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Glyphosate and Glyphosate-Based Herbicides Cancer Hazard Assessment Protocol

Human Cancer Studies

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Integrative Health Assessments Branch, NIEHS, NIH, HHS

About this report

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Note

This protocol was originally intended for internal cancer hazard assessments to reach level-of-evidence conclusions of carcinogenicity from animal and human cancer studies. Currently, we are refocusing the assessment for journal publications that assess and integrate the evidence, as well as discussing strengths, limitations, and research gaps. Thus, the steps related to reaching level-of-evidence conclusions will not be used in the manuscripts.

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1. Evaluating Human Cancer Studies of Exposure to Glyphosate-Based Herbicides (GBH)

1.1. Introduction

Glyphosate is a synthetic compound and active ingredient in various products (e.g., glyphosate-based herbicides, [GBHs]) used as a nonselective systemic herbicide, particularly effective against perennial weeds, for commercial, residential, and agricultural purposes (ATSDR 2020). GBHs are the most widely used herbicide in the United States with substantial increases in total volume applied since the 1990s in large part because of its use on genetically engineered herbicide-tolerant crops (Benbrook 2016). Further increases in the early to mid-2000s, with use increasing more than 12 fold from 1995 to 2014, were due to the increase of its use in harvest aid and green burndown applications (Benbrook 2016). Since it was first registered as a pesticide (1975), over 1.6 billion kilograms of glyphosate active ingredient have been applied in the U.S., representing 19% of the estimated global use of glyphosate. As such, exposure to glyphosate is widespread, with >70% of U.S. National Health and Nutrition Examination Survey (NHANES) participants containing detectable concentrations of glyphosate from urinary biomonitoring sampling (Ospina et al. 2024).

The International Agency for Research on Cancer (IARC) classifies glyphosate as probably carcinogenic to humans (Group 2A) based on (1) sufficient evidence of carcinogenicity from studies in experimental animals and (2) limited evidence of carcinogenicity from studies in humans based on an association between non-Hodgkin lymphoma (NHL) and glyphosate exposure. Mechanistic and other relevant data support the IARC cancer hazard classification. In contrast, other organizations have reached different conclusions. The U.S. Environmental Protection Agency (US EPA), has concluded that glyphosate is "not likely carcinogenic to humans," (EPA 2020) and the European Chemicals Agency (ECHA) concluded that the available evidence did not meet EU criteria to classify glyphosate as carcinogenic (ECHA 2023) based on their assessments of cancer studies in humans and experimental animals.

To address the apparent discrepancy in hazard conclusions among authoritative bodies and from groups conducting meta-analyses, the Division of Translational Toxicology (DTT) is conducting a rapid systematic review of cancer studies, including (1) animal cancer studies on glyphosate and GBHs and (2) human epidemiological studies of lymphohematopoietic cancers and GBH exposure. DTT will use methods outlined in the Report on Carcinogens (RoC) Handbook (2025) and apply the RoC listing criteria to the cancer hazard assessment to reach a draft hazard conclusion for each of the evidence streams separately.

This protocol draws on the general methods outlined in the RoC Handbook and provides specific methods used to conduct the cancer hazard assessment of glyphosate and GBH and human lymphohematopoietic cancers, with a focus on NHL.

1.1.1. Background Information

Glyphosate is produced in the United States as a technical-grade substance, with purity usually above 90%, although products with purity above 80% are also available.

GBHs contain glyphosate concentrations ranging from 0.96% to 94%, and concentrations can vary substantially even within the same commercial product (ATSDR 2020).

The general public may be exposed to glyphosate through dermal contact with consumer products, crops, and contaminated soil; ingestion of plants, crops, and drinking water; and inhalation of mists during product use. The U.S. Food and Drug Administration's (FDA) crop residue testing has shown high detection of glyphosate in crop products like corn and soybeans (up to ~59% detection); however, the concentrations of glyphosate are generally below EPA pesticide tolerance limits (US FDA 2024). Children may also be exposed through hand-to-mouth activities and by playing in contaminated soil. The highest potential for exposure to glyphosate among agricultural workers occurs through dermal contact; however, workers may also be exposed via inhalation, oral ingestion, and ocular contact. Glyphosate is not expected to bioaccumulate in the environment (ATSDR 2020).

