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Full length article

The effect of exposure to radiofrequency fields on cancer risk in the general and working population: A systematic review of human observational studies – Part II: Less researched outcomes

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A R T I C L E I N F O

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Cohort studies
Case-control studies
Systematic review

A B S T R A C T

Background: In the framework of the World Health Organization assessment of health effects of exposure to radiofrequency electromagnetic fields (RF-EMF), we have conducted a systematic review of human observational studies on the association between exposure to RF-EMF and risk of neoplastic diseases. Due to the extremely large number of included exposure types/settings and neoplasm combinations, we decided to present the review findings in two separate papers. In the first one we addressed the most investigated exposure-outcome pairs (e.g. glioma, meningioma, acoustic neuroma in relation to mobile phone use, or risk childhood leukemia in relation to environmental exposure from fixed-site transmitters) (Karipidis et al., 2024). Here, we report on less researched neoplasms, which include lymphohematopoietic system tumours, thyroid cancer and oral cavity/pharynx cancer, in relation to wireless phone use, or occupational RF exposure.

Methods: Eligibility criteria: We included cohort and case-control studies of neoplasia risks in relation to three types of exposure to RF-EMF: 1. exposure from wireless phone use; 2. environmental exposure from fixed-site transmitters; 3. occupational exposures. In the current paper, we focus on less researched neoplasms including leukaemia, non-Hodgkin's lymphoma and thyroid cancer in mobile phone users; lymphohematopoietic system tumours and oral cavity/pharynx cancer in exposed workers. We focussed on investigations of specific neoplasms in relation to specific exposure sources (termed exposure-outcome pair, abbreviated E-O pairs), noting that a single article may address multiple E-O pairs.

Information sources: Eligible studies were identified by predefined literature searches through Medline, Embase, and EMF-Portal.

Risk-of-bias (RoB) assessment: We used a tailored version of the Office of Health Assessment and Translation (OHAT) RoB tool to evaluate each study's internal validity. Then, the studies were classified into three tiers according to their overall potential for bias (low, moderate and high) in selected, predefined and relevant bias domains.

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Data synthesis: We synthesized the study results using random effects restricted maximum likelihood (REML) models.
Evidence assessment: Confidence in evidence was assessed according to the Grading of Recommendations, Assessment, Development and Evaluations (GRADE) approach.
Results: We included 26 articles, which were published between 1988 and 2019, with participants from 10 countries, reporting on 143 different E-O pairs, including 65 different types of neoplasms. Of these, 19 E-O pairs satisfied the criteria for inclusion in quantitative syntheses of the evidence regarding the risks of leukaemia, non-Hodgkin's lymphoma or thyroid cancer in relation to mobile phone use, and the risks of lymphohematopoietic system tumours or oral cavity/pharynx cancer following occupational exposure to RF-EMF.
RF-EMF exposure from mobile phones (ever or regular use *vs* no or non-regular use) was not associated with an increased risk of leukaemia [*meta*-estimate of the relative risk (mRR) = 0.99, 95 % CI 0.91–1.07, 4 studies), non-Hodgkin's lymphoma (mRR = 0.99, 95 % CI = 0.92–1.06, 5 studies), or thyroid cancer (mRR = 1.05, 95 % CI = 0.88–1.26, 3 studies). Long-term (10 + years) mobile phone use was also not associated with risk of leukaemia (mRR = 1.03, 95 % CI 0.85–1.24, 3 studies), non-Hodgkin lymphoma (mRR = 0.99, 95 % CI 0.86–1.15, 3 studies), or thyroid cancer (no pooled estimate given the small number of studies). There were not sufficient studies of any specific neoplasms to perform dose–response *meta*-analyses for either cumulative call time or cumulative number of calls; individual studies did not show statistically significant associations between lifetime intensity of mobile phone use and any specific neoplasm.
Occupational RF-EMF exposure (exposed *vs* unexposed) was not associated with an increased risk of lymphohematopoietic system tumours (mRR = 1.03, 95 % CI = 0.87–1.28, 4 studies) or oral cavity/pharynx cancer (mRR = 0.68, 95 % CI 0.42–1.11, 3 studies). There were not sufficient studies of any specific neoplasms to perform *meta*-analysis on the intensity or duration of occupational RF-EMF exposure; individual studies did not show statistically significant associations with either of those exposure metrics and any specific neoplasms.
The small number of studies, and of exposed cases in some instances, hampered the assessment of the statistical heterogeneity in findings across studies in the *meta*-analyses.
Based on the summary risk of bias, most studies included in the quantitative evidence syntheses were classified at moderate risk of bias. The most critical issue was exposure information bias, especially for occupational studies where the exposure characterization was rated at high risk of bias for all included studies. Outcome information bias was an issue in mortality-based occupational cohort studies investigating non-rapidly fatal neoplasms. Further, the healthy subscriber effect, and (at a lesser extent) the healthy worker effect, were identified as plausible explanations of the decreased risks observed in some studies.
The association of RF-EMF exposure from wireless phone use, or workplace equipment/devices, with other *important* neoplasms was reported by only one or two studies per tumour, so no quantitative evidence syntheses were conducted on these outcomes. It is noted that there were generally no statistically significant exposure-outcome associations for any combinations, independently of the exposure metric and level, with a few studies reporting decreased risks (especially for smoking-related cancers).
There was only one study which assessed the effect of RF-EMF exposure from fixed-site transmitters on less researched neoplasms and it reported no statistically significant associations between exposure from base stations and risk of lymphomas overall, lymphoma subtypes, or chronic lymphatic leukaemia in adults.
Conclusions: For near field RF-EMF exposure to the head from mobile phones, there was low certainty of evidence that it does not increase the risk of leukaemia, non-Hodgkin's lymphoma or thyroid cancer.
For occupational RF-EMF exposure, there was very low certainty of evidence that it does not increase the risk of lymphohematopoietic system tumours or oral cavity/pharynx cancer.
There was not sufficient evidence to assess the effect of whole-body far-field RF-EMF exposure from fixed-site transmitters (broadcasting antennas or base stations), or the effect of RF-EMF from any source on any other *important* neoplasms.
Other: This project was commissioned and partially funded by the World Health Organization (WHO). Co-financing was provided by the New Zealand Ministry of Health; the Istituto Superiore di Sanità in its capacity as a WHO Collaborating Centre for Radiation and Health; and ARPANSA as a WHO Collaborating Centre for Radiation Protection. Registration: PROSPERO CRD42021236798. Published protocol: [(Lagorio et al., 2021) DOI https://doi.org/10.1016/j.envint.2021.106828].

1. Introduction

1.1. Rationale

Radiofrequency (RF) electromagnetic fields (EMF) are part of the non-ionizing radiation region of the electromagnetic spectrum, which means that there is not sufficient energy in a single quantum of RF energy to ionize an atom or a molecule (Barnes et al., 2019). There is currently no established mechanism underpinning the potential carcinogenicity of RF-EMF at exposure levels below international standards (ICNIRP, 2020a; IEEE, 2019). The capacity of RF-EMF to induce genetic damage or other cancer-related effects (Smith and Guyton, 2020) has been assessed in a number of experimental studies. A *meta*-analysis of 225 studies of genetic damage in mammalian cells exposed to RF-EMF *in vitro* found no dose–response, and inverse correlations between effect size and study quality (Vijayalaxmi and Prihoda, 2019). According to a recent systematic review of 159 studies of genotoxicity in mammalian cell cultures exposed to RF-EMF, most experiments (80 % of 1,111) showed no effects of the exposure on the endpoints, especially the irreversible ones, independently of the exposure features, level, and duration, suggesting that RF exposure does not increase the occurrence of genotoxic effects in vitro (Romeo et al., 2024). A systematic review is in progress evaluating the effects of RF-EMF on cancer in experimental animal studies [see the published protocol (Mevissen et al., 2022)].

Independently of the pathogenesis, if exposure to RF-EMF increased the risk of cancer, then this would have serious public health consequences and require population-level preventive strategies, including a revision of the threshold-based limitation principle currently applied to non-ionizing radiation in the radiofrequency range (ICNIRP, 2020b).

RF-EMF was classified by the International Agency for Research on Cancer (IARC) as possibly carcinogenic to humans (group 2B), based on limited evidence in humans, limited evidence in experimental animals,

and weak support from mechanistic studies (IARC, 2013). The evaluation was driven by two large case-control studies showing positive associations between glioma and acoustic neuroma and wireless phone use (Baan et al., 2011). The IARC panel also examined studies of brain tumours, leukaemia/lymphoma, or other malignancies in relation to occupational or environmental RF exposure, and judged this evidence inadequate to formulate conclusions (IARC, 2013).

The IARC Monograph on RF-EMF covers the literature issued by mid-2011. Many new relevant studies have been made available since then. Several expert panels performed updated reviews of this body of evidence (ANSES, 2013, 2016; ARPANSA, 2014; CCARS, 2017; Demers et al., 2014; FDA, 2020; HCN, 2016; ICHENF, 2018; SCENIHR, 2015; SCHEER, 2023; SSM, 2013; 2014; 2015; 2016; 2018; 2019; 2020; 2021; 2022; 2024). Eighteen *meta*-analyses addressing mobile phone use and head tumour risks were published since 2012 (Bielsa-Fernandez and Rodriguez-Martin, 2018; Bortkiewicz, 2017; Bortkiewicz et al., 2017; Carlberg and Hardell, 2017; Chen et al., 2021; Choi et al., 2020; de Siqueira et al., 2017; Gong et al., 2014; Lagorio and Roosli, 2014; Moon et al., 2024; Prasad et al., 2017; Repacholi et al., 2012; Roosli et al., 2019; Safari Variani et al., 2019; Wang et al., 2018; Wang and Guo, 2016; Yang et al., 2017; Yoshikawa et al., 2023), often arriving at conflicting conclusions (Ioannidis, 2018).

None of these evidence syntheses complies in full with the recommendations for the conduct of systematic reviews in toxicology and environmental health research (COSTER) (Whaley et al., 2020), and only one protocol (Mao et al., 2013) of a *meta*-analysis later published in Chinese (Gong et al., 2014) was preregistered in PROSPERO.

The need for a structured updated appraisal of this body of evidence is widely recognised. Non-ionising radiation (radiofrequency) is among the agents recommended with high priority for re-evaluation by the Advisory Group for the IARC Monographs during 2020–2024 (Marques et al., 2019), and again in 2025–2029 (Berrington de Gonzalez et al., 2024). Two registered systematic reviews of epidemiological studies on RF-EMF and cancer are underway, focusing on exposures experienced by the general population (Farhat et al., 2020) and workers (Modenese et al., 2020).

The current systematic review is one of ten (Verbeek et al., 2021) commissioned by the World Health Organization in the framework of the ongoing assessment of health effects of exposure to radiofrequency electromagnetic fields (RF-EMF). We have previously reported selected findings from our systematic review of human observational studies on the effect of exposure to RF-EMF on cancer risk in the general and working population, focussing on most researched neoplasms (termed *critical* outcomes) (Karipidis et al., 2024). Here, we report on less researched neoplasms (termed *important* outcomes). To define the two sets of outcomes (*critical*, *important*), we adopted the terminology suggested by Cochrane (MECIR standard C14) for systematic reviews dealing with a large number of outcomes eligible for inclusion (Higgins et al., 2020).

2. Objectives

The overall aim of this systematic review was to assess the quality and strength of the evidence provided by human observational studies for a potential causal association between exposure to RF-EMF and risk of neoplastic diseases. The specific objectives were: (i) identify the relevant epidemiological literature; (ii) assess risk-of-bias for individual studies; (iii) synthesize the evidence on the exposure-outcome relationship (in terms of magnitude of effects and shape of exposure–response gradients) and evaluate heterogeneity in results across studies; (iv) rate confidence in the body of evidence.

No epidemiological study to date has investigated the risk of neoplastic diseases in relation to individual exposure to RF-EMF from all exposure sources and settings (AGNIR, 2012; ARPANSA, 2014; FDA, 2020; IARC, 2013). Therefore, we separately reviewed three bodies of evidence, addressing neoplasia risk in the general population in relation to RF exposure from near-field (SR-A) or far-field (SR-B) sources, and in working age individuals in relation to occupational RF exposures (SR-C).

The scientific questions expressed as PECO (Population, Exposure, Comparator, Outcome) statements (Morgan et al., 2018) are shown in Table 1.

3. Methods

The methods for this systematic review and *meta*-analysis are described in detail in the published protocol (Lagorio et al., 2021), and summarised below. The amendments to the protocol are reported within the text in each relevant section, and later listed in § 6.2. Findings from the systematic review are reported in accordance with the updated PRISMA-2020 guidelines for reporting systematic reviews (Page et al., 2021b). Findings in relation to the *critical* outcomes are published in a companion paper (Karipidis et al., 2024).

Table 1
PECO statements.

Systematic review of studies on RF-EMF exposure from wireless phone use	
Population	Humans (members of the general population), without restriction based on sex, age, or other individual characteristics.
Exposure	**Definition**: Near-field RF exposure from personal use of mobile or cordless phones, occurring prior to outcome, and based on indirect measures (subscriber status, self-reported history of mobile phone or cordless phone use), traffic data, or modelling. **Classification**: Ever exposed; time since first exposure; cumulative exposure level.
Comparator	No or low-level exposure (never or non-regular users of wireless phones).
Outcomes	**Critical†:** (Incidence-based) glioma/brain cancer in adults; paediatric brain tumours*; meningioma; acoustic neuroma; pituitary gland tumours; salivary gland tumours. **Important‡:** Any other neoplasm investigated in relation to the exposure of interest.
Systematic review of studies on RF-EMF exposure from environmental sources	
Population	Humans (members of the general population), without restriction on sex, age, or other individual characteristics.
Exposure	**Definition:** Far-field RF exposure from radio-television transmitters, base stations or any other fixed-site transmitter, occurring prior to outcome, and based on environmental measures, modelling, or geocoded distance to the sources (the latter limited to broadcast transmitters). **Classification:** Ever exposed; duration of exposure or time since first exposure; average or cumulative exposure level.
Comparator	No or low-level exposure from environmental sources of RF-EMF.
Outcomes	**Critical†:** (Incidence-based) childhood leukaemia, paediatric brain tumours*, glioma/brain cancer in adults, and leukaemia in adults. **Important‡:** Any other neoplasm investigated in relation to the exposure of interest.
Systematic review of studies on occupational exposures to RF-EMF	
Population	Occupationally active individuals, with no further restriction on sex, age, or other individual characteristics.
Exposure	**Definition:** Near- or far-field RF exposure from professional use of hand-held transceivers or RF-emitting equipment in the workplaces, occurring prior to outcome, and based on measurements, estimates of exposure level from job- or source-exposure matrices (JEM, SEM), or indirect measures such job title or task (option limited to studies explicitly aimed at assessing the effect of exposure to well-characterized sources and types of RF-EMF). **Classification:** Ever exposed; exposure frequency; exposure duration or time since first exposure; average or cumulative exposure level.
Comparator	No or low-level occupational exposure to RF-EMF.
Outcomes	**Critical†:** (Incidence-based) Glioma/brain cancer, leukaemia. **Important‡:** Any other neoplasm investigated in relation to the exposure of interest.

*Brain tumours in children, adolescents and young adults; †Findings in relation to the *critical* outcomes are published elsewhere (Karipidis et al., 2024); ‡ Findings relating to neoplasms other than the those defined as "critical" in each subset of the systematic review are presented in the current paper.

3.1. Eligibility criteria

3.1.1. Types of populations

SR-A and SR-B focused on members of the general populations, and SR-C on occupationally active individuals. No restrictions on sex, age, or other individual characteristics were applied.

3.1.2. Types of exposures

Given the lack of a known biological mechanism for a potential carcinogenic effect of RF-EMF, it is unknown which aspect of the exposure may be biologically relevant. Therefore, the choice of the exposure metrics of priority interest was informed by contextual evidence relevant for the types of RF exposure considered in each component of the systematic review, summarized below.

3.1.2.1. RF exposure from wireless phone use. Mobile phones are the most common type of wireless phones and their use is now universal, with 8.6 billion subscriptions in 2022, corresponding to 108 subscriptions per 100 inhabitants (ITU, 2022). Given the short time period since the introduction of 5G technology, we do not expect to identify studies addressing the association between 5G mobile phone use and neoplasia risk. However, epidemiological studies of radar workers exposed to RF-EMF $>$ 6 GHz have been conducted (Karipidis et al., 2021) and were considered for inclusion in SR-C.

In SR-A, we summarized the evidence for the exposure variables most commonly used in the scientific literature: ever use of mobile phones, time since start of mobile phone use (TSS; also called time since first use), cumulative hours of mobile phone use (also called "cumulative call time", CCT), and cumulative number of calls (CNC).

The variable TSS is a crude measure, but it takes into consideration the tumour latency, which may vary between tumour types.

The variables CCT and CNC provide better estimates of the total amount of mobile phone use, but are more greatly affected by recall bias (Aydin et al., 2011; Vrijheid et al., 2009) because past intensity of use is more difficult to recall than current use, especially as mobile phone habits have changed considerably over time.

Cordless phones are another source of near-field exposure to RF-EMF. It is worth noting that the transmission power of cordless phones is 1–2 orders of magnitude lower than that of 1G-2G mobile phones (Lauer et al., 2013) but similar to average transmission power for 3G and 4G network calls. RF-exposure from cordless phones can only be assessed based on indirect measures from interviews or questionnaires (prevalence, amount and duration of use), and there are no objective sources of data against which self-reported information can be validated.

3.1.2.2. Environmental RF exposure from fixed-site transmitters. In SR-B, we included studies addressing neoplasm risks in relation to RF exposure from radio and television masts, base stations or any other fixed-site transmitter. In principle, the average or cumulative whole-body specific absorption rate (SAR) is the exposure measure of interest. As the SAR cannot be directly measured, epidemiological studies have usually relied on measured or modelled levels of electric fields, magnetic fields or power density at the subjects' residence (less often also at schools), or on crude exposure proxies such as distance to the exposure source.

