2001 [101]	Cohort	Dioxins (TCDD)	RR = 4.9[a]	1.5–16.4
Metayer 1998 [88]	Nested case–control	Abattoir workers (viruses)	OR = 12[a]	1.1–130.6
Pahwa 2003 [160]	Case–control	Farmers	OR = 0.95	0.61–1.48
Cerhan 1998 [85]	Mortality	Agriculture	PMR = 1.62[a]	1.04–2.54

[a] Statistically significant with p-level < 0.05.
[b] Confidence interval at 90%.
Meta-RR = Meta risk ratio; Meta-OR = Meta odds ratio; RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Malignant B-cell lymphoproliferative disorders. An association is also suspected between organic solvent exposure and NHL [42–44]. One study in particular [45] suggests that occupational organic solvent exposure is associated with an excess risk of NHL, especially among workers using household insecticides. Several cohort studies found an association between exposure to trichloroethylene (Table 6), a solvent used especially in the aviation sector, and NHL [46–51].

Table 8. Epidemiologic surveys of suspected occupational acute lymphoid leukaemia

References	Study design	Exposure or job	Risk estimation	CI 95%
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 1.01	0.32–2.35
Kheifets 1997 [59]	Meta-Analysis	Electromagnetic fields	Meta-RR = 1.33	0.93–1.92
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 2.07	0.12–35.25
Blair 2001 [74]	Case–control	Electromagnetic fields	OR = 2.4	0.7–8.8
Willett 2003 [65]	Case–control	Electromagnetic fields	OR = 1.70	0.86–3.35
Kelleher 1998 [77]	Cohort	Agriculture	SIR = 1.87[a]	1.13–2.92
Bethwaite 2001 [71]	Case–control	Butchers	OR = 4.0[a]	1.0–16.1
Mele 1994 [72]	Case–control	Agriculture	OR = 1.0	0.4–2.5

[a] Statistically significant with p-level < 0.05.
[b] Dose-effect (or time-effect) relation.
[c] Confidence interval was at 90% in this study.
Meta-RR = Meta risk ratio; Meta-OR = Meta odds ratio; RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Table 9. Epidemiologic surveys of suspected occupational chronic lymphoid leukaemia

References	Study design	Exposure or job	Risk estimation	CI 95%
Huebner 1997 [36]	Cohort	Petrochemical industry	SMR = 1.60	0.85–2.73
Huebner 2000 [52]	Cohort	Petrochemical industry	SIR = 1.22	0.40–2.85
Lewis 2000 [150]	Cohort	Petrochemical industry	SMR = 3.51[a]	1.68–6.45
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 0.8	0.45–1.33
Hunter 1993 [38]	Mortality	Chemical laboratory	SMR = 1.79[a]	1.00–2.95
Kheifets 1997 [59]	Meta-Analysis	Electromagnetic Fields	Meta-RR = 1.55[a]	1.10–2.19
Theriault 1994 [67]	Case–control	Electromagnetic Fields	OR = 1.40	0.52–3.77
Kelleher 1998 [77]	Cohort	Agriculture	SIR = 1.88[a]	1.34–2.56
Amadori 1995 [79]	Case–control	Farmers-breeders	OR = 3.05[a]	1.12–8.32
Lee 2002 [76]	Mortality	Farmers-breeders	PMR = 1.28[a]	1.06–1.53

[a] Statistically significant with p-level < 0.05.
Meta-RR = Meta risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Exposure to other aromatic hydrocarbons in petrochemical plants [29, 52] or in occupations such as painting, printing or aircraft manufacture [53–57] has been suspected of inducing MM, but a meta-analysis of 22 cohorts did not support this association (Table 10) [58].

Electromagnetic fields

Leukaemia. A meta-analysis suggests that occupational exposure to electromagnetic fields, including low frequency and extremely low frequency fields, may be associated with AML (small but significant increase in risk) and CLL in electrical industry workers [59]. A case–control study nested in a cohort of electric utility workers explored the relation between a series of indices of electric and magnetic field exposure and the incidence of leukaemia; its aim was to identify the most appropriate exposure indicator for risk assessment. A significant association was found between exposure to electric fields above 10 V/m and leukaemia [60], but these results have not been confirmed by recent studies in the industry (Tables 1 and 2) [61–65].