Glyphosate is rapidly absorbed from the gastrointestinal tract and likely from the respiratory tract, but relatively little is absorbed through the skin. However, based on in vitro studies, surfactants present in certain GBHs can potentially increase dermal absorption and facilitate absorption through the skin and across the cell membrane. Once absorbed, glyphosate is distributed to tissues via the bloodstream, with no substantial accumulation in any specific tissue. Glyphosate is excreted primarily in the urine and feces, mainly as the parent compound, with only a small percentage metabolized (ATSDR 2020). In humans, the half-life of glyphosate in urine is less than 10 hours; thus urinary measures represent levels concurrent with the timing of sample collection (Connolly et al. 2019). Urinary glyphosate levels are higher among workers applying GBHs than non-working populations and are higher among workers not using gloves (ATSDR 2020; Chang et al. 2024).

1.1.2. Objective and Aims

To reach conclusions about the level of evidence of the carcinogenicity of glyphosate provided by human cancer studies based on the RoC listing criteria.

The assessment will be captured in a report, which will be peer-reviewed by reviewers with relevant expertise.

Specific aims and key questions

1. To conduct a systematic review of lymphohematopoietic cancers outcomes and exposure to glyphosate-based herbicides (GBH).
2. What are the study characteristics and key issues to consider in the evaluation?
    - Which cancer outcomes have an adequate database for review?
    - How is GBH exposure characterized, measured, or quantified in the studies?
    - What are the key scientific issues?
3. How informative (e.g., risk of bias, study sensitivity) are the studies for evaluation?
    - What are the potential confounders and effect modifiers for cancer risk of the tumor sites of interest in these studies?
    - What are the potential biases, the impact of the biases, and the study sensitivity of the study's findings?

4. What is the level of evidence for carcinogenicity for each cancer outcome?
    - Is there a credible association between GBH and cancer across studies?
    - If so, can the relationship between each GBH and cancer be explained by chance, bias, or confounding?

1.1.3. Protocol Contents and Evaluation Process

This document describes the (1) completed scoping and problem formulation steps used to develop the framework (Section 1.2) and (2) proposed methods that will be used to conduct the cancer hazard evaluation, including the study evaluation (Section 1.4) and evidence integration (Section 1.5). Section 1.6 describes data extraction methods and reporting elements. The methods are based on applying the specific issues relevant to GBH exposure to the procedures outlined in the RoC handbook. The roles of the researchers and the literature search terms are described in Appendices A and B.

Figure 1 provides a schematic of how the protocol (Step 2) fits into the cancer hazard evaluation process. The protocol is informed by the scoping and problem formulation (i.e., developing the framework) done in Step 1, and the methods in the protocol are then used to conduct the cancer hazard evaluation and write the RoC monograph (Step 3). Note that Steps 1 and 2 are iterative.