For a given transmitter, the electric field decreases in the beam with 1/distance from the source. Provided that the distance is objectively recorded (e.g., derived from geocodes), distance from the source may be informative for antennas with a roughly isotropic transmission pattern. This is usually the case for large broadcast transmitters, although special care must be taken when different transmitters are included in the same study (Schmiedel et al., 2009). On the contrary, distance from a base station is a poor indicator of exposure to RF-EMF indoors, due to the complex propagation characteristics of emissions from base station antennas, including shielding effects and multiple reflections from house walls and other buildings (Frei et al., 2010).

We restricted eligibility for inclusion to studies based on objective exposure indicators, such as measurements, modelling, or geocoded distance to a broadcast transmitter (but not to a mobile phone base station). Studies based on self-estimated distance to an antenna were not included, as self-reported distance to transmitters is strongly affected by risk perception (Martens et al., 2017) and cannot be considered a reliable exposure indicator. We focused on differences in exposure level (using categorical or continuous exposure data), and according to exposure duration.

3.1.2.3. Occupational RF exposures. Most epidemiological studies conducted so far used job-titles as exposure surrogates. Previous reviews of the relevant publications have considered the evidence uninformative, due to inconsistent results across studies affected by severe limitations in exposure assessment, and uncontrolled confounding (AGNIR, 2012; IARC, 2013). Bias in study identification due to selective mention of RF exposures for occupations found at increased cancer risk, was an additional concern identified in these reviews. In SR-C, we included studies investigating neoplasia risk in relation to exposure to RF-EMF from professional use of hand-held transceivers, or from RF-emitting equipment in the workplace, with exposure assessment based on measurements or estimates of exposure level derived from JEM or SEM. We also considered eligible for inclusion studies with indirect measures of exposure (job title or task), provided that the assessment of the effect of RF-EMF exposure was a predefined research objective, the exposure was well characterized in terms of source and type (equipment/device, frequency band, power), and the requirements concerning the exposure contrasts were met. We excluded studies based on self-reported exposure only (i.e., without information on job, task and/or exposure source). We also excluded studies addressing occupations where exposures to electric and magnetic fields between 0 Hz and 10 MHz were dominant compared to the co-occurring exposure to RF-EMF (e.g., MRI machine operators, arc-welders, or electricity production and distribution workers), or with dominant exposures to established carcinogens, without reliable assessment of RF-exposure and appropriate confounding control. The priority exposure classifications were ever *vs* never exposed, exposure frequency, exposure duration or time since first exposure, average or cumulative exposure level.

3.1.3. Types of comparators

To be eligible for inclusion, studies must have compared the occurrence of the outcome between exposed and unexposed subjects, or between at least two groups with different exposure frequency, intensity, duration, time since first exposure, average or cumulative exposure level.

3.1.4. Types of outcomes

3.1.4.1. Critical and important outcomes. While no eligibility restriction on tumour type was applied, we split the findings of this systematic review into two papers. In a companion article (part I), we focussed on the most researched, which we termed *critical* tumours: i.e., neoplasms of the central nervous system (brain, meninges, pituitary gland, acoustic nerve) and salivary gland tumours (SR-A); brain tumours and leukaemias (SR-B, SR-C) (Karipidis et al., 2024).

The current paper (part II) focuses on all other (termed "*important*") neoplasms.

3.1.4.2. Diagnostic methods and measures of occurrence. We considered eligible for inclusion studies including newly diagnosed (incident) cases of the diseases of interest, either histology-confirmed or based on unequivocal diagnostic imaging (the latter criterion only applies to central nervous system tumours), ascertained through cancer registries, hospitals, or other sources with adequate coverage of the study base during

the observation period. We excluded studies based on self-reported outcomes, as well as on hospital admissions only (due to uncertainties about the date of diagnosis). Information from death certificates was considered the least valid basis of diagnosis for neoplasms (Jensen et al., 1991). Studies based on cancer-related causes of death were eligible for inclusion in the "*important*" outcome subset, reviewed herein, only for cohort studies (see 3.1.5.1). Note that the studies examined in part I of the current review (Karipidis et al., 2024) were all incidence-based.

3.1.5. Types of studies

3.1.5.1. Inclusion criteria. Eligibility for inclusion was restricted to cohort and case-control studies, comprising all related typologies (Gail et al., 2019). If the measures of effect were based on cancer mortality, eligibility for inclusion was further restricted to cohort and cohort-nested case-control studies; population-based case-control studies with deceased cases and controls were not included, because this study design renders the identification of the study base difficult or impossible.

3.1.5.2. Exclusion criteria. Case reports and case series were ineligible for inclusion due the lack of a control group. We also excluded comparative studies such as ecological studies (geographical correlation and time-trend analyses), cross-sectional studies, and case-case analyses of case-control studies, because these study designs do not allow calculating the intended measures of effect.

3.1.5.3. Complementary evidence. In line with the triangulation approach (Arroyave et al., 2021; Lawlor et al., 2016; Steenland et al., 2020), we systematically searched for and included studies aimed at estimating the amount and direction of exposure measurement errors or other distortions (termed "bias studies"), conducted in the framework of included studies, or directly relevant to the investigated E-O pairs. In addition, we included source-specific RF dose-modelling, and studies based on incidence time trends of specific types of CNS tumours, but these were used in our other paper on "*critical*" neoplasms and are not relevant in the current paper on *important* neoplasms.

3.1.5.4. Years considered. No restriction on publication date was applied.

3.1.5.5. Publication language. We did not exclude any article based on language, but the search queries included English terms only. During screening articles for inclusion, publications in languages other than the ones spoken by the reviewers (English, French, German, Greek, Italian, Portuguese) were translated into English using Google Translate. Actually, we did not find potentially relevant articles where we were in doubt about inclusion after automatic translation, and the intervention of a human translator was not necessary.

3.1.5.6. Publication types. We included peer-reviewed journal articles reporting original data from eligible study types. We considered indexing in Medline as evidence of peer-review status. We excluded reviews, *meta*-analyses, conference articles and proceedings, editorials, comments and letters, with the exception of correspondence related to the included studies (such as letters by the authors reporting errors in the published analysis, providing more detailed or extended data analyses, or discussing study strengths and biases).

3.1.6. Types of effect measures

We focused on studies reporting incidence-based estimates of the relative risk of disease conditional on the exposure: rate ratio (RR) or hazard ratio (HR) in cohort studies and odds ratios (OR) in case-control studies. We also included cohort studies with mortality-based estimates of relative risk (i.e., standardized mortality ratios – SMR, or mortality rate ratios – MRR). Because of the rarity of the neoplasms of interest, the HR and the OR can be considered equivalent to a RR (Higgins et al., 2021a). Moreover, possible *meta*-analyses were performed on log-transformed measures of effect and confidence limits (CLs).

3.2. Information sources and search strategy

Eligible studies were identified by literature searches through Medline and Embase. We also consulted the EMF Portal (https://www.emf-portal.org/en), a dedicated database of the scientific literature on the health effects of exposure to electromagnetic fields, with documented high coverage of the topic (Drießen et al., 2017). The search timeframe (as in-print publication) extended from the database inception dates (1946 for Medline; 1947 for Embase) to 11 March 2021 (i.e., the date of the actual literature searches).

To comply with the MECIR requirement and COSTER recommendation 2.7 to update the searches within 12 months before publication of the review (Higgins et al., 2020; Whaley et al., 2020), we conducted repeated selective monitoring of the EMF-Portal to identify relevant studies published up to 31 December 2022. This was an amendment to the protocol, which envisaged to update the searches through all main databases (see § 6.2 *Amendments to the protocol*, point 1), introduced because the precision [1-(excluded record / total retrieved)] of EMF-Portal was much greater than that of the other two sources (0.34 *vs* 0.05 for Medline, and 0.04 for Embase).

The Medline and Embase queries are reported in Annex 1 (§ 2–3). The search on EMF-Portal took advantages of the in-built facilities; to identify cohort and case-control, we toggled "Epidemiological studies" (as Topic), and "Radio frequency ($\geq$ 10 MHz)" or "Mobile communications" (as Frequency range), with "cancer" OR "tumour" as keywords; for exposure validation and dosimetry studies, we selected "Technical/dosimetric studies" and the above frequency ranges. As an additional source, we used a library of over 400 "seed" studies (see Annex 1, § 1, Table 1), taken from the reference lists of 19 recent comprehensive reviews (AGNIR, 2012; ANSES, 2013, 2016; ARPANSA, 2014; CCARS, 2017; Demers et al., 2014; FDA, 2020; HCN, 2016; IARC, 2013; ICHENF, 2018; ICNIRP, 2020a; SCENIHR, 2015; SSM, 2013; 2014; 2015; 2016; 2018; 2019; WHO 2014). We used this library to calibrate and assess the performance of draft Medline queries, intentionally designed to privilege sensitivity over precision (0.89 *vs* 0.09, in the final version of the queries; Annex 1, § 1, Table 2).

As secondary sources of unidentified relevant articles, we also hand-searched the reference lists of included studies and consulted the authors' own archives.

3.3. Selection process

EndNote 20 was used for the assemblage of the results of the literature searches, duplicate removal, and data management during the study selection process (Bramer et al., 2017; Peters 2017). We categorized the identified records by coherence with the subject of the systematic review and other features relevant to assess compliance with the predefined inclusion/exclusion criteria. This categorization occurred at the title/abstract or full-text screening levels of the review, as appropriate. Two reviewers (DB, MSP) independently assessed the relevance of the identified articles, and their eligibility for inclusion in any of the three systematic reviews. Then, both reviewers shared their EndNote libraries with two other team members (KK, SL) who revised and finalized the study selection. All four reviewers, provided with written instructions on categorization scheme, variable coding, and treatment of multiple publications per study, participated in a pilot testing of the study selection procedures undertaken on a small subset of the references retrieved.

3.3.1. Selection of eligible articles

Full-text articles were retrieved for all records classified as certainly or possibly relevant. Eligible article types (original studies and related

Table 2
Data extraction elements.

Topic	Items
Article	First author and publication year, full reference
Study	Study design: cohort; nested case-control study; population-based case-control study hospital-based case-control study; other design variants (specify)
	Study acronym (if any)
Subjects	Study population (description)
	Geography (country, region, state, etc.)
	Dates of study and sampling time frame (period of case ascertainment)
	Demographics (sex; age or lifestage at exposure and at outcome assessment)
	Number of subjects (target, enrolled, number per group in analysis)
	Person-years of observations, length of follow-up and follow-up rates per exposure group [cohort]
	Participation rates of cases and controls (possibly for exposed and unexposed separately, in each series) [case-control]
Methods	Inclusion/exclusion criteria and recruitment strategy
	Case ascertainment: cancer register; hospital-based; other source (specify)
	Case type: incident cases; cases alive at enrolment; deceased cases
	Reference group description [cohort]
	Control type: population based (source and sampling method); hospital based (type of diagnoses); other types (specify) [case-control]
	Proportion of proxies interviewed among cases and controls [case-control]
	Outcome type(s): one or more of the following: glioma, brain tumours (when only topography available), paediatric brain tumours*, meningioma, acoustic neuroma, pituitary tumour, salivary gland tumours; childhood leukaemias†; adult leukaemias; other type (specify)
	Outcome assessment: diagnostic methods (histology-based, %; imaging-based, %; cause of death only; not given)
	Exposure assessment timing: prospective *vs* retrospective (i.e., before *vs* after outcome occurrence, diagnosis or ascertainment)
	Exposure assessment methods (self-administered questionnaire, personal interview; computer assisted personal interview, network-operator customer lists; measurements, modelling, geocoded distance to a broadcast transmitter; JEM, SEM; occupational sector, job title, task)
	Exposure variables used in the analyses (e.g., ever *vs* never exposed; length of exposure; time since first exposure; exposure frequency; exposure level; cumulative exposure; others – specifying the variable unit and type: dichotomous/categorical/continuous)
	Statistical methods (specify)
Results	Mean/median exposure value within each exposure interval (for all relevant metrics)
	Number of cases and persons-years or total number of subjects per exposure level, including unexposed [cohort];
	Number of cases and controls per exposure level, including unexposed [case-control];
	Type of relative risk estimate (OR, HR, IRR, SMR)
	Measures of effect and confidence limits (CI) for each prioritized exposure contrast
	Confounders or modifying factors and how they were considered in analysis (i.e., list of factors included in final model, or considered for inclusion but found to have little or no impact on the measures of effect and therefore not included in the final model)
Funding	Funding source

*Usually referring to diagnoses in the age range 0–19 years; †Usually referring to diagnoses in the age range 0–14 years.

correspondence) were further categorized by study design, setting/source of exposure to RF-EMF (mobile phone and/or cordless phone use; environmental sources; occupational sources), and investigated neoplasm(s). Eligibility for inclusion was then assessed based on compliance with the predefined inclusion/exclusion criteria. At completion of this stage, all identified articles were divided into four groups: (i) irrelevant; (ii) relevant but ineligible for inclusion, with reason(s) for exclusion specified (recording "various" and specifying which, if more than one applied); (iii) relevant and eligible for inclusion in one of the three systematic reviews (or in more than one, if multiple types of RF-EMF exposure were investigated); (iv) included as complementary evidence (or in both the aetiological and complementary evidence group, when appropriate).

3.3.2. Selection of eligible studies

We classified all the included articles by the investigated exposure (s), and outcomes(s), and our definition of the term "study" corresponds to each identified homogeneous exposure-outcome (E-O) pair (i.e., articles addressing multiple E-O pairs had multiple corresponding studies).

3.3.3. Disagreement between reviewers

Disagreements between reviewers involved in article and study selection (including decisions on between-study overlap) were resolved by discussion; if no consensus could be reached, a final decision was made by the two reviewers in charge of the study selection for each line of evidence.

3.3.4. Reporting of information flow

We documented the selection process in a study flow diagram according to the PRISMA-2020 reporting guidelines (Page et al., 2021b).

3.4. Data extraction process

For each included study, a standard set of details was extracted from the relevant publications (Table 2). The study design is reported in brackets when data refer to either cohort or case-control studies (including variants thereof); lack of specification means relevance for both main study designs.

For all prioritized exposure contrasts, we extracted from each neoplasm-specific study the most (appropriately) adjusted measure of effect and 95 % confidence interval per exposure category.

From the entire dataset of included studies, six subsets of equivalent size were assigned to as many team members (DB, CB, CN, KK, TL, MSP) who extracted and recorded the relevant data in the predefined templates (Study Key-Feature tables, and Summary of Findings tables). Three reviewers (CB, KK, SL) merged and checked the extracted information for completeness and accuracy as a quality control measure. Information inferred, converted, or estimated after data extraction, was recorded in the analytical datasets, and annotated with a rationale.

3.4.1. Missing data

We requested missing data considered important for the review (e.g., study key-features, and/or data required to conduct a *meta*-analysis) from the corresponding author by email, using the contact details available from the study report. We made two attempts of contact, two weeks apart. In case of no response within one month of the second, we considered the attempt unsuccessful.

3.5. Risk of bias assessment

3.5.1. Risk of bias in studies

To assess the study's internal validity, or risk of bias (RoB), we followed the method developed by the National Toxicology Program – Office of Health Assessment and Translation (NTP-OHAT 2019). As per the OHAT's approach, we created a version of the OHAT RoB tool (NTP-OHAT 2015) tailored to the topic of our review, focussing on the bias questions applicable to the study designs eligible for inclusion. The bias domains of relevance for observational cohort and case-control studies were: confounding; selection bias; attrition/exclusion/missing data bias; confidence in the exposure characterization; confidence in the outcome assessment; selective reporting; and appropriateness of statistical methods. In the sections addressing selection and outcome-information biases, the RoB tool developed by the Office of the Report on Carcinogens (NTP-ORoC 2015) was also referred to. Detailed information on the customization process, along with the tailored bias rating instructions and answer option forms, are provided in the annexed RoB protocol (Annex 2).

We performed the RoB assessment at the exposure-outcome level, as many studies eligible for inclusion in the current review reported on different neoplasms and multiple types/sources/settings of exposure to

RF-EMF. This was in line with the Cochrane approach (Higgins et al., 2021b; Sterne et al., 2021), COSTER recommendation 5.2 (Whaley et al., 2020), and other guidance on conducting systematic reviews of observational studies of aetiology and risks from environmental or occupational exposures (Arroyave et al., 2021; Dekkers et al., 2019; Radke et al., 2019).

The potential for bias of each neoplasm-specific study and related exposure-outcome contrasts was rated in duplicate by two assessors. The number of studies to be rated were divided approximately equally amongst two assessor pairs (DB-TL, MSP-KK). No assessor evaluated studies that they co-authored. Rating conflicts were resolved by consensus of all four assessors. All assessors were trained in two working sessions, and a pilot-study (based on five studies per rater pair) was undertaken right after completion of the study selection. A final consistency check of all the ratings was carried out by three assessors (DB, KK, RM).

Contrary to what was envisaged in the protocol, managing the RoB through the Health Assessment Workplace Collaborative (HAWC) platform (Shapiro et al., 2018) proved unfeasible; we used ad hoc paper forms for the ratings (Annex 2, § 1.7, Table 5) and Excel for the production of the heat maps (see § 6.2 *Amendments to the protocol*, point 2).

3.5.2. Summary assessments of risks of bias

We applied the OHAT's 3-level tiering of the quality of individual studies, based on summary assessments of risk of bias for the domains most relevant to the specific systematic review (NTP-OHAT 2019). This tiering differs from scaling and is consistent with the Cochrane's overall risk-of-bias judgement (Higgins et al., 2021b; Sterne et al., 2021). We focused on four key-items including selection/attrition biases, and exposure/outcome information biases.