Malignant B-cell lymphoproliferative disorders. Some data from the electricity and telecommunications industries suggest a relation between electromagnetic field exposure and NHL or MM (Tables 6 and 10) [66–69]. A slight positive association between electromagnetic field exposure for a duration up to 20 years and low-grade NHL was found in a cohort of electric utility workers [68]. No association was observed between electromagnetic fields and MM in this study. A cohort study of workers in the engineering industry did, however, suggest an association between high levels of extremely low frequency magnetic fields and MM [63].

Table 10. Epidemiologic surveys of suspected occupational multiple myeloma

References	Study design	Exposure or job	Risk estimation	CI 95%
Sonoda 2001 [130]	Meta-Analysis	Benzene/solvents	Meta-OR = 0.74[a]	0.6–0.9
Sonoda 2001 [130]	Meta-Analysis	Petroleum	Meta-OR = 1.11	0.96–1.28
Sonoda 2001 [130]	Meta-Analysis	Engine exhaust	Meta-OR = 1.34[a]	1.14–1.57
Lee 2003 [129]	Cohort	Engine exhaust (diesel)	RR = 1.3[a]	1.00–1.77
Wong 1995 [20]	Cohort	Benzene	SMR = 2.91	0.79–7.45
Rinsky 2002 [26]	Cohort	Benzene	SMR = 2.04	0.66–4.76
Wong 1999 [33]	Nested case–control	Benzene 3–5%	SMR = 0.79	0.46–1.24
Wong 1997 [58]	Meta-Analysis	Petrochemical industry	SMR = 0.93	0.81–1.07
Divine 1999 [29]	Cohort	Petrochemical industry	SMR = 1.01	0.7–1.4
Huebner 2000 [52]	Cohort	Petrochemical industry	SIR = 1.39	0.64–2.64
Blair 1998 [46]	Cohort	Trichloroethylene	RR = 1.3	0.5–3.4
Axelson 1994 [50]	Cohort	Trichloroethylene	SIR = 0.57	0.01–3.17
Anttila 1995 [47]	Cohort	Trichloromethane or tetrachloroethane	SIR = 15.98[a]	1.93–57.7
Lundberg 1998 [161]	Cohort	Organic solvents > 5 years	SIR = 3.8	0.8–11
Sathiakumar 1998 [108]	Cohort	Butadiene and styrene	SMR = 0.92	0.5–1.55
Brown 2002 [57]	Cohort	Male painters	SIR = 1.0	0.8–1.2
Demers 1993 [54]	Case–control	Painters > 10 years	OR = 4.1[a, b]	1.8–10.4
Massoudi 1997 [55]	Case–control	Chemical industry	OR = 2.39[a]	1.04–5.48
Baysson 2000 [18]	Cohort	Ionizing radiation and chemicals	SMR = 8.38[a]	1.44–26.2
Schroeder 1997 [68]	Cohort	Electromagnetic fields > 20 years	RR = 0.9	0.4–1.8
Hakansson 2002 [63]	Cohort	Electromagnetic fields 0.164–0.25 μT (medium exposure)	RR = 2.9[b]	0.8–10.7
Hakansson 2002 [63]	Cohort	Electromagnetic fields 0.25–0.53 μT (high exposure)	RR = 3.8	0.9–15.6
Theriault 1994 [67]	Case–control	Electromagnetic fields	OR = 1.06	0.44–2.53
Baker 1999 [80]	Meta-Analysis	Teachers	Meta-PMR = 1.58[a]	1.47–1.70
Robinson 1999 [162]	Mortality	Teachers	PMR = 1.23[a]	1.06–1.43
Blair 1992 [73]	Meta-Analysis	Agriculture	Meta-RR = 1.12[a]	1.04–1.21
Pukkala 1997 [159]	Cohort	Agriculture (men only)	SIR = 0.95	0.82–1.10
Swaen 1992 [102]	Cohort	Pesticides	SMR = 8.15[a]	1.64–23.82
Baris 2004 [163]	Cohort	Pesticides	OR = 1.3	0.9–1.8
Lee 2004 [99]	Cohort	Alachlor herbicides	RR = 5.66	0.70–45.7
Metayer 1998 [88]	Nested case–control	Supermarket meat-cutters	OR = 18[a]	1.6–207.95
Nanni 1998 [103]	Case–control	Organochlorine	OR = 2.4[a]	1.0–5.9
Demers 1993 [54]	Case–control	Pesticides (high exposure)	OR = 5.2[a, b]	1.96–21.1
Cocco 1997 [78]	Mortality	DDT (pesticides)	SMR = 3.41[a]	1.10–7.95
Cerhan 1998 [85]	Mortality	Agriculture	PMR = 1.17	0.98–1.40
Lee 2002 [76]	Mortality	Breeders	PMR = 1.19[a]	1.03–1.38

[a] Statistically significant with p-level < 0.05.