Figure 1. Cancer Hazard Evaluation Process

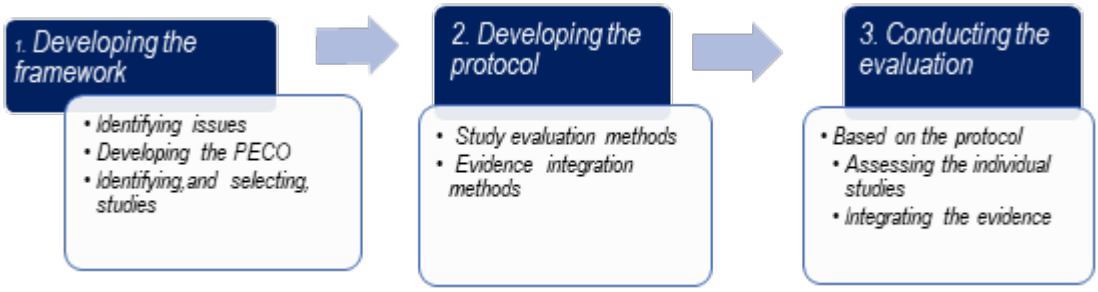

Figure 1 depicts the cancer hazard evaluation process. Scoping, problem formulation, and evidence mapping lead to the development of the framework (Step 1), which includes the overall objective and aims, PECO statements (i.e., body of evidence) to address the study objective(s), and identification of hazard specific issues to be explored in the evaluation. This step has been completed, and the findings are reported in Section 1.2. This step also informed the methods, i.e., the protocol (Step 2) for conducting the cancer hazard evaluation (Step 3), the results of which will be captured in the report. The methods focus on study evaluation (bias and study sensitivity, Section 1.3) and evidence integration (Section 1.4). PECO = Population, Exposure, Comparison group, and Outcome.

1.2. Developing the Framework

Preliminary scoping and problem formulation activities informed the evaluation framework for the evaluation of human cancer epidemiology studies using the methods described in this protocol.

These activities informed the research questions for human epidemiology studies (e.g., Specific Aim 1: overall objective) and the body of evidence to answer the research questions. For human

studies, the body of evidence is defined by the PECO (**P**opulation, **E**xposure, **C**omparison Group, **O**utcome) Statements.

Table 1. Population, Exposure, Comparison, and Outcome Statement (PECO)

Element	Criteria
Population	All human-only study populations (no restrictions)
Exposure	Glyphosate-based herbicides (GBH), including: glyphosate (active ingredient), glyphosate formulations (e.g., glyphosate-based herbicides), glyphosate metabolites (e.g., AMPA, MPA)
Comparison	No or low exposure to GBH
Outcome	Lymphohematopoietic (LHC) cancers – adult and childhood

Abbreviations: AMPA = Aminomethylphosphonic acid, MPA = Methylphosphonic acid

1.2.1. Identifying and Selecting the Literature

In consultation with an information specialist, we develop a literature search strategy based on the PECO (see Table 1). Biomedical citation databases, including PubMed, Scopus, and Web of Science, were searched for human cancer studies by combining search terms for exposure to glyphosate and GBH (see Appendix B), cancer (see RoC Search String Document), and human cancer studies (see RoC Search String Document) using the procedures outlined in the 2025 RoC Handbook. The search was not limited by language. Exposure specific search terms included terms specific for glyphosate (including trade names, e.g., RoundUp), synonyms, and metabolites of the glyphosate (e.g., AMPA). In addition, we searched references from authoritative and systematic reviews to identify human cancer studies reporting on glyphosate and GBH. We also conducted a full-text search a PDF library of occupational case-control studies for mentions of glyphosate, RoundUp, or AMPA to identify studies where the chemical name is not mentioned in the title, abstract, or keywords. Finally, we developed a new PDF library of human epidemiology studies of pesticide or herbicide manufacturers and conducted a full-text search of of the literature in this library that had available searchable full-text (approximately 30% of the full library).

Search results are processed in EndNote and imported into a content management system [e.g., Health Assessment Workplace Collaborative (HAWC)] software to select relevant literature (Shapiro et al. 2018). Studies are included in HAWC based on the PECO; additional studies were identified by targeted searches:

- Primary studies: Studies were included in the evaluation if they were primary studies (analytical epidemiologic studies or pooled analyses of multiple studies) reporting a risk estimate for LHC and meeting the PECO statement.
- Supporting studies: Studies providing supporting information for topics relevant to the evaluation of the human epidemiologic evidence. These include, but are not limited to, qualitative reviews or letters to the editor, and information on co-exposures or potential confounders.
- Reviews and meta-analyses: Meta-analyses, and systematic and narrative reviews for relevant topics.