Tier-1 comprised studies with definitely or probably low risk of bias for all key-items and most of other items; tier-3 included studies with definitely or probably high risk of bias for all key-items and most of other items; and studies non-compliant with the above criteria were classified as tier-2.

The choice of the exposure information bias and the selection/attrition bias as key-domains for the tiering, was driven by the expected features of the dataset, as known from previous reviews on the topic at the stage of the protocol drafting, and confirmed after performing the review.

Over two thirds of exposure-outcome pairs reviewed in this paper (101 out of 143) are from cohort studies, and the latter design is especially common in studies investigating the effect of occupational RF-exposure on several outcomes reviewed herein (56 out of 62 E-O pairs).

Exposure to RF-EMF is particularly difficult to assess, and all studies included in the current review are prone to random, systematic and differential exposure measurement errors. Random exposure misclassification or mismeasurement usually bias the study results towards the null. Noteworthy, if the exposure had no effect on the outcome (under the null), random errors result in loss of precision with no bias. The potential for recall bias depends on the study design. Differential exposure misclassification cannot occur in cohort studies with prospective exposure assessment independent of the outcome. Recall bias, leading to overestimates of the exposure effect, is of concern in case-control studies with retrospective exposure assessment based on self-reports.

Exposure characterization is usually far from satisfactory in cohort studies of workers exposed to RF-EMF (AGNIR 2012; IARC 2013). In studies with exposure assessment based on job- or source-exposure matrices (JEM, SEM), different types of error can affect the two components of the matrix: (a) measurement-based data on exposure level by job/task; (b) individual occupational history data. JEM/SEM-based studies with occupational histories from independent records (e.g., census-based occupational cohort studies, industry-based cohort studies, and case-control studies nested in these cohorts), are susceptible to random exposure misclassification. When a JEM/SEM is used in a case-control study with self-reported occupational histories, both random errors and recall bias are of concern. Few validation studies of indicators of occupational exposure to RF-EMF have been performed to date. In a cohort of British police officers (Gao et al., 2019), self-reported data on TETRA use were compared with objective radio usage records; for weekly use, participants under-reported the number of calls and over-reported the duration of calls by a factor of around 4 and 1.6 respectively, and bias was higher for daily usage (Vergnaud et al., 2016). Few details are provided on a validation of an industry-based JEM in the cohort study of Motorola employees, mentioned in the paper reporting on cancer mortality (Morgan et al., 2000).

Within the selection bias domain (Q3 answer option form), the OHAT RoB tool consider two issues specific of occupational cohort studies, currently interpreted as a form of confounding by health status (Green-McKenzie 2017; Naimi et al., 2013):

- The selection of healthy workers into the workplace (healthy-worker hire effect, HWHE), of concern in occupational cohort studies using the general population as the reference group (i.e., reporting standardized incidence or mortality rate ratios as measures of effect);
- The selection of unhealthy workers out of the workplace (healthy worker survival effect, HWSE), a form of time-varying confounding, which may be an issue in cohort studies enrolling prevalent (as opposed to incident) exposed workers, regardless of the type of reference group.

However, we assessed the potentials for HWHE/HWSE and selection bias separately.

Compared to cohort studies with exhaustive case ascertainment independent of the exposure, the case-control design is much more susceptible to selection/attrition bias via several mechanisms (e.g., differential participation, and differential missing data at enrolment or at the analysis stage, just to quote the major ones). The reasons why we considered selection and attrition biases (as per the OHAT RoB tool) as essentially equivalent in terms of bias structure are provided in our RoB protocol (Annex 2).

The outcome information bias was considered as an additional key-bias. Many studies reviewed herein are mortality-based cohort studies, possibly liable to errors in outcome ascertainment, especially for non-rapidly fatal neoplasms.

3.6. Synthesis methods

We summarized the main features of all included studies in tables grouped and ordered by exposure type/setting/source (SR-A, SR-B, and SR-C), neoplasm, and study design. Templates of the key study characteristic tables for cohort and case-control studies, as well as for the summary of findings tables were provided in the online annexes to the published protocol (Lagorio et al., 2021).

The outcome, the exposure, and age at diagnosis are the most relevant factors affecting comparability between studies eligible for inclusion in our review. We did not combine exposure-outcome pairs of different tumours (in terms of ICD-O-3 main site or histology groups), neoplasm-specific risks from different exposure types and metrics, or risk of a specific tumour in relation to a given exposure type/metric in adults and paediatric population (0–19 years).

For homogenous datasets (in terms of outcome, exposure type/metric, and subjects' lifestage), we set a minimum size requirement for amenability to a *meta*-analysis (i.e., at least 3 reported effect estimates). This was a deviation from the protocol (see § 6.2. *Amendments to the protocol*, point 3). To address concerns about the large uncertainty in heterogeneity statistics from *meta*-analyses based on few studies (Fu et al., 2008; Ioannidis et al., 2007), we calculated the confidence intervals of the I^2 statistics using the Stata heterogi module (Higgins and Thompson 2002; Orsini et al., 2005).

The synthesis of findings from the study subsets not meeting the

requirements for inclusion in a *meta*-analysis was based on a structured tabulation of study key-features (Annex 5) and results(Annex 7). Contrary to what was envisaged in our protocol, we did not prepare visual plots (Anzures-Cabrera and Higgins 2010; McKenzie and Brennan 2021) for studies that were not included in *meta*-analyses (see § *6.2. Amendments to the protocol*, point 4). This was due to the large number of exposure-outcome combinations with one or two studies per pair (124 E-O pairs, see distribution by exposure and neoplasms in Table 3).

We summarize below the pre-planned *meta*-analyses of studies included in SR-A. A similar approach was followed if a quantitative synthesis of data from other lines of evidence (SR-B, SR-C) was considered feasible.

3.6.1. Meta-analyses of studies on wireless phone use and risk of neoplasms in the head region

The *meta*-analyses were neoplasm- and exposure-specific in relation to usage of each type of wireless phone (mobile or cordless). We used the natural logarithms of the most (appropriately) adjusted point estimates of relative risk (IRR, HR, OR), and related 95 % CLs, extracted from the relevant articles as input for the *meta*-analyses, focussing on the exposure metrics and contrasts below.

- For the binary exposure variable "ever *vs* never" (regular) use, we performed *meta*-analyses stratified on study design and based on random-effects restricted likelihood (REML) models, using the I^2 statistic (Higgins et al., 2003) to assess the statistical heterogeneity in results across studies. To describe the degree of heterogeneity detected via the I^2 index, we tried to be consistent with the Cochrane's guidance (Deeks et al., 2021), whereby: 0 % to 40 %: might not be important; 30 % to 60 %: may represent moderate heterogeneity; 50 % to 90 %: may represent substantial heterogeneity; and 75 % to 100 %: considerable heterogeneity. For studies reporting results separately for men and women we combined these using inverse variance weighted average (IVWA) fixed effects models. Differences between cohort and case-control studies were assessed using the test for group differences (Q_b statistics).
- For the categorical variable TSS, we had planned subgroup *meta*-analyses for "standard" classification cut-points, namely short-term (<5 years), mid-term (5–9 years), and long-term (≥10 years) use *vs* no exposure, but there were only enough studies to conduct *meta*-analyses for long-term use.
- Although we had planned to perform dose–response *meta*-analyses of neoplasm risks per CCT and CNC, there were not enough studies of any *important* neoplasms with these exposure metrics available.

The analyses were performed using the *meta*-analysis software developed in Stata 18 (Palmer and Sterne 2016).

3.6.2. Secondary analyses

Although we had planned to perform different secondary analyses, including cumulative *meta*-analyses and sensitivity analyses with various exclusions (Lagorio et al., 2021), there were not enough studies in the evidence base reviewed herein.

3.6.3. Reporting bias assessment

Reporting bias (or "*meta*-bias" (Shamseer et al., 2015)), comprises several kinds of distortions due to missing data in a synthesis (Page et al., 2021a; Sedgwick 2015). We attempted to minimize language bias by including studies in any language. We used both funnel plots and the Egger's test to examine funnel plot asymmetry.

3.7. Certainty assessment

We assessed the confidence in evidence for specific exposure-outcome combinations, as described in the predefined protocol (see Annex 3 for details).

In brief, we followed the OHAT GRADE-based method (NTP-OHAT 2019). Based on this approach, the level of confidence in the exposure-outcome association was classified according to four descriptors:

- **High (++++)**: The true effect is highly likely to be reflected in the apparent relationship.
- **Moderate (+++)**: The true effect may be reflected in the apparent relationship.
- **Low (++)**: The true effect may be different from the apparent relationship.
- **Very Low (+)**: The true effect is highly likely to be different from the apparent relationship.

The process consisted of three steps. At first, we assigned an initial rating of "moderate" confidence to all studies included in our systematic review. This is in line with the GRADE approach which foresees that an initial "high confidence" rating is assigned only to experimental studies, complying with 4 criteria (controlled exposure, exposure prior to outcome, individual outcome data, and use of a comparison group).

During the second step, we considered five possible downgrading factors (summary risk of bias, unexplained inconsistency; indirectness; imprecision; publication bias), and three possible upgrading factors (large magnitude of effect; dose response; residual confounding or other factors counter to the observed effect). Note that the OHAT approach considers the evidence provided by epidemiological studies as directly relevant to the assessment of human health hazards; therefore, we did not downgrade for indirectness.

In the third step, we assessed the confidence in evidence across multiple exposure types for specific neoplasms, and across multiple outcomes for specific exposures. As a change to our original protocol, we did not provide a confidence in evidence rating where the evidence consisted of less than three studies because, in the lack of a *meta*-analysis, several items required to perform the assessment are not available (i.e., inconsistency, effect size, and dose–response) (see § *6.2. Amendments to the protocol*, point 3).

In formulating our overall conclusions, we took into account the exposure-outcome specific certainty in evidence ratings, and the internal coherence of the original study findings (based on ranking of RF sources by exposure level as inferred from dosimetric studies).

To enhance clarity in conveying findings from our systematic review, we formulated our conclusive statements in line with the wording suggested by the GRADE guidelines 26 (Santesso et al., 2020); this was not originally envisaged (see § *6.2. Amendments to the protocol*, point 5).

4. Results

4.1. Study selection

From the searches through Medline (2,068 records), Embase (2,752 records), and EMF-Portal (240 records) we identified 5,060 records, of which 1,193 were duplicates, leaving 3,867 records for screening. In addition, 42 records were retrieved from the previously mentioned "seed-study" library (n = 18), citation searching (n = 6), selective monitoring of EMF-Portal up to December 2022 (n = 16), and the team members' archives (n = 2). Details about the study identification and screening process are provided in Fig. 1. Note that the flow-diagram refers to the whole process of study identification and selection, independent of the type of outcome (*critical* or *important*), recalling that the splitting of our systematic review's report into two papers was only due to the extremely large number of exposure-outcome pairs in the dataset.

4.1.1. Excluded articles

The 3,867 records identified through the main literature databases were pre-screened using EndNote scripts supplemented by human revision. This process excluded 1,877 records, leaving 1,990 records plus the 42 records identified via other sources (total of 2,032 records)

Table 3
Included studies of "*important*" neoplasms (26 articles) by exposure-outcome (E-O) pairs (n = 143). (See below-mentioned references for further information.)

SR subset	RF-Exposure Type, Source, Setting	Neoplasm	ICD-10 Code	Article	Design	Outcome measure	E-O Pairs	Total
SR1-A	Mobile phones	Basal Cell Carcinoma (head & neck)	C44	(Poulsen et al. 2013)	Cohort	Incidence	1	71
		Basal cell carcinoma-torso & legs	C44	(Poulsen et al. 2013)	Cohort	Incidence	1	
		Bladder cancer	C67	(Schuz et al. 2006)	Cohort	Incidence	2	
			C67	(Benson et al. 2013)	Cohort	Incidence		
		Breast cancer (women)	C50	(Schuz et al. 2006)	Cohort	Incidence	2	
			C50	(Benson et al. 2013)	Cohort	Incidence		
		Colon cancer	C18-21	(Johansen et al. 2001)	Cohort	Incidence	2	
			C18-21	(Benson et al. 2013)	Cohort	Incidence		
		Endometrium cancer	C54	(Benson et al. 2013)	Cohort	Incidence	1	
		Eye cancer	C69	(Schuz et al. 2006)	Cohort	Incidence	2	
			C69	(Benson et al. 2013)	Cohort	Incidence		
		Kidney cancer	C64-66	(Schuz et al. 2006)	Cohort	Incidence	2	
			C64-66	(Benson et al. 2013)	Cohort	Incidence		
		Larynx cancer	C32	(Schuz et al. 2006)	Cohort	Incidence	1	
		Leukaemias (Any type)	C91-95	(Schuz et al. 2006)	Cohort	Incidence	4	
			C91-95	(Kaufman et al. 2009)	CaCo	Incidence		
			C91-95	(Cooke et al. 2010)	CaCo	Incidence		
			C91-95	(Benson et al. 2013)	Cohort	Incidence		
		Leukaemia subtype – ALL	C91	(Kaufman et al. 2009)	CaCo	Incidence	2	
			C91	(Cooke et al. 2010)	CaCo	Incidence		
		Leukaemia subtype – AML	C92	(Kaufman et al. 2009)	CaCo	Incidence	2	
			C92	(Cooke et al. 2010)	CaCo	Incidence		
		Leukaemia-Subtype: CLL	C91	(Satta et al. 2012)	CaCo	Incidence	1	
		Leukaemia subtype – CML	C92	(Kaufman et al. 2009)	CaCo	Incidence	2	
			C92	(Cooke et al. 2010)	CaCo	Incidence		
		Liver cancer	C22	(Schuz et al. 2006)	Cohort	Incidence	1	
		Lung cancer	C33-34	(Schuz et al. 2006)	Cohort	Incidence	2	
			C33-34	(Benson et al. 2013)	Cohort	Incidence		
		Lymphomas (any type, adults)	C81-86	(Satta et al. 2012)	CaCo	Incidence	1	
		Hodgkin's lymphoma	C81	(Johansen et al. 2001)	Cohort	Incidence	1	
		Non-Hodgkin's lymphoma	C82-86	(Johansen et al. 2001)	Cohort	Incidence	5	
			C82-86	(Hardell et al. 2005)	CaCo	Incidence		
			C82-86	(Linet et al. 2006)	CaCo	Incidence		
			C82-86	(Satta et al. 2012)	CaCo	Incidence		
			C82-86	(Benson et al. 2013)	Cohort	Incidence		

SR subset	RF-Exposure Type, Source, Setting	Neoplasm	ICD-10 Code	Article	Design	Outcome measure	E-O Pairs	Total
		Lymphoma-subtype: B-cell	C83	(Hardell et al. 2005)	CaCo	Incidence	2	
			C83	(Satta et al. 2012)	CaCo	Incidence		
		Lymphoma-subtype: Diffuse	C83.9	(Linet et al. 2006)	CaCo	Incidence	1	
		Lymphoma-subtype: Diffuse Large B-cell	C83.3	(Satta et al. 2012)	CaCo	Incidence	1	
		Lymphoma-subtype: Follicular	C84	(Linet et al. 2006)	CaCo	Incidence	1	
		Lymphoma-subtype: Other specified	C84.7	(Linet et al. 2006)	CaCo	Incidence	1	
		Lymphoma-subtype: NOS	C85	(Linet et al. 2006)	CaCo	Incidence	1	
		Lymphoma-subtype: T-cell	C84, C86	(Hardell et al. 2005)	CaCo	Incidence	1	
		Lymphoma-subtype: T-cell, certain, e.g. cutaneous/leukaemia type	[C84, part]	(Hardell et al. 2005)	CaCo	Incidence	1	
		Melanoma	C43	(Benson et al. 2013)	Cohort	Incidence	1	
		Melanoma-eye (uveal melanoma)	C69-72	(Stang et al. 2009)	CaCo	Incidence	1	
		Melanoma-head & neck	C43	(Poulsen et al. 2013)	Cohort	Incidence	2	
			C43	(Hardell et al. 2011)	CaCo	Incidence		
		Melanoma-torso & legs	C43	(Poulsen et al. 2013)	Cohort	Incidence	1	
		Multiple myeloma	C90	(Benson et al. 2013)	Cohort	Incidence	1	
		Oesophagus cancer	C15	(Schuz et al. 2006)	Cohort	Incidence	2	
			C15	(Benson et al. 2013)	Cohort	Incidence		
		Oral Cavity/Pharynx cancer	C09-14	(Schuz et al. 2006)	Cohort	Incidence	1	
		Ovary cancer	C56	(Johansen et al. 2001)	Cohort	Incidence	2	
			C56	(Benson et al. 2013)	Cohort	Incidence		
		Pancreas cancer	C25	(Schuz et al. 2006)	Cohort	Incidence	2	
			C25	(Benson et al. 2013)	Cohort	Incidence		
		Prostate cancer	C61	(Schuz et al. 2006)	Cohort	Incidence	1	
		Rectum cancer	C20	(Johansen et al. 2001)	Cohort	Incidence	2	
			C20	(Benson et al. 2013)	Cohort	Incidence		
		Squamous cell carcinoma-head	C44	(Poulsen et al. 2013)	Cohort	Incidence	1	
		Squamous cell carcinoma-torso & legs	C44	(Poulsen et al. 2013)	Cohort	Incidence	1	
		Stomach cancer	C16	(Schuz et al. 2006)	Cohort	Incidence	2	
			C16	(Benson et al. 2013)	Cohort	Incidence		
		Testicular cancer	C62	(Schuz et al. 2006)	Cohort	Incidence	2	
			C62	(Hardell et al. 2007)	CaCo	Incidence		
		Thyroid cancer	C73	(Johansen et al. 2001)	Cohort	Incidence	3	
			C73	(Benson et al. 2013)	Cohort	Incidence		
			C73	(Luo et al. 2019)	CaCo	Incidence		
		Corpus uteri cancer	C54	(Johansen et al. 2001)	Cohort	Incidence	1	
		Cervix-Uteri cancer	C53	(Schuz et al. 2006)	Cohort	Incidence	1	