[b] Dose-effect (or time-effect) relation.

μT = microtesla Meta-RR = Meta risk ratio; Meta-OR = Meta odds ratio; Meta-PMR = Meta proportional mortality ratio; RR = Risk ratio; SMR = Standardized mortality ratio; SIR = Standardized incidence ratio; OR = Odds ratio; PMR = proportional mortality ratio; CI95% = Confidence interval at 95%.

Based in part on studies of haematopoietic cancer, IARC considers electromagnetic fields to be a possible carcinogenic hazard (group 2B). Further studies are needed [70].

Infectious agents

Myeloid malignancies. Some case–control studies suggest that myeloid diseases, especially AML, may be related to exposure to infectious agents among occupational groups as diverse as butchers [71] and teachers [72]. These studies did not, however, take into account confounding factors, such as exposure to chemicals in the meat industry [71].

Malignant B-cell lymphoproliferative disorders. Among agricultural workers, occupational exposure to infectious agents [73–78] is suspected of increasing the risk of lymphoproliferative malignancies (Tables 6–9). A case–control study among meat industry workers showed a significant association and a time-effect

relation between exposure to infectious agents in abattoirs and leukaemia (especially acute lymphoid leukaemia) [71]. A population-based case–control study found a significant association between work as a farmer-breeder and CLL [79]. Although some animal retroviruses, such as bovine leukaemia virus (BLV) and bovine immunodeficiency virus, are suspected, no specific infectious agents have been identified.

Some studies support a causal association between occupational exposure to viruses and NHL, especially among teachers [80] and breeders (Table 6) [31, 76, 81]. Two case–control studies found a positive association between working with animals and NHL [79, 82]. These results are consistent with the results of a meta-analysis of NHL risk among farmers [83]. Several viruses are suspected: Epstein Barr (EBV) and human T-cell leukaemia for human transmission and BLV for animal contacts [82, 83].

Exposure to infectious agents is also thought to increase the risk of Hodgkin disease [73, 84, 85] (Table 7), mainly in the teaching and medical sectors, where its incidence is higher than in the general population [86, 87]. Nevertheless, no specific agent except EBV has been clearly related to occupational Hodgkin disease [84].

Infectious agents, especially viruses, are also suspected of causing MM mortality: two studies found elevated mortality from MM among teachers (Table 10) [80]. A nested case–control study in a cohort of meat industry workers also showed an increased risk of MM among male supermarket cutters [88].

Pesticides

Malignant B-cell lymphoproliferative disorders. Several studies report a consistent, significant, and positive association (Table 6) between occupational pesticide exposure and NHL [89–100]. The classes of pesticides involved have not been clearly identified. A pooled data meta-analysis examined organochlorine exposure but found no strong consistent evidence for its association with NHL [89]. A population-based case–control study suggests an increased risk of NHL associated with carbamate pesticides (insecticides and herbicides) [90]. Several different case–control and cohort studies [91–93] suggest that phenoxyacetic acid exposure may be linked to NHL (Table 6). Others, however, suggest that these results are due to the contamination of phenoxyacetic acid herbicides by dioxins (TCDD) [83], which are also possible haematopoietic carcinogens, as indicated by data from the Seveso cohort [101]. A multicenter case–control study found several other pesticides, including insecticide oils and triazine, to be significantly associated with NHL (combined with CLL) [94].

Excess risks of CLL (Table 9) and of MM (Table 10) have been found in various studies among farmers [54, 73, 102], and pesticide exposure has been suggested as the cause. Except for chlorinated pesticides [78, 103], no specific agents were identified [76, 77, 79].

Three case–control studies also point to an association between occupational pesticide exposure and hairy cell leukaemia (Table 5) [104–106].

Myeloid malignancies. An excess risk of myelodysplastic syndromes was only associated with pesticide exposure in a case–control study (Table 4) [40].