1.2.2. Mapping the Evidence

Citations in HAWC were tagged as described above by cancer type (lymphohematopoietic cancer (LHC) vs other cancer) and by type of study (primary study, supporting study, reviews and meta-analyses). Primary epidemiology studies that specified GBH exposure and LHC included 36 papers, 24 case control studies, 11 cohort. Ecologic (n=1, Taiba et al. 2025) and survivorship studies (n=1, Poh et al. 2022) were excluded from evaluation. Also excluded was one cohort study that reported standardized mortality ratios among herbicide applicators, but did not calculate an association of glyphosate use with mortality (Swaen et al. 2004); one case-control study that considered exposure crop density, but did not estimate glyphosate exposure (Malagoli et al. 2016); and one cohort study that did not report estimates for glyphosate exposure and LHC outcomes (Flower et al. 2004). Of the remaining 31 case-control and cohort studies that reported LHC incidence, there was a fair amount of overlapping study populations, with publications on the same study populations often split into multiple publications by cancer outcome, by strata, or methods papers using the same data.

For subsequent study evaluation, studies that had the largest level of pooling, or the most recent follow-up were included as the primary study for evaluation. If other studies include information not included in the largest or most recent analyses, such as stratified analyses or analyses of other cancer endpoints, that information will be extracted from these additional publications.

Cohort Studies

Of the 8 publications from cohort studies (Table 2), 6 studies were from the Agricultural Health Study (AHS), though some reporting on differing sub-populations, with differences in follow-up, or methods papers using AHS data. One pooled cohort study, with two publications, was the Agriculture and Cancer Cohort (AGRICOH), which included three cohort studies. AGRICOH included AHS, however the AHS participants included in AGRICOH did not fully overlap with the AHS specific publications, and included a shorter follow-up period. AGRICOH included a cohort in France that did not have an identified independent report. The third cohort included was in Norway. Therefore, AGRICOH was retained despite some, but not complete overlap with the AHS specific publications. Overall these were considered 2 unique study populations (AHS, AGRICOH).

Table 2. Identified publications from cohort studies for evaluation (n=8).

Cohort/Study	Study evaluation*	Author	Year	Title	Cancer outcome	Note
AHS	Primary	Andreotti	2018	Glyphosate Use and Cancer Incidence in the Agricultural Health Study	all cancers	Most up to date follow up.
	Additional study	Andreotti	2010	Body mass index, agricultural pesticide use, and cancer incidence in the Agricultural Health Study cohort	all cancers	Includes risk estimates stratified by BMI, older follow up.
	Additional study	De Roos	2005	Cancer incidence among glyphosate-exposed pesticide applicators in the Agricultural Health Study	all cancers	older follow up

Cohort/Study	Study evaluation*	Author	Year	Title	Cancer outcome	Note
	Additional study	Sorahan	2015	Multiple myeloma and glyphosate use: a re-analysis of US Agricultural Health Study (AHS) data	MM	reanalysis/methods
	Additional study	Burstyn	2016	Visualizing the heterogeneity of effects in the analysis of associations of multiple myeloma with glyphosate use. Comments on Sorahan, T. Multiple myeloma and glyphosate use: A re-analysis of US Agricultural Health Study (AHS) data	MM	reanalysis/methods
	Additional study	Lash	2007	Bias analysis applied to Agricultural Health Study publications to estimate non-random sources of uncertainty	MM	reanalysis/methods
AGRICOH	Primary	Leon	2019	Pesticide use and risk of non-Hodgkin lymphoid malignancies in agricultural cohorts from France, Norway and the USA: a pooled analysis from the AGRICOH consortium	NHL	Includes AHS data
	Primary	Kim	2023	Exposure to pesticides and risk of Hodgkin lymphoma in an international consortium of agricultural cohorts (AGRICOH)	HL	Includes AHS data

Abbreviations: AHS = Agricultural Health Study, AGRICOH = Agriculture and Cancer Cohort, MM = Multiple Myeloma; HL = Hodgkin Lymphoma, NHL = Non-Hodgkin Lymphoma
*Additional studies may contain additional risk estimates for additional outcomes or strata and/or additional information on study methods.