(*continued on next page*)

Table 3 (*continued*)

SR subset	RF-Exposure Type, Source, Setting	Neoplasm	ICD-10 Code	Article	Design	Outcome measure	E-O Pairs	Total
SR1-A	Cordless phones	Leukaemias (any type)	C91-95	(Kaufman et al. 2009)	CaCo	Incidence	1	7
		Non-Hodgkin's Lymphoma	C82-86	(Hardell et al. 2005)	CaCo	Incidence	1	
		Lymphoma-subtype: B-cell	C83	(Hardell et al. 2005)	CaCo	Incidence	1	
		Lymphoma-subtype: T-cell	C84, C86	(Hardell et al. 2005)	CaCo	Incidence	1	
		Lymphoma-subtype: T-cell, certain, e.g. cutaneous/leukaemia types	[C84, part]	(Hardell et al. 2005)	CaCo	Incidence	1	
		Melanoma-head & neck	C43	(Hardell et al. 2011)	CaCo	Incidence	1	
		Testicular cancer	C62	(Hardell et al. 2007)	CaCo	Incidence	1	
SR1-B	Broadcast Transmitters	No study available		-	-	-	0	0
SR1-B	Base Stations	Lymphomas (any type, adults)	C81-86, C96	(Satta et al. 2018)	CaCo	Incidence	1	4
		Lymphoma-Subtype: B-cell	C83	(Satta et al. 2018)	CaCo	Incidence	1	
		Lymphoma-Subtype: Diffuse Large B-cell	C83.3	(Satta et al. 2018)	CaCo	Incidence	1	
		Leukaemia-Subtype: CLL	C91	(Satta et al. 2018)	CaCo	Incidence	1	
SR1-C	**Occupational exposures** Multiple sources (JEM)	Acoustic Neuroma	C72.4	(Schlehofer et al. 2007)	CaCo	Incidence	1	40
	Occupational exposure Radar (Belgian Military personnel, Men)	Bone/Connective Tissue/Skin/Breast cancer	C40-41, C43-44, C47, C49, C50	(Degrave et al. 2009)	Cohort	Mortality	2	
	Occupational exposure Radar (French Navy personnel, Men)		C40-41, C43-44, C47, C49, C50	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure RF exposed-JEM (Motorola, mostly Men)	Brain cancer	C71	(Morgan et al. 2000)	Cohort	Mortality	2	
	Occupational exposure Radar (US Navy Korean war, Men)		C71	(Groves et al. 2002)	Cohort	Mortality		
	Occupational exposure Radar (US Navy Korean war, Men)	Breast cancer	C71	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (Belgian Military personnel, Men)	Digestive Organs cancer	C15-26	(Degrave et al. 2009)	Cohort	Mortality	2	
	Occupational exposure Radar (French Navy personnel, Men)		C15-26	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure Radar (Belgian Military personnel, Men)	Eye/Brain/Nervous System cancer	C69-72	(Degrave et al. 2009)	Cohort	Mortality	2	

SR subset	RF-Exposure Type, Source, Setting	Neoplasm	ICD-10 Code	Article	Design	Outcome measure	E-O Pairs	Total
	Occupational exposure Radar (French Navy personnel, Men)		C69-72	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure Radar (Belgian Military personnel, Men)	Genitourinary Organs cancer	C51-68	(Degrave et al. 2009)	Cohort	Mortality	2	
	Occupational exposure Radar (French Navy personnel, Men)		C51-68	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposures TETRA radio (recorded use)	Head & Neck cancer (atypical definition)	C00–14, C30, C32, C69-C73	(Gao et al. 2019)	Cohort	Incidence	1	
	Occupational exposure RF exposed-JEM (Motorola, mostly Men)	Hodgkin's lymphoma	C81	(Morgan et al. 2000)	Cohort	Mortality	1	
	Occupational exposure RF exposed-JEM (Motorola, mostly Men)	Leukaemias (any type)	C91-95	(Morgan et al. 2000)	Cohort	Mortality	2	
	Occupational exposure Radar (US Navy Korean war, Men)		C91-95	(Groves et al. 2002)	Cohort	Mortality		
	Occupational exposure Radar (US Navy Korean war, Men)	Leukaemia-Subtype-ALL	C91	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Leukaemia-Subtype-CLL	C92	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Leukaemia-Subtype-AML	C92	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Leukaemia-Subtype-CML	C92	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Non-lymphocytic leukaemia	C91	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Trachea, bronchus, and lung cancer	C33-34	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Lympho-Hematopoietic System Tumours (any)	C81-C96	(Groves et al. 2002)	Cohort	Mortality	4	
	Occupational exposure RF exposed-JEM (Motorola, mostly Men)		C81-C96	(Morgan et al. 2000)	Cohort	Mortality		
	Occupational exposure Radar (Belgian Military personnel, Men)		C81-C96	(Degrave et al. 2009)	Cohort	Mortality		
	Occupational exposure Radar (French Navy personnel, Men)		C81-C96	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure Radar (US Navy Korean war, Men)	Lymphoma and Multiple Myeloma	C81-86, C90	(Groves et al. 2002)	Cohort	Mortality	1	

(*continued on next page*)

Table 3 (*continued*)

SR subset	RF-Exposure Type, Source, Setting	Neoplasm	ICD-10 Code	Article	Design	Outcome measure	E-O Pairs	Total
	Occupational exposure RF exposed-JEM (Motorola, mostly Men)	Non-Hodgkin's Lymphoma	C82-86	(Morgan et al. 2000)	Cohort	Mortality	2	
	Occupational exposures Multiple sources (JEM)		C82-86	(Karipidis et al. 2007)	CaCo	Incidence		
	Occupational exposures Radar (work history + expert assessment, as in (Baumgardt-Elms et al. 2002))	Melanoma-eye (uveal melanoma)	C69	(Behrens et al. 2010)	CaCo	Incidence	1	
	Occupational exposures Multiple sources (JEM)	Meningioma	C70	(Vila et al. 2018)	CaCo	Incidence	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Oesophageal cancer	C15	(Groves et al. 2002)	Cohort	Mortality	1	
	Occupational exposure Radar (US Navy Korean war, Men)	Oral Cavity/Pharynx cancer	C09-14	(Groves et al. 2002)	Cohort	Mortality	3	
	Occupational exposure Radar (Belgian Military personnel, Men)		C09-14	(Degrave et al. 2009)	Cohort	Mortality		
	Occupational exposure Radar (French Navy personnel, Men)		C09-14	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure Radar (Belgian Military personnel, Men)	Respiratory/Intrathoracic Organs cancer	C30-39	(Degrave et al. 2009)	Cohort	Mortality	2	
	Occupational exposure Radar (French Navy personnel, Men)		C30-39	(Dabouis et al. 2016)	Cohort	Mortality		
	Occupational exposure Radar (US Navy Korean war, Men)	Testicular cancer	C62	(Groves et al. 2002)	Cohort	Mortality	3	
	Occupational exposures Radar (unspecified, jobs + expert assessment)		C62	(Baumgardt-Elms et al. 2002)	CaCo	Incidence		
	Occupational exposures Radar (unspecified, jobs + expert assessment)		C62	(Walschaerts et al. 2007)	CaCo	Incidence		
SR1-C	**Amateur Radio Operators**	Bladder cancer	C67	(Milham 1988)	Cohort	Mortality	1	21
		Brain cancer	C71				1	
		Kidney cancer	C64-66				1	
		Large Intestine cancer	C18-21				1	
		Lympho-Hematopoietic System tumours (any)	C81-96				1	
		Leukaemias (Any type)	C91-95				1	
		Leukaemia subtype – ALL	C91				1	
		Leukaemia subtype – CLL	C91				1	
		Leukaemia subtype – AML	C91				1	
		Leukaemia subtype – CML	C91				1	
		Leukaemia subtype – Monocytic	C93				1	
		Lymphosarcoma/reticulosarcoma	C82-83, C85.9				1	
		Hodgkin's disease	C81				1	
		Other tumours of lymphatic tissue	C81-C96				1	
		Liver cancer	C22				1	
		Oesophageal cancer	C15				1	
		Pancreas cancer	C25				1	
		Prostate cancer	C61				1	
		Rectum cancer	C20				1	
		Respiratory System cancer	C30-C34				1	
		Stomach cancer	C16				1	
		Total Important Outcomes				**143**		

CaCo = Case-control.
Cells highlighted in blue consist of studies included in *meta*-analyses.
Gender = When not specified (Men, Women), the study population include both male and female subjects.
ICD-10 code = International Classification of Disease, v 10; the ICD-10 code is shown also for studies reporting neoplasm codes in previous ICD versions.
Leukaemia subtypes: ALL = Acute Lymphocytic Leukaemia**; AML** = acute myeloid leukaemia**; CLL** = Chronic Lymphocytic Leukaemia**; CML** = Chronic Myeloid Leukaemia**.**
The exposure-outcome (E-O) pairs from studies of cohort design are more common than those from case-control studies, both overall (101 out of 143 E-O pairs, 70%), and especially in the occupational exposure (SR-C) subset (56 out of 62 E-O pairs, 90%).

for title/abstract screening. The title/abstract screening excluded 1,393 records, leaving 639 articles for full-text screening. Finally, the full-text screening excluded 492 articles, leaving 147 articles for inclusion in our systematic review.

In total 3,764 records were excluded, comprising retracted articles (n = 5), studies of irrelevant topics (n = 3,319), ineligible publication types (n = 250), studies of ineligible design (n = 95), plus 93 articles reporting on studies not compliant with our additional predefined inclusion criteria. The list of studies from the latter group, with reasons for exclusion, is provided in Annex 4, Table S1. Note that Table S1 consists of 96 records; 93 of these relate to the excluded articles, while 3 records are exposure-specific data not meeting our inclusion criteria in SR-B and/or SR-C from two studies included in SR-A (Baldi et al., 2011; Spinelli et al., 2010).

Several articles were excluded because they presented findings included in previous publications (meeting our definition of "duplicate

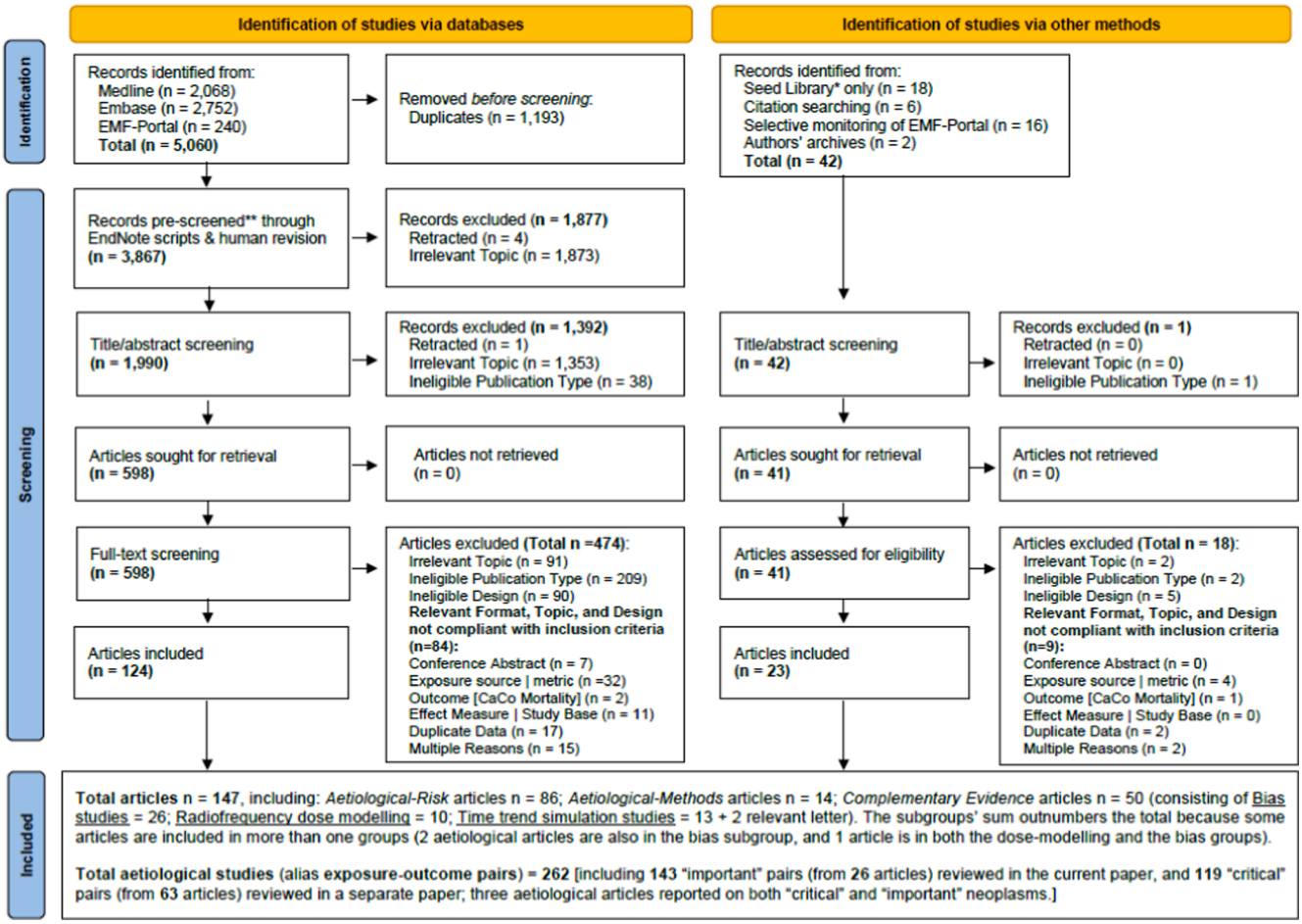

Fig. 1. PRISMA-2020 flow-diagram. Exposure source/metric = the excluded article deals with an ineligible exposure source (e.g., medical exposure) or report analyses based on ineligible exposure metric (e.g., only analysis per unit increase in mobile phone amount of use). **Effect measure / study base** = the excluded article reports ineligible measure of effect (e.g., survival; prevalence-OR, mortality-OR), or the study base is unidentifiable (that is, the reported RR is by default unreliable estimate of the effect of exposure).

data", n = 19), the study base was not identifiable (n = 11), the measure of outcome occurrence was cause-specific mortality (n = 3), or due to the publication type (conference abstracts, n = 7, all identified through Embase). Many articles were excluded due to ineligible exposure assessment methods, ineligible exposure metrics, or because exposures to RF and other types of EMFs were not discernible (n = 36 in total); the exposure-related exclusions were particularly common among articles potentially eligible for inclusion in SR-C.

4.1.2. Total included articles

In total, independent of the type of outcome (*critical* or *important*) and the exposure source/setting (SR-A, SR-B, SR-C), we considered eligible for inclusion 147 articles.

Of these articles, 86 reported on 262 distinct aetiological studies, *alias* E-O pairs, investigating the association between RF-EMF exposure from wireless phone use, fixed-site transmitters, or workplace sources and either "*important*" outcomes (26 articles, and 143 E-O pairs) addressed herein, or "*critical*" outcomes (63 articles, and 119 E-O pairs) which are the subject of a separate paper (Karipidis et al., 2024).

We identified and included 14 articles reporting on methodological aspects of a number of included studies (SR-A = 11 articles, and SR-C = 3 articles; see Annex 4 – Table S2). Additionally, we included 50 articles in the "Complementary Evidence" dataset used to support this review, dealing with topic-relevant bias studies (n = 26); RF-dose modelling (n = 10); and simulation studies of glioma incidence rate time trends (n = 13) which are only relevant for our analysis on "*critical*" outcomes (Karipidis et al., 2024); these articles are listed in Annex 4 – Tables S3-S5.

Please note that the detailed figures per group outnumber the total included articles because some articles reported on more than one topic or E-O pair: two articles were assigned to both the aetiological and bias-studies groups (Momoli et al., 2017; Turner et al., 2016); one article was included in both the dose-modelling and the bias-studies groups (Calderon et al., 2022); and three articles reported on studies investigating *critical* and *important* neoplasms (Schlehofer et al., 2007; Schuz et al., 2006; Vila et al., 2018).

4.1.3. Included studies of important outcomes

The 143 E-O pairs from the 26 aetiological articles reporting on "important" outcomes are shown in Table 3.

In SR-A, there were 71 studies investigating risks of 45 neoplasms in relation to mobile phone use, the majority being lymphohematopoietic system tumours (leukaemia/leukaemia subtypes and lymphoma/lymphoma subtypes, especially non-Hodgkin's lymphoma). Thyroid cancer was investigated in three studies. For other neoplasms there were only one or two studies per pair. There were also seven studies investigating cordless phone use and mainly different lymphohematopoietic system tumours.

In SR-B, there were no studies investigating possible risk for *important* tumours following RF exposure from broadcast transmitters, and four E-O pairs from a single article (Satta et al., 2018) reporting on risks of all lymphomas, two lymphoma subtypes, and chronic lymphatic

leukaemia in adults, in relation to RF exposure from mobile phone base stations.

In SR-C, there were 41 studies investigating the association between occupational RF exposure, mainly in military personnel (but some in other industries), and 26 neoplasms, the majority being again lympho-hematopoietic system tumours. Other neoplasms investigated in a few studies included oral cavity/pharynx cancer and testicular cancer (3 studies each). There was only one cohort of amateur radio operators (Milham 1988), reporting on risk of 21 neoplasms.