Other chemical agents

1,3-butadiene. 1,3-butadiene, mostly used in the rubber and plastic industries, is considered a probable carcinogen by IARC (group 2A). Several studies show it to be positively associated with haematological malignancies, especially leukaemia [107–111]. However, a study of the haematological data of employees from a petrochemical facility possibly exposed to 1,3-butadiene monomer found a mortality rate from all lymphohaematopoietic cancers approximately the same as in the reference population (SMR, 1.06; 95% CI, 0.22–3.11) [112].

Styrene. Styrene, also used in the plastic industry, is considered a possible carcinogenic agent by IARC (group 2B), and its metabolite styrene oxide a probable carcinogen (group 2A). A historical cohort study left open a possible excess risk of leukaemia in styrene-exposed workers [113], but no excess risk of mortality from leukaemia was observed in a cohort study of 5204 such workers [114].

Ethylene oxide. Ethylene oxide is used as a sterilizing agent or intermediary in chemical synthesis and has been classified by IARC as certainly carcinogenic to the haematopoietic system (group 1) [115–117]. An extended mortality follow-up of a cohort of 18,235 men and women exposed to ethylene oxide found no overall excess of haematopoietic cancer or any specific type of haematopoietic cancer (Table 1), but a significant trend was seen in the exposure-response (haematopoietic cancer) relation among men [118]. An other extended mortality follow up of workers exposed to ethylene oxide also concluded that balance of evidence from epidemiologic data indicates that haematopoietic cancer from ethylene expose are low [117].

Alkylating antineoplastic drugs. In recent decades, conclusive evidence has demonstrated that high dose of alkylating antineoplastic drugs can cause AML in patients with cancer. These drugs are thus suspectedof increasing the risk of leukaemia among healthcare workers [119, 120], and occupational studies have indeed found an excess risk of leukaemia in this occupational category [119, 121–123]. These data are based on relatively few cases, however, and need to be confirmed by larger studies.

Formaldehyde. Exposure to formaldehyde has reportedly been associated with leukaemia, especially in three broad occupational groups: embalmers, anatomists, and formaldehyde industrial workers [124]. Two large cohort studies on industrial workers have recently found a significant association between formaldehyde exposure and increased leukaemia rates [125, 126]. However, a recent large cohort study with higher exposure to formaldehyde and a larger number of workers than the two others failed to confirm this conclusion [127]. The lack of consistency of the data across epidemiology studies [127, 128] and the absence of biological plausibility lead us to conclude that there is no demonstrated evidence of a causal relation between formaldehyde exposure and leukaemia [124].

Miscellaneous. Causal relations have been also proposed but not demonstrated between exposure to engine exhaust and MM [129, 130], wood exposure and Hodgkin disease [86], hair dye exposure and lymphoproliferative cancers [131], and biomedical laboratory products and haematological malignancies [132–137].

Discussion and conclusion

Causal relations are now well-documented between haematopoietic cancers, especially AML, and high levels of exposure to benzene and to ionizing radiation. Infectious agents and pesticides are strongly suspected of inducing lymphoproliferative cancers. Some studies also show associations between haematological malignancies and exposure to low levels of benzene, ionizing radiation, organic solvents, cytostatic drugs, and ethylene oxide.

Despite the many studies of occupational risk factors, haematological malignancies due to occupational exposure have not been adequately studied in epidemiologic situations. Their relative rarity, histological diversity, long latency periods, and confounding factors, including genetic factors, all help explain this lack of study. Moreover, precise exposure assessment, both qualitative and quantitative, is too often absent. Specifically, too little information is collected about the types of pesticides, solvents and infectious agents [138–140]. Nevertheless, epidemiologic studies have provided hypotheses about several occupational hazards. Their role in the development of haematological malignancies must be documented by other research, including molecular analysis and toxicological experiments to explain mechanisms and pathways [39, 141].

Similarly, at the individual level, clinicians find the occupational associations of their patients' diseases obscured by long latency periods, insufficient information about occupational history, and other factors. Aid from specialists in occupational health and in toxicology is essential to enable clinicians to make the difficult linkage between occupation and disease. Improved recognition of the occupational source of haematological malignancies is needed. Workers' compensation is available when these diseases are considered to be related to benzene or ionizing radiation, for instance, in France [5], Germany [142], and Italy [143]. Better identification of these occupational haematological malignancies by clinicians might also improve their prevention.

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