Case-control studies

Of the 23 identified case-control publications, several of the independent studies were subsequently included in subsequent pooling efforts. These pooling efforts are summarized below and in Table 3.

From Sweden there are three studies which were then pooled into a larger analysis. In the U.S., there are three case-control studies out of the U.S. National Cancer Institute (NCI) that all used the same study design and instruments in four states (Iowa, Minnesota, Kansas, and Nebraska), these were later pooled together into a pooled analysis (De Roos et al. 2003). In Canada, the Cross-Canada Study of Pesticides and Health (CCSPH) study was a male only population-based study designed to examine pesticide exposure and health across Canada. The CCSPH and the three NCI studies were later pooled together into a larger pooled analysis, the North American Pooled Project (NAPP).

In Europe, the Epilymph case-control study brought together data from six countries, the Czech Republic, France, Germany, Italy, Ireland, Spain. The data from the individual countries were not included in any identified literature. A subsequent case-control pooling effort, InterLymph, included 10 case-control studies from Europe, North America, and Australia, including Epilymph as one of the 10 studies. Of the 23 case-control publications, 2 were on childhood

leukemia (Park et al. 2020; Ward et al. 2023). Overall there were 3 pooled study projects considered (NAPP, Swedish, InterLymph) and 3 individual case-control studies (Meloni et al. 2021; Park et al. 2020; Ward et al. 2023).

Table 3. Identified publications from case-control and case-control pooling studies for evaluation (n=23 publications)

Pooled study	Included studies	Cancer outcome/References	Note
U.S. National Cancer Institute (NCI) Pooled case-control studies	NCI case-control study: Iowa/Minnesota (single study in two states)	NHL: Cantor et al. 1992; De Roos et al. 2003; Lee et al. 2004 MM: Brown et al. 1993	All used the same methods
	NCI case-control study: Kansas	NHL: De Roos et al. 2003; Lee et al. 2004	
	NCI case-control study: Nebraska	NHL: De Roos et al. 2003; Lee et al. 2004	
North American Pooling Project (NAPP)	Pooled (NCI and CCSPH)	Pooled: NHL: Pahwa et al. 2019 MM: Presutti et al. 2016 HL: Latifovic et al. 2020	Pools together CCSPH and the 3 NCI studies (listed above, "NCI Pooled case-control studies")
	Cross-Canada Study of Pesticides and Health (CCSPH)	Individual CCSPH: NHL: McDuffie et al. 2001 MM: Kachuri et al. 2013; Pahwa et al. 2012 HL: Hohenadel et al. 2011; Karunanayake et al. 2012	
Swedish Pooled case-control studies	3 case-control studies	Pooled: NHL and HCL: Hardell et al. 2023; Hardell et al. 2002 Individual studies: NHL: Eriksson et al. 2008; Hardell and Eriksson 1999 HCL: Nordström et al. 1998	
International Lymphoma Epidemiology Consortium (InterLymph)	10 case control studies	Lymphoma and subtypes: Cocco et al. 2013 (Epilymph), Orsi et al. 2009	Includes Epilymph (6 country study)
Individual study	Italian case control	Lymphoma and subtypes: Meloni et al. 2021	
Individual study – childhood	California home study	Childhood leukemia, Ward et al. 2023	
Individual study – childhood	California registry study	Childhood leukemia, Park et al. 2020	

1.3. Study Evaluation of Individual Epidemiologic Studies

The aim of this cancer hazard evaluation is to determine the level of evidence for carcinogenicity (sufficient or limited) from human cancer epidemiology studies. Systematic review and evidence-integration methods are used to conduct the evaluation.

Each primary study is systematically evaluated for its ability to inform the cancer hazard evaluation using five domains related to risk of bias – selection and attrition bias, exposure assessment, outcome assessment, potential confounding, and analysis – and one domain related to study sensitivity [or the ability of the study to detect a true effect (Cooper et al. 2016)]. The methods are adapted from the RoC Handbook. Reporting quality may also be noted (e.g., missing information).