Among the included 143 studies, we identified 19 studies that satisfied inclusion for *meta*-analyses of homogenous datasets in terms of exposure type/metric and type of neoplasm (Table 3). Regarding RF-EMF exposure from mobile phone use (SR-A), these included studies of the exposure metric "ever (regular) use" and risk of leukaemia (4 studies), non-Hodgkin's lymphoma (5 studies), and thyroid cancer (3 studies). For some of these studies (3 of leukemias, and 4 of non-Hodgkin's lymphoma) the metric "long-term (10 + years) use" also satisfied the criteria for *meta*-analyses. There were not sufficient studies of any specific neoplasms to perform dose–response *meta*-analyses either for CCT or CNC. There were also not enough studies to perform *meta*-analyses of any exposure metrics concerning cordless phone use and any specific neoplasms.

The four E-O pairs investigating the effect of RF-EMF exposure from mobile phone base stations (SR-B) evaluated the effect on different neoplasms, and were not independent because reported in a single paper (Satta et al., 2018), so no *meta*-analyses could be conducted.

For occupational exposure to RF-EMF (SR-C), two *meta*-analyses were feasible, that included studies comparing risk of lymphohematopoietic system tumours (4 studies), or risk of oral cavity/pharynx cancer (3 studies), in exposed *vs* not exposed workers.

4.2. Study characteristics

Detailed information about the main characteristics of all included studies is provided in Annex 5**,** Tables S**6.1 to S6.5** (Study Key-Features tables).

Table 4
Heat map illustrating the risk of bias assessment results for studies included in evidence syntheses.

	Selection	Attrition	Exposure	Outcome	Healthy Worker Effect	Confounding	Selective reporting	Statistical methods	Summary bias tier
Mobile phone use and risk of leukaemia									
Schuz et al. 2006	(+)	(+)	(+)	(++)	NA	(+)	(++)	(++)	1
Kaufman et al. 2009	(-)	(++)	(--)	(+)	NA	(+)	(++)	(++)	2
Cooke et al. 2010	(+)	(-)	(+)	(++)	NA	(++)	(++)	(++)	2
Benson et al. 2013	(+)	(+)	(+)	(++)	NA	(+)	(+)	(++)	1
Mobile phone use and risk of non-Hodkin's lymphoma									
Johansen et al. 2001	(+)	(+)	(+)	(++)	NA	(+)	(++)	(++)	1
Hardell et al. 2005	(+)	(+)	(--)	(++)	NA	(+)	(++)	(++)	2
Linet et al. 2006	(+)	(-)	(-)	(++)	NA	(+)	(++)	(++)	2
Satta et al. 2012	(+)	(+)	(-)	(++)	NA	(+)	(++)	(++)	2
Benson et al. 2013	(+)	(+)	(+)	(++)	NA	(+)	(+)	(++)	1
Mobile phone use and risk of thyroid cancer									
Johansen et al. 2001	(+)	(+)	(+)	(++)	NA	(+)	(++)	(++)	1
Benson et al. 2013	(+)	(+)	(+)	(++)	NA	(+)	(+)	(++)	1
Luo et al. 2019	(+)	(+)	(-)	(++)	NA	(+)	(++)	(++)	2
Occupational RF exposure and risk of lymphohematopoietic system tumours									
Groves et al. 2002	(+)	(+)	(--)	(--)	(--)	(-)	(++)	(++)	2
Morgan et al. 2000	(++)	(+)	(--)	(--)	(--)	(-)	(+)	(++)	2
Degrave et al. 2009	(+)	(--)	(--)	(--)	(+)	(+)	(++)	(++)	2
Dabouis et al. 2016	(++)	(-)	(--)	(--)	(+)	(-)	(++)	(++)	2
Occupational RF exposure and risk of oral cavity/pharynx cancer									
Groves et al. 2002	(+)	(+)	(--)	(--)	(--)	(-)	(++)	(++)	2
Degrave et al. 2009	(+)	(--)	(--)	(-)	(+)	(+)	(++)	(++)	2
Dabouis et al. 2016	(++)	(-)	(--)	(--)	(+)	(-)	(++)	(++)	2

(++) = Definitely Low; (+) = Probably Low; (−) = Probably High; (−-) = Definitely High; NA = Not applicable.

Table 5
Evidence profile.

Certainty assessment									**Summary of findings**		**Final ConfidenceRating** High (++++) Moderate (+++) Low (++) Very Low (+)
Initial Confidence for Each Body of Evidence (Number of studies by design) **Moderate (+++)**	**Factors decreasing confidence** (" –" if no or low concern; "↓" if seriousconcern to downgrade confidence)					**Factors increasing confidence** (" –" if not present; "↑" if sufficientto upgrade confidence)					
	Risk of Bias	Inconsistency*	Indirectness	Imprecision	Publication Bias	Large Magnitude	Dose-Response	Confounding	No. of exposed cases	mRR (95 % CI)	
Outcome: Lymphohematopoietic system tumours											
Near-field, head localized, exposure from **mobile phones** (SR-A) and risk of **leukaemia**											
Ever *vs* Never use (2 Coh and 2 CaCo)	–	↓†	–	–	–	–	–	–	1,538	0.99 (0.91 to 1.07)	Low
Long-term (10 + years) use (2 Coh and 1 CaCo)	–	↓†	–	–	–	–	–	–	260	1.03 (0.85 to 1.24)	
Near-field, head localized, exposure from **mobile phones** (SR-A) and risk of **non-Hodgkin's lymphoma**											
Ever *vs* Never use (2 Coh and 3 CaCo)	–		–	–	–	–	–	–	2,179	0.99 (0.92 to 1.06)	Low
Long-term (10 + years) use (1 Coh and 3 CaCo)	–	↓†	–	–	–	–	–	–	295	0.99 (0.86 to 1.15)	
Near field/far-field **occupational exposure** (SR-C) and risk of **lymphohematopoietic system tumours**											
Exposed *vs* Unexposed (4 Coh)	–	↓†	–	↓††	–	–	–	–	215	1.05 (0.87 to 1.28)	Very low
Outcome: Thyroid cancer											
Near-field, head localized, exposure from **mobile phones** (SR-A) and risk of **thyroid cancer**											
Ever *vs* Never use (2 Coh and 1 CaCo)	–	↓†	–	–	–	–	–	–	1,040	1.05 (0.88 to 1.26)	Low
Outcome: Oral cavity/pharynx cancer											
Near field/far-field occupational exposure (SR-C) and oral cavity/pharynx cancer											
Exposed *vs* Unexposed (3 Coh)	–	↓†	–	↓††	–	–	–	–	34	0.68 (0.42 to 1.11)	Very low

Coh = Cohort study; **CaCo** = Case-control study; **mRR** = *meta*-estimate of the relative risk; **CI** = confidence interval of the mRR.
* = The OHAT GRADE-based method (NTP-OHAT 2019) considers the evidence provided by epidemiological studies as directly relevant to the assessment of human health hazards; therefore, we did not downgrade for indirectness.
† = Large confidence interval of the I^2; downgraded by one level.
†† =Two studies had measures of effect with upper to lower confidence limit ratios greater than 10; downgraded by one level.

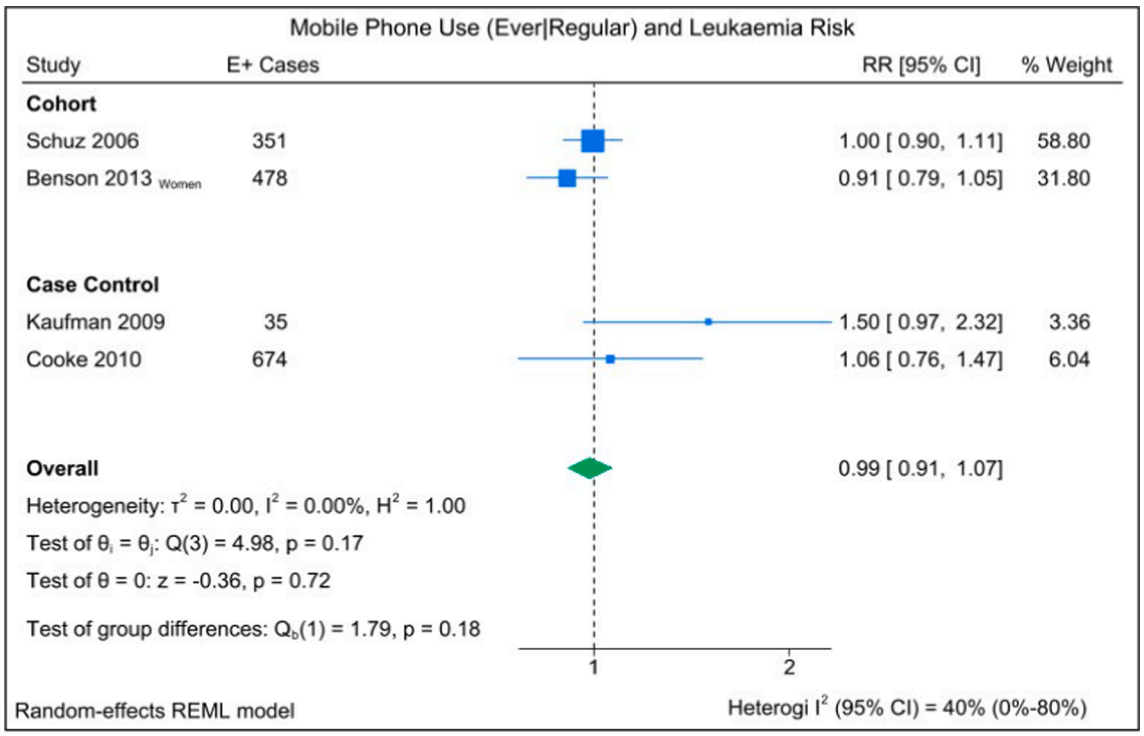

Fig. 2. Meta-analysis of mobile phone use (Ever or Regular) and leukaemia. Fig. 2 Footnotes: We combined using IVWA fixed effects models the measures of effects reported separately for men and women by Schuz et al., (2006). The studies were comparable in terms of leukaemia subtypes, with the exception of Cooke et al., 2010 who did not include chronic lymphocytic leukaemia.

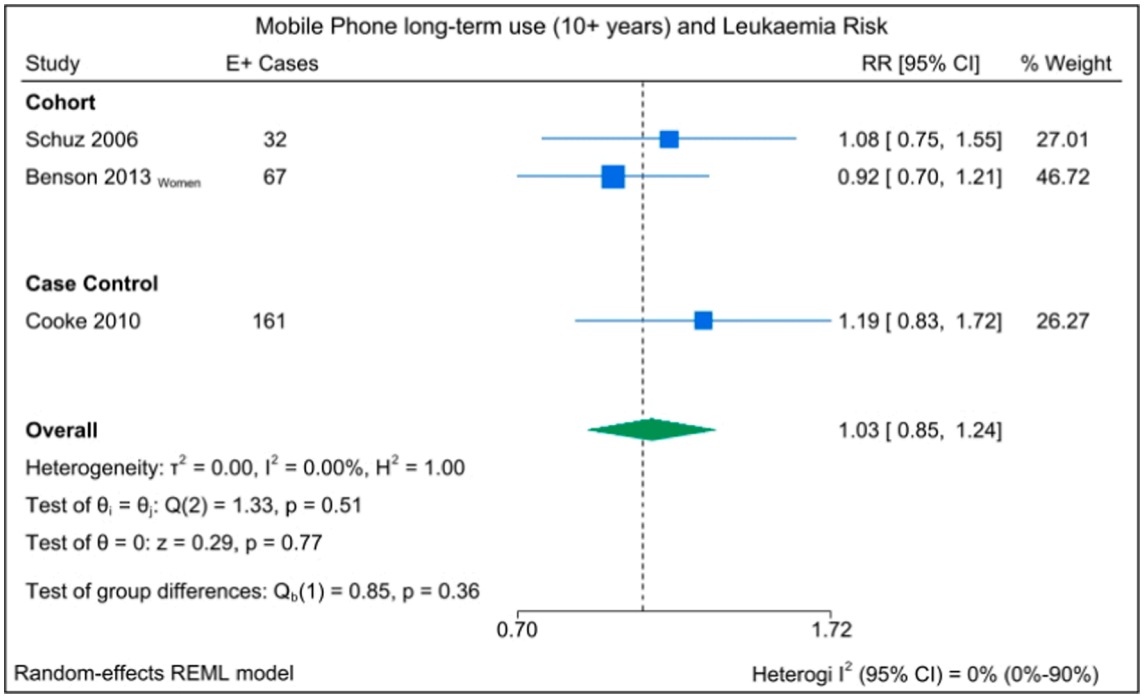

Fig. 3. Meta-analysis of long term (10 + years) mobile phone use and leukaemia. Fig. 3 Footnotes: We combined using IVWA fixed effects models the measures of effects reported separately for men and women by Schuz et al., (2006), as well as those for the time since start use categories "10-14 years" and "15 + years" reported separately by Cooke et al., 2010.

4.3. Results of the assessment of risk of bias

4.3.1. Risks of bias in studies

The RoB assessment forms for all examined studies are provided in Annex 6 where information on the rating rationale for each study can be found. Table 4 shows the heat map of the RoB assessments for the 19 studies included in the *meta*-analyses, consisting of homogenous datasets in terms of exposure source/type/metric and type of neoplasm, with at least three reported effect estimates. At the individual study level, the most critical issue was exposure characterization, especially for studies

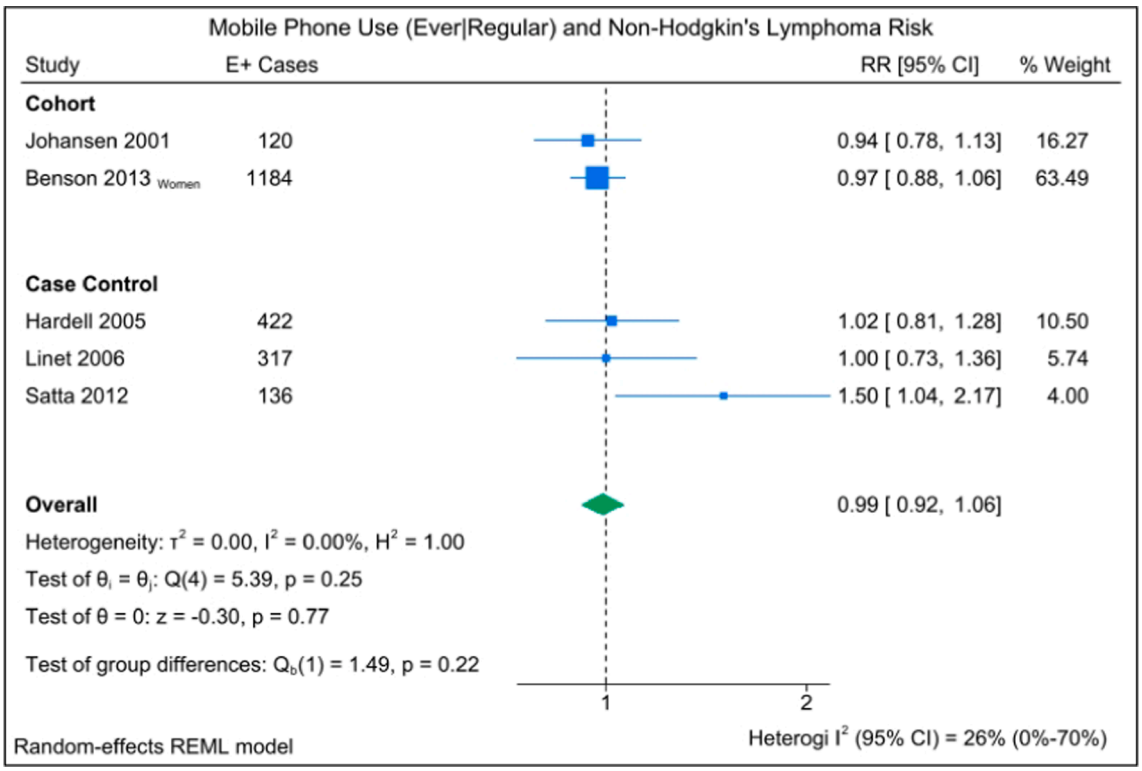

Fig. 4. Meta-analysis of mobile phone use (Ever or Regular) and non-Hodgkin's lymphoma. Fig. 4 footnotes: We combined using IVWA fixed effects models the measures of effects reported separately for men and women by Johansen et al., (2001). The measures of effect concerning Hardell et al., (2005) refer to the B-cell subtype of Non-Hodgkin's lymphoma (NHL, representing 90% of all NHL cases), because the data for all NHL cases were only reported in Table 5 for multiple combinations of wireless phone types and comprised an incongruous number of total exposed cases and controls; for B-cell NHL, to avoid double counting of individual data, we extracted from Table 2 the risk estimates based on the largest number of exposed cases (for ever use: digital phones).