The evaluation of the potential for bias (i.e., risk of bias) in each domain is captured by core questions for each domain. For each core question, a series of signaling and follow-up questions are used to address specific issues related to the core question. These questions are used to provide guidance and transparency for the domain-level judgment (options for domain-level judgements are described below). Responses to signaling and follow-up questions are then captured in the rationale for the response to the core question. These questions are meant to provide guidance, not to be a checklist. When adequate study information is available a domain-level judgment is made for the direction and distortion of each bias.

- ***No/minimal concern:*** The study characteristics being evaluated for the domain closely resemble ideal study characteristics. The potential for bias is considered minimal, recognizing the general limitations of observational studies.
- ***Some concern:*** The study design or methodologies are less than ideal for this domain. However, although there may be possible bias, these studies are generally considered informative for the cancer hazard evaluation.
- ***Moderate/major concern:*** The study design or methodologies suggests that the potential for a specific type of bias is high. However, depending on the direction and distortion of the potential bias, the study may still be informative for cancer hazard evaluation but should be viewed with caution.
- ***Critical concern:*** Distortion of bias would make the study findings unreliable for cancer hazard identification. This category is rare.
- ***No information:*** The information in the study is inadequate to evaluate the level of concern for the domain.
- ***Direction of bias:*** ↑Away from the null or overestimate of the effect (e.g., false positive); ↓towards the null or underestimate of the effect (e.g., false negative); not known (↔, unable to determine).
- ***Magnitude of bias:*** Probably minimal, moderate, major, or unknown. In most cases this is subjective but, if available, will likely be informed by sensitivity analysis.

Our approach will be to evaluate the components of study quality separately for studies reporting on each cancer type using the questions, domain level ratings, and guidelines given below.

1.3.1. Study Informativeness Guidance

Study informativeness guidance for human epidemiology studies are presented in the sections below. The study informativeness assessment for epidemiology studies encompasses five bias domains- selection bias, exposure misclassification or mis-measurement, outcome misclassification, confounding, and analyses- and a study sensitivity domain.

1.3.2. Selection and Attrition Bias

Selection bias occurs when selection into the study is related to both exposure and outcome; the relationship between the exposure and outcome is different for those who participated in the study (study population) than for the source population (all those who were eligible for the study, including those who did not participate) (Rothman et al. 2008).

Concern about selection bias is greater in case-control studies, owing to different probabilities of selection for cases and controls (Pearce et al. 2007); however, selection bias in most instances is less likely if participation was high among both cases and controls, given that the initial selection strategy was not biased.

When there is complete recruitment and follow-up, selection bias is usually less of a concern in cohort studies than in case-control studies and cross-sectional studies, because the cohort itself acts as the source population (Pearce et al. 2007); however, attrition bias or selection out of the cohort is a concern. Note that some specific biases could be considered in multiple domains. The RoC considers the healthy worker effect (HWE)—both the healthy worker-hire effect (HWHE) and healthy worker survivor effect (HWSE)—as a type of selection bias, but it can also be considered as confounding (Pearce et al. 2007). Selection bias is concern for all case-control studies if the selection of the control population does not adequately reflect the underlying source population of the cases.

Core question: Is there concern that selection into the study (or out of the study) was related to both exposure and outcome?

Table 4. Selection and Attrition Bias: Questions and Guidelines

Signaling Questions	Guidance	Response Options
Selection into the study: All study designs Is there concern that the selection methods were not adequate? Did the eligibility criteria (inclusion or exclusion) or recruitment strategies differ among study participants, such as between cases and controls (case-control studies) or exposed and unexposed subjects (cohort studies)?	Participation should not be an effect of both outcome and exposure status (e.g., conditioning on a collider). This usually biases an estimate away from the null, but in some cases can bias it toward the null (Hernán et al. 2004). Consider if participation or inclusion differs by covariate/confounding factors, such as region or race/ethnicity. This may bias the estimate in either direction if not controlled for in the analysis.	**Minimal concern** There is no evidence that selection of the subjects was related to both exposure and outcome. Cases and controls or exposed and nonexposed were selected from the same source population by similar methods and criteria. For cohort studies, the cohort is clearly defined (e.g., includes groups of those exposed and unexposed for a specific time period/location, with no evidence

Signaling Questions	Guidance	Response Options
• Is there concern that cases and controls in a case-control study or exposed or non-exposed in a cohort are not representative of the same underlying population during a similar time period? • In case-control and cross-sectional studies, is there a concern that participation differs (response rates) according to outcome status and is related to exposure status?	Ideally, the study participants should be similar in all respects except for exposure status (cohort) or disease status (case-control) and be drawn from the same underlying population. Note: this does not apply to cohorts where the cohort is not sampled based on exposure status. For **case-control studies**, controls should be from the same source population as the cases and be at risk of the outcome. Inappropriate selection of control groups that do not represent the underlying population from which the cases are selected can be a major concern. Selection bias is less likely if there is high participation among both cases and controls given the initial strategy was not biased (Pearce and Richiardi 2014; Snoep et al. 2014). The following scenarios indicate potential bias for **cohort and/or case-control studies**: • Differential participation and/or response related to outcome and exposure status (cohort, case-control). • Self-selection into the study (cohort, case-control). This can be assessed by comparing participants' characteristics with those of the target population (cohort and case-control). • Berkson's bias (case-control). This can be largely attenuated by limiting enrollment to incident cases and further attenuated by excluding cases and controls (if controls are from a diseased group) with a co-occurring condition that is not the reason for hospitalization (Pearce and Richiardi 2014; Snoep et al. 2014). • Cohort studies: Bias can occur if both exposure and outcome change over time (dos Santos Silva 1999; Soskolne et al. 2021; Vandenbroucke et al. 2007).	that follow-up differs between the exposed and nonexposed). **Some or major concern** There is some evidence that study selection may be related to both exposure and outcome. There is some evidence of HWE, HWHE or other occupation related selection bias. There is some evidence that the control population does not represent the underlying source population for the cases. **Critical concern** There is strong evidence that selection or attrition of subjects was clearly related to both exposure and outcome. Control selection is inappropriate.

Signaling Questions	Guidance	Response Options
• In cohort studies, is there concern that the health of a participant may have affected selection into the study (e.g., the HWHE[a] or healthy volunteer effect), leading to underlying differences in disease risk between exposed and unexposed individuals in the target population?	In occupational cohorts, standardized mortality or incidence ratios below 1 for mortality from all causes, all cancers, cardiovascular disease, or non-malignant respiratory diseases relative to the target population may be an indication of the HWE. The HWHE[a] generally biases the findings toward the null.	
Selection out of the study: Cohort study Is there concern about attrition bias or incomplete follow-up? • Is there concern that selection out of the study was related to both exposure and outcome status? • Is there concern that an analysis conditioned on censoring is related to both exposure and outcome?	Ideally, rigorous methods should be used to ascertain case status and should not differ by exposure and outcome status. Incomplete follow-up unrelated to either exposure or outcome (and therefore nondifferential) can reduce the statistical power of the study, but does not result in a biased effect estimate (Kristman et al. 2004). Ideally, proxy-reported deaths should be supplemented or confirmed with data from a national mortality registry to reduce loss to follow-up whenever possible (James et al. 1997). The proportion of cases using or confirmed by supplemental data should be reported.	