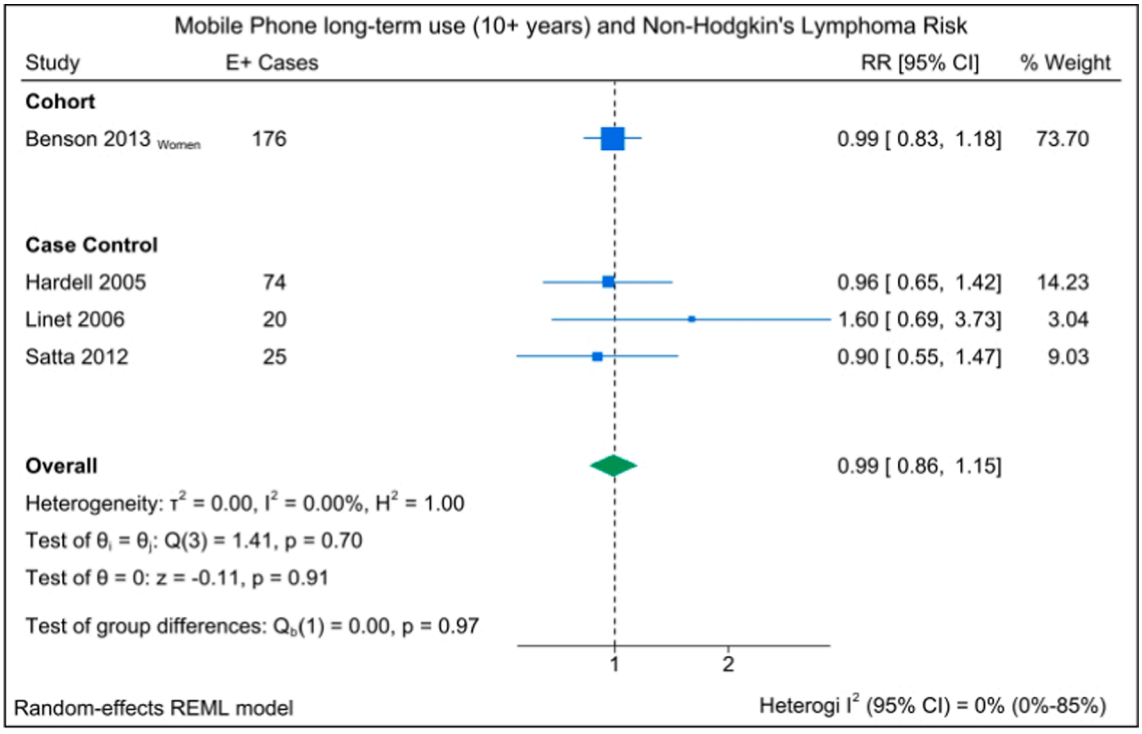

Fig. 5. Meta-analysis of long term (10 + years) mobile phone use and non-Hodgkin's lymphoma. Fig. 5 footnote: The measures of effect concerning Hardell et al., (2005) refer to the B-cell subtype of Non-Hodgkin's lymphoma (NHL, representing 90 % of all NHL cases), because the data for all NHL cases were only reported in Table 5 for multiple combinations of wireless phone types and comprised an incongruous number of total exposed cases and controls; for B-cell NHL, to avoid double counting of individual data, we extracted from Table 2 the risk estimates based on the largest number of exposed cases (for long-term use: analogue phones).

investigating occupational RF exposure where exposure characterization was rated at high risk of bias for all the included studies. Mortality-based occupational cohort studies were considered at high risk of outcome information bias. Confounding was also an issue and most occupational studies were rated as at high risk of this bias. Selective reporting and statistical methods were considered at low risk of bias in all studies.

4.3.2. Summary risk of bias (study tiering)

In the summary RoB assessment, focussed on predefined most relevant biases (i.e., selection/attrition, exposure and outcome information), the majority of studies were classified at moderate risk of bias (tier-2; n = 13, 68 %) and the rest were at low risk (tier-1; n = 6, 32 %); none of the studies were at high risk (tier-3) (Table 4, last column). Looking specifically at studies on mobile phone use, there was an equal number of studies that were classified at low risk (tier-1; n = 6, 50 %), all being cohort studies, and moderate risk (tier-2; n = 6, 50 %), all being case-control studies. The studies on occupational exposure were all rated at moderate risk of bias.

4.4. Effects of the exposure

4.4.1. Results of individual studies

The whole set of findings extracted from the included cohort and case-control studies is provided in Annex 7, Tables **S7.1 to S7.5**. (Summary of findings tables).

4.4.2. Data synthesis

4.4.2.1. SR-A – Mobile phone use and risk of leukaemia. a. Ever *vs* Never use of mobile phones and leukaemia risk.

The main *meta*-analysis of mobile phone use and risk of leukaemia stratified on design included data from two cohort and two case-control studies, with a total of 1,538 exposed cases (829 from cohort studies and 709 from case-control-studies) with available information on the exposure contrast "Ever or Regular" use *vs* "No use" (Fig. 2). The design-weighted *meta*-relative risk (mRR) was 0.99 (95 % CI = 0.91 – 1.07). The I^2 from the random effects REML model was 0 %, while that calculated using the heterogi module was 40 %, with wide 95 % confidence limits (0 %-80 %).

b. Time since start use (TSS) of mobile phones and leukaemia risk.

For the analyses by TSS use of mobile phones, there was sufficient data from three studies to conduct a *meta*-analysis for long term (10 + years) mobile phone use (Fig. 3). The *meta*-analysis included two cohort and one case-control study with a total of 260 exposed cases (99 from cohort studies and 161 from the case-control-study). The mRR was 1.03 (95 % CI = 0.85 – 1.24). The I^2 and related 95 % CI were 0 % (0 %-90 %).

c. Lifetime intensity of mobile phone use and leukaemia risk.

Only one case-control study reported on mobile phone CCT or CNC (Cooke et al., 2010), and no statistically significant increased risks of leukaemia were observed in the highest categories of either CCT (>1156 h, OR = 1.19, 95 % CI = 0.79 – 1.80), or CNC (>16,062 calls, OR = 1.03, 95 % CI = 0.68 – 1.56).

d Mobile phone use and risk of leukaemia sub-types.

The effect of mobile phone use on leukaemia sub-types, including acute lymphoblastic leukaemia (ALL), acute myeloblastic leukaemia (AML) and chronic myelogenous leukaemia (CML), was reported in only one or two studies per sub-type, so no *meta*-analyses were conducted. There were no statistically significant exposure-outcome associations in any metric-specific analyses, including ever *vs* never use, long term use, or cumulative intensity of use in any of the individual studies (see Annex 7, Table S7.1). Only one study, of lymphomas, reported also on risk of chronic lymphatic leukaemia (CLL) in relation to mobile phone use (Satta et al., 2012); the OR for ever *vs* never use was 1.8 (95 % CI 1.0–3.6), based on 36 exposed cases; this result was driven by findings in short-terms users (<5 years), while the OR in long-term users (10 + years) was 1.3 (95 % 0.4–3.8), based on 6 exposed cases.

4.4.2.2. SR-A – Mobile phone use and risk of non-Hodgkin's lymphoma. a. Ever *vs* Never use of mobile phones and non-Hodgkin's lymphoma risk.

The risk of non-Hodgkin's lymphoma in relation to ever *vs* never mobile phone use was investigated in two cohort studies including 1,304 exposed cases, and in three case-control studies with 875 exposed cases (Fig. 4). The overall mRR was 0.99 (95 % CI = 0.92 – 1.06). The I^2 from

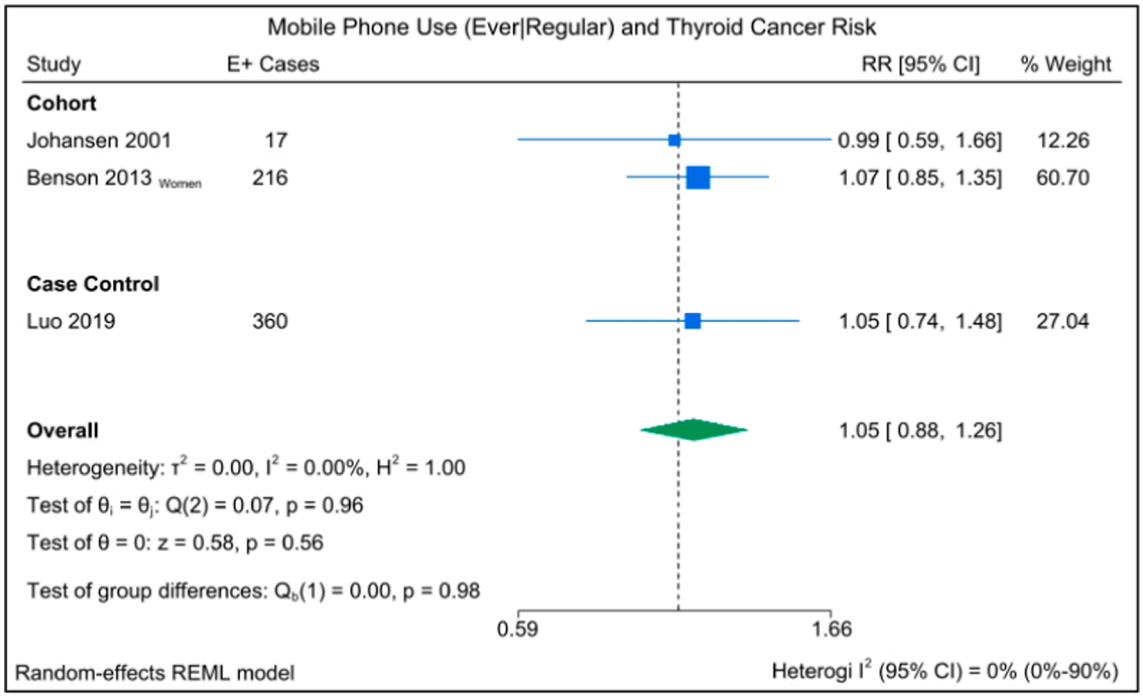

Fig. 6. Meta-analysis of mobile phone use (Ever or Regular) and thyroid cancer. Fig. 6 footnote: We combined the measures of effects reported separately for men and women by Johansen et al., (2001), using IVWA fixed effects models.

the random-effects REML model was 0 %, and that calculated using the heterogi module was 26 % with 95 % CLs = 0 %-70 %.

b. Time since start use (TSS) of mobile phones and non-Hodgkin's lymphoma.

For the analyses by TSS use of mobile phones, there was sufficient data from four studies to conduct a *meta*-analysis for long term (10 + years) mobile phone use (Fig. 5). The *meta*-analysis included one cohort and three case-control studies with a total of 295 exposed cases (176 from the cohort study and 119 from the case-control-studies). The mRR was 0.99 (95 % CI = 0.86 – 1.15). The I^2 was 0 %, with a wide 95 % confidence interval (0 %-85 %).

c. Lifetime intensity of mobile phone use and non-Hodgkin's lymphoma risk.

Only one case-control study reported on mobile phone CCT or CNC (Linet et al., 2006). No statistically significant increased risks of non-Hodgkin's lymphoma were observed in the highest categories of either CCT (>208 h, OR = 1.1, 95 % CI = 0.6 – 2.1), or CNC (>100 calls, OR = 0.9, 95 % CI = 0.6 – 1.4).

d Mobile phone use and risk of other lymphoma sub-types.

The effect of mobile phone use on other lymphoma sub-types, including Hodgkin's lymphoma, and histology-specific subgroups of non-Hodgkin's lymphoma (i.e., B-cell lymphoma, T-cell lymphoma, diffuse lymphoma, or follicular lymphoma), was reported in only one or two studies per tumour, so no *meta*-analyses were conducted. In individual studies there were generally no statistically significant associations between the outcomes and any exposure metric, including ever *vs* never use, long term use, or cumulative intensity of mobile phone use (see Annex 7, Table S7.1). The only statistically significant association, with a large confidence interval, was reported in one case-control study (Linet et al., 2006) for unspecified lymphoma in the TSS category "≥6 years" (OR = 3.2, 95 % CI = 1.2 – 8.4). Another case-control study (Satta et al., 2012) reported no statistically significant associations between ever (OR = 1.5, 95 % CI = 1.0 – 2.1) or long-term (OR = 0.9, 95 % CI = 0.6 – 1.6) mobile phone use and lymphomas overall, nor between mobile phone use (ever or long-term) and lymphoma subtypes (for the latter, see Annex 7, Table S7.1).

4.4.2.3. SR-A – Mobile phone use and risk of thyroid cancer. a. Ever *vs* Never use of mobile phones and thyroid cancer risk.

The risk of thyroid cancer in relation to ever *vs* never mobile phone use was investigated in two cohort studies (233 exposed cases), and one case-control study (360 exposed cases) (Fig. 6). The overall mRR was 1.05 (95 % CI = 0.88 – 1.26). Based on the I^2 point estimate (0 %), the heterogeneity across studies might not be important, but the I^2 95 % confidence interval was very wide (0 %-90 %).

b. Time since start use (TSS) of mobile phones and thyroid cancer.

Only two studies reported on long-term mobile phone use and thyroid cancer, so no *meta*-analyses were conducted. No association between 10 + years of mobile phone use and thyroid cancer (RR = 1.06, 95 % CI = 0.71 – 1.61) was observed in the Million Women cohort (Benson et al., 2013). Likewise, in the only available case-control study (Luo et al., 2019) no statistically significant associations between risk of thyroid cancer and long-term mobile phone use were reported, with ORs of 0.94 (95 % CI = 0.63 – 1.42) and 1.29 (95 % CI = 0.83 – 2.00) in the TSS categories of 12–15 years and > 15 years, respectively.

c. Lifetime intensity of mobile phone use and thyroid cancer risk.

Only one case-control study reported on mobile phone CNC or CCT (Luo et al., 2019); there was no statistically significant increase in the risk of thyroid cancer in the highest categories of either CNC (>32,850 calls, OR = 1.20, 95 % CI = 0.78 – 1.84) or CCT (>9,490 h, OR = 1.58, 95 % CI = 0.98 – 2.54).

4.4.2.4. SR-A – Mobile phone use and risk of other important neoplasms. The effect of mobile phone use on other *important* neoplasms was reported in only one or two studies per tumour, so no *meta*-analyses were conducted. There were generally no statistically significant associations with any exposure metrics, including ever *vs* never use, long term use or cumulative intensity of use in individual studies (see Annex 7, Table S7.1). Only one cohort study (Schuz et al., 2006), i.e., the second follow-up of the Danish mobile phone subscribers' cohort, showed an increase in kidney cancer risk among women (SIR = 1.42, 95 % CI = 1.02 – 1.92; 42 observed *vs* 37 expected cases) but not in men (SIR = 0.98, 95 % CI = 0.88 – 1.09; 366 observed *vs* 372 expected cases) ever using a mobile phone. However, another cohort study (Benson et al., 2013) investigating mobile phone use and kidney cancer in women did not find an association for either ever use (RR = 1.05, 95 % CI = 0.92 – 1.2; 584 exposed cases) or 10 + years of use (RR = 1.16, 95 % CI = 0.91 – 1.48; 92 exposed cases).

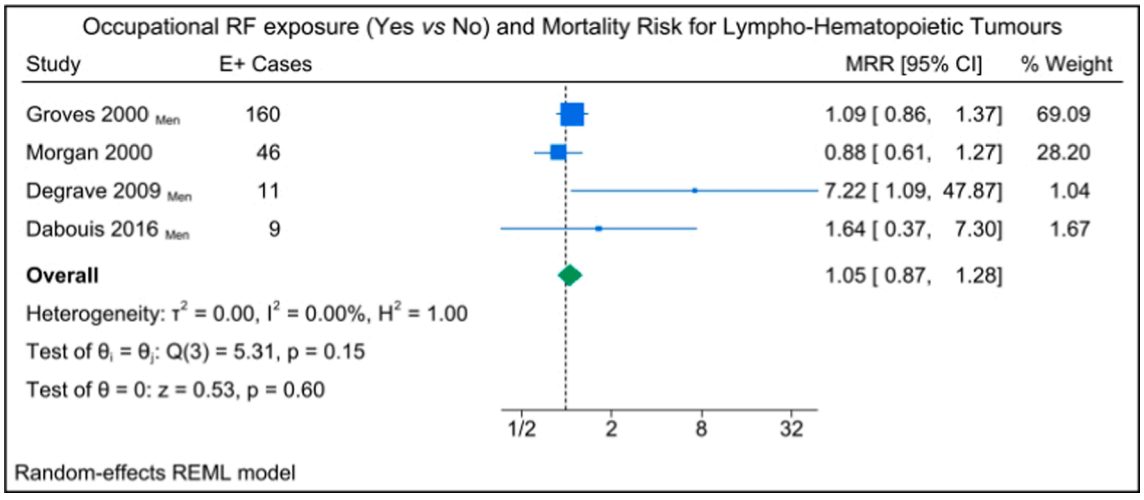

Fig. 7. Meta-analysis of occupational exposure (Exposed *vs* Not exposed) and risk of lymphohematopoietic system tumours. Fig. 7 footnotes: All studies are of cohort design, the investigated RF-exposure sources are radar in the military setting (Groves 2000; Degrave 2009; Dabouis 2016) or radiofrequency fields in the manufacture of wireless devices (Morgan 2000), and the reported findings are from analyses based on internal comparisons. For the study by Groves et al., (2000), the MRR estimates for all lymphohematopoietic system tumours (ICD-9 = 200–208) were calculated combining the estimates for all leukaemias (ICD-9 = 204–208) and for lymphoma and multiple myeloma (ICD-9 = 200–203) using IVWA fixed effects models; furthermore, in this study, the exposure contrast is high *vs* low potential exposure. To obtain the MRR estimates for "RF-exposure" from Morgan et al., (2000), we combined the original measures of effect for low, medium, and high exposure using IVWA fixed effects models.

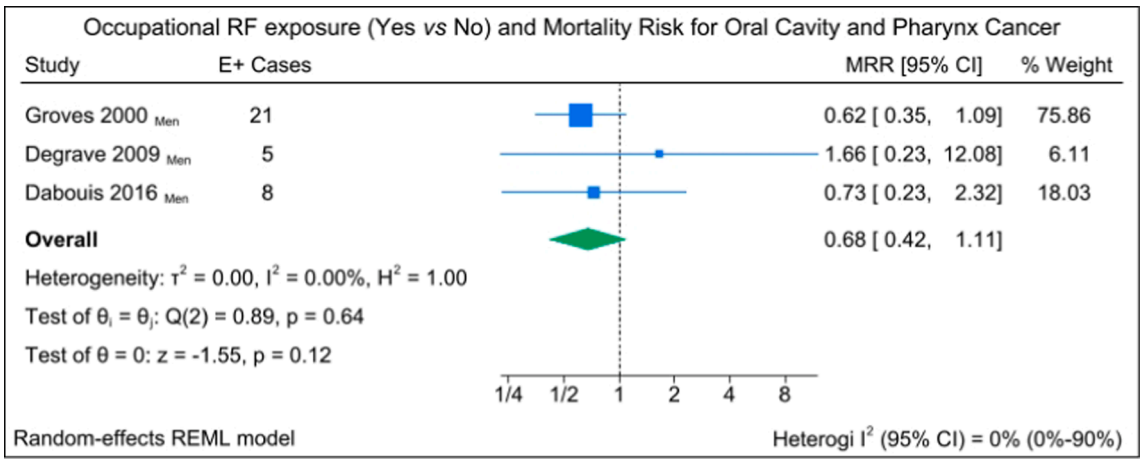

Fig. 8. Meta-analysis of occupational exposure (Exposed *vs* Not exposed) and risk of oral cavity/pharynx cancer. Fig. 8 **footnote**: All studies are of cohort design, the investigated RF-exposure source is radar, and the reported findings are from analyses based on internal comparisons. The exposure contrast extracted from Groves et al., (2000) is high *vs* low exposure potential.

There were also some sparse results showing statistically significant decreased risks for certain neoplasms, mainly from the second follow-up of the cohort of Danish subscribers (Schuz et al., 2006). In this study, decreased risks were observed for several smoking-related cancers among men, but not in women. The cancer sites for which decreased risks were observed included: lung cancer in men (SIR = 0.82, 95 % CI = 0.78 – 0.87) but not in women (SIR = 1.08, 95 % CI = 0.94 – 1.24); oral cavity/pharynx in men (SIR = 0.63, 95 % CI = 0.53 – 0.75) but not in women (SIR = 1.05, 95 % CI = 0.52 – 1.87); oesophageal cancer in men (SIR = 0.83, 95 % CI = 0.71 – 0.96) but not in women (SIR = 0.73, 95 % CI = 0.32 – 1.45); liver cancer in men (SIR = 0.8, 95 % CI = 0.65 – 0.97) but not in women (SIR = 0.43, 95 % CI = 0.11 – 1.09); pancreatic cancer in men (SIR = 0.86, 95 % CI 0.75–0.97), but not in women (SIR = 0.97, 95 % CI 0.68–1.35) (Schuz et al., 2006). These findings were very likely attributable to confounding from smoking via socio-economical differentials. Early mobile phone subscribers were better off than the general population in comparable age strata, and the authors checked the hypothesis that there were fewer smokers among male (but not female) subscribers. They examined the prevalence of smoking by increasing income (5 strata) in the prospective Danish cohort "Diet and Cancer", and found a clear inverse linear trend among men, but not among women (Schuz et al., 2006). In the Million Women cohort (Benson et al., 2013), a decreased risk in lung cancer was detected in women ever using a mobile phone (RR = 0.89, 95 % CI = 0.84 – 0.95), as well as in the category of 10 + years of use (RR = 0.88, 95 % CI = 0.78 – 1.0). The authors noted that phone users in the cohort were less likely than non-users to be current smokers at baseline, and it is possible that the slightly reduced risk of lung cancer reflects some residual confounding with smoking (Benson et al., 2013).

4.4.2.5. SR-A – Cordless phone use and risk of important neoplasms. The effect of cordless phone use was investigated in 7 studies, all on different neoplasms (mainly reporting on different types of lymphohematopoietic system tumours), so no *meta*-analyses were conducted. There were no statistically significant associations with any exposure metrics, including ever *vs* never use, long term use or cumulative intensity of use in any of the individual studies (see Annex 7, Table S7.2).

4.4.2.6. SR-B – RF exposure from fixed-site transmitters and risk of important neoplasms. Only one study (Satta et al., 2018) investigated the effect of environmental RF exposure from mobile phone base stations on risk of lymphoma (any type), and separately on three lymphoma sub-types (B-cell lymphoma, diffuse large B-cell lymphoma, and chronic lymphocytic leukaemia). There were no statistically significant associations between modelled RF levels and the lymphohematopoietic system tumours that were investigated (see Annex 7, Table S7.3). There were no included studies on cancer risk in relation to RF exposure from broadcast antennas or any other fixed-site transmitters.

4.4.2.7. SR-C – Occupational RF exposure and risk of lymphohematopoietic system tumours. We identified four mortality-based cohort studies of occupational exposure to RF-EMF and risk of lymphohematopoietic system tumours. One of the studies, which was on employees from a communications devices manufacturing company, used a JEM to estimate the individual exposure level (Morgan et al., 2000) and provided a risk estimate for low, medium or high exposure *vs* no exposure. The other three studies compared cause-specific mortality in military personnel working with radar *vs* personnel not working with radar and provided risk estimates for high *vs* low exposure (Groves et al., 2002) or exposed *vs* not exposed (Dabouis et al., 2016; Degrave et al., 2009). To synthesize the evidence, we combined the estimates for low, medium and high exposure from the Motorola worker cohort (Morgan et al., 2000) into a single estimate of exposed *vs* not exposed. We included in a quantitative synthesis the measures of effect for high *vs* low exposure from the Korean war navy cohort (Groves et al., 2002) together with the exposed *vs* not exposed estimates from the other two radar worker cohorts (Dabouis et al., 2016; Degrave et al., 2009) and the combined exposed *vs* not exposed estimate from the Motorola worker cohort (Morgan et al., 2000). The *meta*-analysis included 226 exposed cases and the mRR was 1.05 (95 % CI = 0.87 – 1.28), (Fig. 7). However, this result should be interpreted with caution, not only due to the differences in the exposure definition and level across studies, but especially because two of the measures of effect (Dabouis et al., 2016; Degrave et al., 2009) have very large confidence intervals, with upper to lower confidence limit ratios equal to44 (Degrave et al., 2009) and 20 (Dabouis et al., 2016).In addition, although the I^2 from the random effects REML model was 0 %, the value obtained using the Stata heterogi module was 44 % with a large confidence interval (0 %-81 %).

The effect of occupational exposure on different leukaemia and lymphoma sub-types was also reported but in only one or two studies per sub-type, so no *meta*-analyses were conducted. There were no statistically significant increased risks in relation to any exposure contrasts, including exposed *vs* not exposed, exposure level or duration of exposure in any of the individual studies (see Annex 7, Table S7.4). One study (Morgan et al., 2000) reported a statistically significant decreased risk of non-Hodgkin's lymphoma in communications devices manufacturing employees, but only in the shortest exposure duration stratum of < 5 years (MRR = 0.14, 95 % CI 0.02–0.43), and the finding was based on 1

exposed case only.

4.4.2.8. SR-C – Occupational RF exposure and risk of oral cavity/pharynx cancer. There were three mortality-based cohort studies on military personnel investigating working with or near radar and risk of oral cavity/pharynx cancer. One of the studies (Groves et al., 2002) provided a risk estimate for high *vs* low exposure level, whereas the other two studies (Dabouis et al., 2016; Degrave et al., 2009) provided risk estimates for the contrast exposed *vs* not exposed. To synthesize the evidence, we included in a quantitative synthesis the measures of effect for high *vs* low exposure from the Korean war navy cohort (Groves et al., 2002) together with the exposed *vs* not exposed RR estimates from the other two radar worker cohorts (Dabouis et al., 2016; Degrave et al., 2009). The *meta*-analysis included only 34 exposed cases; the mRR was 0.68 (95 % CI = 0.42 – 1.11); two out of three RR estimates were imprecise, with upper to lower confidence limit ratios > 10 (Dabouis et al., 2016; Degrave et al., 2009); and the I^2 had an extremely large confidence interval (I^2 95 % CI = 0 %-90 %) (Fig. 8). Again, this result should be interpreted with caution, due to the small number of studies, the few exposed cases, and the differences in the exposure level definition across studies.

4.4.2.9. SR-C – Occupational RF exposure and risk of other important neoplasms. The effect of occupational RF exposure on other *important* neoplasms was reported in only one or two studies per E-O pair, so no *meta*-analyses were conducted. Three reported effect estimates testicular cancer were available, from two case-control studies (Baumgardt-Elms et al., 2002; Walschaerts et al., 2007) and one cohort study (Groves et al., 2002); the case-control studies were incidence-based, while the cohort study was mortality-based, so these results were not combined in a *meta*-analysis. However, there were no statistically significant increased risks of testicular cancer in relation to any exposure metrics, including exposed *vs* not exposed, exposure level or duration of exposure, in any of the individual studies (see Annex 7, Table S7.4). One study (Groves et al., 2002) reported a statistically significant decreased risk of trachea, bronchus and lung cancer for military personnel exposed to high *vs* low RF levels (MRR = 0.73, 95 % CI = 0.63 – 0.83). In the INTEROCC case-control study (Vila et al., 2018), a statistically significant decreased risk of meningioma was observed in relation to cumulative exposure to RF-electric fields, but only in the analysis stratified on exposure duration, and only in the 5–9 year of exposure duration category (OR = 0.60, 95 % CI 0.38–0.97).

4.4.2.10. SR-C – Amateur radio operators and risk of other important neoplasms. Only one mortality-based cohort study (Milham 1988) investigated the risk of numerous types of neoplasms, especially different types of lymphohematopoietic system tumours, among amateur radio operators (see Annex 7, Table S7.5). There were no statistically significant associations, apart from an increased risk for "other lymphatic tissue cancers", which includes multiple myeloma and non-Hodgkin's lymphoma (SMR = 1.62, 95 % CI = 1.17—–2.18), and a decreased risk of pancreatic cancer (SMR = 0.64, 95 % CI = 0.42 – 0.94).

4.4.3. Assessment of reporting bias

There was no evidence of publication/small study bias in any of the investigated exposure-outcome combinations (Annex 8, Figs. S1.1 to S1.7), but the assessment was hampered by the small number of studies.

4.5. Confidence in evidence assessment

The results of the confidence in evidence assessment are shown in an Evidence Profile in Table 5. The considerations that emerged from the assessment are presented in the Discussion (section 5.1).

5. Discussion

5.1. Summary of the evidence and interpretation of the results

We performed an extensive systematic review of human observational studies investigating neoplasia risks in relation to three types of RF exposure: near-field, head-localized, exposure from wireless phone use (SR-A); far-field, whole body, environmental exposure from fixed-site transmitters (SR-B); near/far-field occupational exposures from use of hand-held transceivers or RF-emitting equipment in the workplace (SR-C). While no restrictions on tumour type were applied, this paper is part II of our review, focussed on less researched (what we termed "*important*") neoplasms. In a companion paper (part I), we have reviewed incidence-based studies of the most researched (what we termed "*critical*") neoplasms, including central nervous system and salivary gland tumours for SR-A and brain tumours and leukaemias for SR-B and SR-C (Karipidis et al., 2024).

The current review on *important* neoplasms included 26 aetiological articles, published between 1988 and 2019, with participants from 10 countries, investigating 143 different exposure-outcome pairs (comprising 65 different neoplasms). More than half of all studies (55 % of E-O pairs) addressed the association between wireless (mainly mobile) phone use and various neoplasms, mainly different types of lymphohematopoietic tumours (SR-A). Only four E-O pairs from a single case-control (Satta et al., 2018) examined the effect of exposure from fixed-sites transmitters on the risk of any lymphoma, three lymphoma subtypes, and also chronic lymphatic leukemia (SR-B). A large number of studies investigated occupational exposure (40 % of E-O pairs) and again mainly the risk of different types of lymphohematopoietic tumours, but also oral cavity/pharynx cancer (SR-C). The fourth review subset included 21 neoplasm-specific studies from a single article on cause-specific deaths in a cohort of amateur radio operators.

In line with our protocol, we performed the confidence in evidence assessment at the exposure-outcome level (Annex 3). Although there were 143 exposure-outcome pairs in total, only 19 of them, consisting of homogenous datasets in terms of exposure type/metric and neoplasm, with at least three reported effect estimates, could be included in *meta*-analyses and in the confidence of evidence assessment.

5.1.1. Lymphohematopoietic system tumours

RF-EMF exposure from mobile phones, measured as ever or regular use *vs* no or non-regular use, was not associated with risk of leukaemia (mRR = 0.99, 95 % CI 0.91–1.07; 4 studies and 1,538 exposed cases; Fig. 2). There was also no association between long-term (10 + years) use of mobile phones and risk of leukaemia (mRR = 1.03, 95 % CI 0.85, 1.24; 3 studies and 260 exposed cases; Fig. 3). Due to the to the small number of studies, the I^2 statistics had wide confidence intervals, and we downgraded 1-level the confidence in evidence for uncertainties in the assessment of heterogeneity. Therefore, the certainty in the observed absence of association between mobile phone use and leukaemia was rated as low.

RF-EMF exposure from mobile phones, measured as ever or regular use *vs* no or non-regular use, was not associated with risk of non-Hodgkin's lymphoma (mRR = 0.99, 95 % CI 0.92–1.06; 5 studies and 2,179 exposed cases; Fig. 4). There was also no association between long-term mobile phone use and risk of non-Hodgkin's lymphoma (mRR = 0.99, 95 % CI 0.86, 1.15; 4 studies and 295 exposed cases; Fig. 5). The result for long-term mobile phone use had a large confidence interval of the I^2, so a 1-level downgrade for inconsistency was applied. Therefore, the certainty in the observed absence of association between mobile phone use and non-Hodgkin's lymphoma was rated as low.

Risk of lymphohematopoietic system tumours following occupational RF-EMF exposure was not increased for the contrast exposed *vs* unexposed (mRR = 1.05, 95 % CI 0.87–1.28, 4 studies and 226 exposed cases; Fig. 7), with a large variation in the point estimates of the measures of effect, and a wide confidence interval of the I^2. Two studies had

measures of effect with upper to lower confidence limit ratios far greater than 10. We downgraded 2-levels, one each for inconsistency and for imprecision. Therefore, the confidence in the absence of association between occupational RF exposure and lymphohematopoietic system tumours was rated as very low.

5.1.2. Thyroid cancer

RF-EMF exposure from mobile phones, measured as ever or regular use *vs* no or non-regular use, was not associated with risk of thyroid cancer (mRR = 1.05, 95 % CI 0.88–1.26; 3 studies and 593 exposed cases; Fig. 6). The assessment of inconsistency in findings across studies was hampered by the small number of studies, reflected in the wide confidence limits of the I^2, and a 1-level downgrade was applied. Therefore, the certainty in the observed absence of association between mobile phone use and thyroid cancer was rated as low.

5.1.3. Oral cavity/pharynx cancer

Risk of oral cavity/pharynx cancer was not increased following occupational RF-EMF exposure for the contrast exposed *vs* unexposed (mRR = 0.68, 95 % CI 0.42–1.11; 3 studies and 34 exposed cases; Fig. 8). Due to the small number of studies and exposed cases, the I^2 had a large confidence interval (1-level downgrade for inconsistency). Two out of the three studies had measures of effect with upper to lower confidence limit ratios far greater than 10 (1-level downgrade for imprecision). Therefore, the certainty in the observed absence of association between mobile phone use and oral cavity/pharynx cancer was rated as very low.

5.1.4. Other important neoplasms

The effect of occupational RF exposure on testicular cancer was reported in three studies; however, two of the studies were incidence-based and one was mortality-based, so these results were not combined in a quantitative synthesis; the individual studies did not report any statistically significant increased risks of testicular cancer.

The effect of RF-EMF exposure from any of the investigated sources and settings on other *important* neoplasms was reported in only one or two studies per pair, so no evidence syntheses were conducted. However, it is noted that there were generally no statistically significant exposure-outcome associations, with a few studies reporting decreased risks among the exposed. The large majority of these decreased risks were observed in the second follow-up of the Danish subscribers' cohort among men but not in women, concerned smoking-related cancers, and were likely attributable to a "healthy subscriber effect" via a confounding effect of socioeconomic status and related differential prevalence of smoking in the male sub-cohort compared to the Danish general population of similar sex and age.

5.1.5. Risk of bias

There was limited variation in susceptibility to relevant biases in the dataset, with most studies classified in the tier-2 group, and no tier-3 studies. For studies on mobile phone use, the included cohort studies were all classified as tier-1, while the case-control studies were all classified as tier-2. There were no differences in results between cohort and case-control studies amenable to *meta*-analysis, implying that there were no differences in findings between tier-1 and tier-2 studies. All the included studies on occupational exposure were classified as tier-2.

5.1.6. Strengths of the systematic review

The major strengths of this systematic review are the transparency and reproducibility of the extensive protocol, the comprehensive literature search, the clear definition of inclusion and exclusion criteria, and the detailed RoB assessment.

5.1.7. Conclusive statements

Our conclusive statements, formulated in accordance with the GRADE guidelines 26 (Santesso et al., 2020), are provided below.

- For near field RF-EMF exposure to the head from mobile phones, there was low certainty of evidence that it does not increase the risk of leukaemia, non-Hodgkin's lymphoma or thyroid cancer.
- For occupational RF-EMF exposure, there was very low certainty of evidence that it does not increase the risk of lymphohematopoietic system tumours or risk of oral cavity/pharynx cancer.
- There was not sufficient evidence to assess whole-body far-field RF-EMF exposure from fixed-site transmitters (broadcasting antennas or base stations) or the effect of exposure to RF-EMF (from any source) on any other *important* neoplasms.

5.2. Limitations in the evidence

We believe that the study identification was complete, with little evidence that we missed major investigations. The funnel plots and the Egger tests did not generally detect publication bias.

The main limitation in this second paper on findings from our systematic review was the small number of studies per tumour type (which in some ways is inherent to this paper, that is dedicated to less researched neoplasms). A formal synthesis of the evidence was only possible for a few types of neoplasms, mainly different types of lymphohematopoietic system tumours, as well as thyroid and oral cavity/pharynx cancers. There were 54 types of neoplasms which were investigated in only one or two studies and did not satisfy the criteria for a quantitative synthesis of the evidence, but none of these showed evidence of an effect of RF EMF.

Further, although we had planned to perform secondary analyses, including cumulative *meta*-analyses and sensitivity analyses with various exclusions (Lagorio et al., 2021), there were not sufficient studies in the evidence base reviewed herein to conduct such analyses.

Looking at specific sources of RF-EMF exposure, the majority of the evidence was on mobile phone use. There was only sufficient evidence to assess the effect of ever (or regular) use *vs* no (or non-regular) use, as well as of long-term (10 + years) use. There was not sufficient evidence on lifetime intensity of mobile phone use, including cumulative call time and cumulative number of calls, so we could not conduct a dose–response analysis.

For occupational RF-EMF exposure there was sufficient evidence to assess the risk of lymphohematopoietic system tumours and oral cavity/pharynx cancer, and for these tumours the analyses were limited to the contrast of exposed *vs* not exposed workers. In these occupational studies the published data on risk by exposure duration or cumulative intensity was not sufficient, or not comparable in terms of metrics, to allow a quantitative synthesis. Another issue with the occupational studies included in the evidence syntheses is that the measure of disease occurrence was mortality, and given that lymphohematopoietic system tumours and oral cavity/pharynx cancer have relatively favourable survival rates (Jayasekara et al., 2010; Listl et al., 2013), various other factors such as therapeutic access can influence mortality occurrence.

All measures of effect included in the above-mentioned *meta*-analyses were mortality rate ratios (MRR), obtained from internal comparisons of cause specific death rates in the exposed and unexposed subcohorts. We did not consider any risk estimates based on comparison with the general population, and using SMR as the measure of effect. Therefore, the healthy worker hiring effect (HWHE) was not an issue. Moreover, all the risk estimates included in the *meta*-analyses were from cohort studies with long enrolment periods (usually 20 years), with the single exception of the US cohort of veterans serving in the Korean war (Groves et al., 2002), where inclusion in the study was based on a specific event. All cohorts had long follow-up periods: at least 20 years, and 40 years in the US cohort of veterans from the Korean war (Groves et al., 2002). Actually, the latter study was the single one reporting a decreased risk (of lung cancer) in the analyses based on internal comparison. This study was characterized by lack of information on the causes of death for about 300 subjects, but the missing data was equally distributed between the sub-cohorts of veterans at low and high exposure potential to

radar. Therefore, the decreased risk of lung cancer was more likely attributable to lack of control of confounding from smoking, rather than to the healthy worker survival effect (HWSE).

There was not sufficient evidence to assess the effect of RF-EMF from fixed-site transmitters (broadcasting antennas or base stations) on other *important* neoplasms, different from paediatric brain tumours and childhood leukaemia considered in the companion paper on critical outcomes (Karipidis et al., 2024).

In the RoB assessment performed at the individual study level, the most critical issue was exposure characterization, especially for studies investigating occupational RF exposure where exposure characterization was rated at high risk of bias for all the included studies. Outcome information bias was also an issue in the occupational cohort studies based on mortality data for non-rapidly fatal neoplasms such as lymphohematopoietic systems tumours and oral cavity/pharynx cancer. In addition, confounding was of concern and rated as high risk of bias for most of the occupational studies. Selective reporting and statistical methods were considered at low risk of bias in all the studies.

The reviewed bodies of evidence are likely affected by common limitations of epidemiological studies. Case-control study designs, with retrospective exposure assessment based on self-reported information, are inherently susceptible to any type of information bias (random misclassification, systematic errors, and differential errors), and to various sources of selection bias. Studies of cohort design are susceptible to attrition (Howe et al., 2016), and the occupational studies were all retrospective cohort studies with much of the information on exposure and outcome relying on records. Regarding the occupational studies of cohort design, we note that the most likely type of exposure measurement error was random misclassification, which results in the underestimation of potential true exposure-outcome associations, but only in loss of power with no bias under the null scenario. Most articles discuss such drawbacks in detail, and a few studies also estimated the impact of exposure measurement errors on the study findings through side validation studies (Schuz and Johansen 2007; Vergnaud et al., 2018).

Inadequate adjustment for confounding variables was an additional limitation in some studies. Most studies controlled for critical confounders (age, sex), but few studies had detailed and accurate information on socio-economic status, and exposure to occupational and lifestyle risk factors. However, residual confounding may not be a major issue because, except ionizing radiation, no strong risk factors for the investigated neoplasm are known. For further details on potential critical confounders see **Annex2, § III.1, pp. 40–42**. Uncontrolled confounding, especially from smoking habits and alcohol consumption, was a major limitation only in the occupational studies. The concern is mitigated, however, by the observed lack of associations between exposure to RF-EMF and the large majority of investigated neoplasms. Confounding from exposure to ionizing radiation, emitted from one type of radar (Hawk radars) before the technical improvements brought at the end of the 1970 s, is explicitly mentioned as a possible explanation of the increase risk of lymphohematopoietic system tumours observed in the Belgian cohort of military personnel (Degrave et al., 2009).

5.3. Limitations in the review process

Among the studies reviewed herein, we sought relevant information (such as such as number of exposed cases and controls, details on the control selection procedures, and response rates among controls by reason) missing from two papers (Hardell et al., 2011; Hardell et al., 2007). The authors responded to our emails, but did not provide the data.

Regarding the assessment of publication bias, we note that our syntheses included only a few studies and funnel plots and other approaches are less reliable when there are only a few studies (NTP-OHAT 2019). It has been recommended that tests for funnel plot asymmetry be used only when there are at least 10 studies included in the *meta*-analysis, because the power of tests is low (Harbord et al., 2009; Page et al., 2021a). Further, we note that interpretation of funnel plots and Egger's test was challenging, as it is difficult to identify whether an association between precision of a study and reported exposure/treatment effect is due to true heterogeneity, biases in individual studies, selective reporting, publication bias, or a combination of these (Hartwig et al., 2020; Sterne et al., 2011).

We share the opinion that the a-priori downgrading of human observational studies is the most challenging feature of evidence assessment methods adapted from clinical epidemiology, because the cohort or case-control designs may be the only feasible or ethical option to provide evidence on environmental health hazards (Arroyave et al., 2021; Krewski et al., 2022; Steenland et al., 2020).

The finalization of the current paper was a lengthy process (spanning 4 years, from the protocol drafting to the publication of results). A drawback common to this and other systematic reviews, is the risk of becoming obsolete already before being published.

Three further studies were published after the end-date of our literature searches (see § 6.3. below). The UK Biobank cohort study did not find an association between mobile phone use and a number of important neoplasms, including leukaemia, non-Hodgkin's lymphoma and thyroid cancer (Zhang et al., 2024). The study did report small associations with non-melanoma skin cancer, prostate cancer in men and vulva cancer in women; it is noted that mobile phone use in the study was only assessed at baseline, based on self-reports. A retrospective cohort study of Israel Defence Force service members who served in aerial defence units did not find an association between exposure to radar and haematological tumours or testicular cancer (Shapira et al., 2023). An international case-control study investigated the risks of glioma and meningioma in relation to occupational exposure to RF-EMF (Turuban et al., 2025). Cumulative and time-weighted average (TWA exposures were estimated based on lifetime job histories linked to the INTEROCC RF-JEM (Migault et al., 2019), using three methods: (1) by considering RF-EMF intensity among all exposed jobs, (2) by considering RF-EMF intensity among jobs with an exposure prevalence >/= the median exposure prevalence of all exposed jobs, and (3) by considering RF-EMF intensity of jobs of participants who reported RF-EMF source use. No clear exposure-outcome associations were identified. A few statistically significant associations were observed, including an increased risk of meningioma for cumulative exposure in the 5- to 9-year time window for electric fields (E) in the third JEM application method. The inclusion of this study would have introduced no changes in current review, because the study population coincides with that of the single included study of meningioma in RF-exposed workers (Vila et al., 2018). The difference concerns the exposure assessment method, which in the previously published article was based on a source-exposure matrix (Vila et al., 2016).

5.4. Implications of practice and policy

We did not observe an adverse effect of mobile phone use on the limited number of *important* neoplasms that satisfied the criteria for *meta*-analysis, neither overall, nor among long-term (10 + years) users. Most participants in the reviewed studies had used mobile phones operating on 1G-2G networks, and mobile phones of newer technology (3G-4G) have substantially lower average output power (Iyare et al., 2021; van Wel et al., 2021). For these reasons, the evidence assessed is informative regarding possible cancer risk from exposure levels higher than those experienced today. Notwithstanding the intrinsic limitations of the reviewed body of evidence, the exposure from mobile phones evaluated in the included studies is presumed to have been below the exposure limits of the current international RF exposure guidelines (ICNIRP 2020a). We also did not observe an adverse effect of occupational RF exposure on the limited number of *important* neoplasms amenable to a quantitative synthesis. Occupational exposure in the included studies is also presumed to have been below the exposure limits of the current international RF exposure guidelines. However, it is

important to note that the purpose of this systematic review was not to investigate the validity of the ICNIRP guidelines.

5.5. Implications for research

The exposure assessment is a critical issue in the body of evidence examined in this systematic review. Substantial improvements have been made in the ongoing COSMOS multicentre cohort study (Reedijk et al., 2023; Reedijk et al., 2024), which is expected to contribute valuable information on cancer risk in relation to mobile phone use. Actually, the first results on risks of CNS tumours (relevant to part I of our systematic review on *critical* outcomes) among over 250,000 members of the COSMOS cohort, with a long mobile phone use history already at baseline, and an average follow-up of about 7 years, have recently been published (Feychting et al., 2024). As it is unlikely that similar improvements may be introduced in studies relying on retrospective self-reported exposure information, further case-control studies on this topic are not recommended. Additional prospective cohort studies, similar to the COSMOS study, that pay particular attention to the assessment of exposure to assist in future dose–response analyses, have been recommended (ARPANSA 2017; SCENIHR 2015). Given that wireless communications have only recently started to use RF frequencies above 6 GHz, there are no epidemiological studies investigating 5G mobile networks directly as yet, but it is envisaged that future prospective cohort studies should cover this and other future planned technologies.

For occupational exposure new data is expected from the extended follow-up of the cohort of UK police officers (Airwave Health Monitoring Study) investigating possible health risks associated with the use of TETRA, a digital communication system used by police forces and other emergency services in the UK (Gao et al., 2019). Further on occupational exposure, a number of new job-exposure matrices (JEM) have been developed, e.g., the INTEROCC JEM (Migault et al., 2019) and CANJEM (Siemiatycki and Lavoue 2018), which provide much improved information on exposure assessment for many occupations. Future occupational studies should utilize these improved JEMs for assessing RF exposure.

We are aware of a single personal measurement survey of exposure from RF-emitting equipment in the workplaces (Turuban et al., 2023), followed by a comparison of the findings with those from a source-based job-exposure matrix (Turuban et al., 2024), and consider similar studies as an additional research priority.

Possible risk of bias remains an issue in epidemiological studies investigating RF-EMF and cancer. Well-designed side validation studies should be planned in any new epidemiological study (Fox and Lash 2017; Lash and Ahern 2012; Lash et al., 2009; Lash et al., 2016), and this is a high-priority issue for those investigating the exposure-outcome associations examined in the current review.

6. Other information

6.1. Registration and protocol

The protocol has been registered in PROSPERO (CRD42021236798), and published [(Lagorio et al., 2021), DOI https://doi.org/10.1016/j.envint.2021.106828].

6.2. Amendments to the protocol

There were five amendments to the published protocol (Lagorio et al., 2021):

1. Instead of updating the literature searches on all main databases (Medline, Embase and EMF-Portal), we carried out periodic searches of relevant studies on EMF-Portal only, because the precision [1-(excluded record / total retrieved)] of this topic-specific literature database was much greater than that of the other two sources (0.34 *vs* 0.05 for Medline, and 0.04 for Embase).
2. We assessed the risk of bias (RoB) using paper forms, and Excel to produce the related heat maps, because the envisaged management through the HAWK platform (Shapiro et al., 2018) proved unfeasible due to the complexity of our tailored question–answer forms.
3. For homogenous datasets (in terms of outcome, subjects' lifestage, and exposure type/metric), we did set a minimum size requirement for amenability to a *meta*-analysis (at least 3 reported effect estimates).
4. The synthesis of findings from the study subsets not meeting the requirements for inclusion in a *meta*-analysis was based on a structured tabulation of the key-features (Annex 5, Tables S6.1-S6.5) and results (Annex 7, Tables S7.1-S7.5) of the individual exposure- and outcome-specific studies. We did not prepare visual plots (Anzures-Cabrera and Higgins 2010; McKenzie and Brennan 2021) for studies that were not included in *meta*-analyses, contrary to what envisaged in our protocol.
5. The statements to convey findings from our systematic review were formulated in accordance with the wording suggested by the GRADE guidelines 26 (Santesso et al., 2020).

6.3. New relevant studies issued after the literature search end date

At the last selective monitoring of EMF-Portal, performed on 17 May 2024, we identified the following relevant articles, potentially or definitely eligible for inclusion in this second paper of our systematic review:

1. Studies (meeting our inclusion criteria)
   - Occupational Exposure to Nonionizing Radiation and Risk for Malignancy in Young Adults (Shapira et al., 2023)
   - Mobile Phone Use and Risks of Overall and 25 Site-Specific Cancers: A Prospective Study from the UK Biobank Study (Zhang et al., 2024).
   - Occupational exposure to radiofrequency electromagnetic fields and brain tumor risk: Application of the INTEROCC job-exposure matrix (Turuban et al., 2025).
2. Complementary evidence – RF dose modelling (meeting our inclusion criteria)
   - Modelling of daily radiofrequency electromagnetic field dose for a prospective adolescent cohort (Eeftens et al., 2023).
   - Dosimetric assessment in the brain for downlink EMF exposure in Korean mobile communication networks (Lee and Choi 2023).
   - Determining the relationship between mobile phone network signal strength and radiofrequency electromagnetic field exposure: protocol and pilot study to derive conversion functions [version 1; peer review: awaiting peer review] (Sandoval-Diez et al., 2024).
3. Complementary evidence – Exposure assessment (not meeting our inclusion criteria, but very relevant because to our knowledge these are the first personal measurement survey in the workplace, and the first validation of a RF-JEM using personal measurements)
   - Personal exposure to radiofrequency electromagnetic fields in various occupations in Spain and France (Turuban et al., 2023).
   - Comparison of a radiofrequency electric and magnetic field source-based job-exposure matrix with personal radiofrequency exposure measurements (Turuban et al., 2024).

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CRediT authorship contribution statement

Ken Karipidis: Writing – original draft, Project administration, Methodology, Investigation, Funding acquisition. **Dan Baaken:** Writing – review & editing, Project administration, Investigation, Data curation. **Tom Loney:** Writing – original draft, Methodology, Investigation. **Maria Blettner:** Writing – original draft, Methodology, Conceptualization. **Rohan Mate:** Writing – review & editing, Investigation, Data curation. **Chris Brzozek:** Writing – review & editing, Investigation, Data curation. **Mark Elwood:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Clement Narh:** Writing – review & editing, Investigation. **Nicola Orsini:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Martin Röösli:** Writing – review & editing, Methodology, Conceptualization. **Marilia Silva Paulo:** Writing – review & editing, Investigation, Data curation. **Susanna Lagorio:** Writing – original draft, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Mark Elwood has given expert advice on topics in electromagnetic fields and health, and on the objective interpretation of epidemiological and other scientific information, over many years to individuals and groups, including government ministries, environmental regulators, community groups, commercial organisations, and formal inquiries by government and professional groups including parliamentary and legal proceedings. Some of this work has been financially supported, by universities, health care organisations, research bodies, or by government, professional or commercial groups. Some work has been reported 'blind', with the client being unidentified. Susanna Lagorio was principal investigator (April 2019 – March 2020) of the research project "BRiC 2018/06 – Systematic reviews of exposure to radiofrequency fields and cancer", supported by the Italian Workers' Compensation Authority, a public no-profit entity (grant code I85B19000120005). Her employment duties involved provision of advice on health hazards from exposure to RF-EMF to the Italian Ministry of Health and Higher Health Council (she retired on August 1st, 2023). Martin Röösli's research is entirely funded by public entities or not for profit foundations. He has served as advisor on potential health effects of exposure to non-ionizing radiation to several national and international public advisory and research steering groups, including the World Health Organization, the International Agency for Research on Cancer, the International Commission on Non-Ionizing Radiation Protection, the Swiss Government (member of the working group "Mobile phone and radiation" and chair of the expert group BERENIS), the German Radiation Protection Commission (member of the committee Non-ionizing Radiation (A6) and member of the working group 5G (A630)) and the Independent Expert Group of the Swedish Radiation Safety Authority. From 2011 to 2018, M.R. was an unpaid member of the foundation board of the Swiss Research Foundation for Electricity and Mobile Communication, a non-profit research foundation at ETH Zurich. Neither industry nor nongovernmental organizations are represented on the scientific board of the foundation. Chris Brzozek and Rohan Mate as part of their employment are involved in the provision of advice to the Australian Commonwealth Government, Australian States and Territories and the general public on the risks and health effects of exposure to ionising and non-ionising radiation.

The other authors declare that they have no known conflicts of interest.

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Availability of other material

Data, analytic codes, or other materials will be made available upon request addressed to the corresponding author (ken.karipidis@arpansa.gov.au), specifying the intended use, and provided that the request is approved by the co-leaders (SL and MB) along with the other team members.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2025.109274.

Data availability

Data will be made available on request.

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