Signaling Questions	Guidance	Response Options
Is there concern about the timing of follow-up (e.g., follow-up time did not coincide with the start of exposure, especially for outcomes with shorter latencies)? This may overlap with these concerns: • Prevalent hires • Left truncation • HWSE	Ideally, studies enroll participants at the start of exposure and follow them for an adequate period, which is determined by the relative latency periods for the specific type of cancer. However, start of exposure is not usually known (sometimes it is available for occupational studies), and consideration of latency period is not typical. Cohorts that consist entirely of workers identified at one point in time (i.e., including both prevalent and incident hires) have been found to over-represent long-term, healthy workers (possible HWSE) for outcomes not observable at the time of hiring (left truncation) (Applebaum et al. 2011; Picciotto et al. 2013). Although the HWSE typically biases results toward the null (Stayner et al. 2003), its magnitude can be estimated, given sufficient information on the proportions of prevalent participants or workers and the length of the follow-up period.	
All studies Were analyses conducted to control for any selection bias (in or out) or sensitivity analyses conducted to address the extent of any bias?	Ideally, studies should conduct sensitivity analysis to estimate the extent of selection bias or to control for it, if such methods are available.	
If there is concern about the potential for selection or attrition bias, what is the predicted direction or distortion of the effect estimate (if there is enough information)?	When there is enough information to assess the predicted direction and/or distortion of selection or attrition bias, this assessment should be used in making the domain-level judgment for potential bias.	

Hover over underlined terms for pop-up definitions.
[a]The HWHE and HWSE are part of the healthy worker effect (HWE), which can be considered as both a type of selection bias and a type of confounding: however, it is addressed here under selection bias (Pearce et al. 2007).

1.3.3. Exposure Measurement Error and Exposure Misclassification

One of the most important aspects of an epidemiologic study is its ability to correctly classify study participants at the individual level with respect to their exposure status. This involves several dimensions: carefully defining the exposure used in the study, knowing information about the exposure setting, selecting appropriate data collection tools and methods for using or modeling the exposure data, evaluating the quality of the exposure assessment methods, determining whether individuals can be adequately separated in terms of their level of exposure, and assessing whether knowledge of the outcome may have affected the reporting of exposure.

Assessing the potential for bias from measurement error (or exposure misclassification) includes consideration of: (1) how well the exposure proxy approximates the exposure of interest; (2) how accurately and precisely the exposure is assessed (e.g., measured or estimated); and (3) differential recall bias or observational bias.

Any evaluation of exposure assessment includes complex consideration of the manner by which exposure data are collected, the way data are used to classify participants into exposure groups, and how missing data are addressed.

Glyphosate exposure occurs through contaminated air, water, or soil. The highest exposure is in farmworkers, those living in agricultural areas, and those working in landscaping and gardening, though there is also exposure in the general population due its widespread use. Those who work in the manufacturing of glyphosate are also subject to high exposure; in our search of the literature we did not identify any epidemiologic studies on this population. Glyphosate has a short elimination time in the body with an estimated half-life of 5-10 hours (Connolly et al. 2019; Zoller et al. 2020). A subcohort study within the Agricultural Health Study (AHS) examining urinary glyphosate levels in recently exposed farmworkers, farmworkers with high lifetime use but not recent use, farmers with minimal use, and non-farmworkers with no home garden use found detectable levels in all groups, however levels were strongly related to recency of use (0.89 vs 0.59 μg/g creatinine comparing farmworkers with recent use and those with high lifetime but not recent use) (Chang et al. 2024). Though glyphosate exposure may be higher in certain populations, the prevalence of glyphosate exposure is widespread, with approximately 80% of U.S. NHANES study population with detectable levels in urine samples, thus a truly non-exposed population is unlikely as a comparison group (Wu et al. 2025).

Most epidemiologic studies estimate glyphosate and GBH exposure from questionnaire data, which can be a reasonable tool to estimate long term exposure. Biomarker measurement for glyphosate can be done in urine, however given the short half-life of glyphosate, can provide an inaccurate estimation of long-term exposure and can miss capturing exposure given the often seasonable and intermittent use of glyphosate, e.g. if urine measures were done in the winter, however glyphosate was used heavily in the growing season, a urine measure might miss capturing exposure entirely. A study that includes samples from multiple time points may overcome these limitations in estimating long-term GBH exposure. Due to the inability of glyphosate biomarkers to capture long term-exposure from a single sample, and thus potentially inadequately represent the most biologically meaningful exposure period, the use of questionnaire data is a reasonable method to capture exposure.

Critical exposure windows for GBH for LHC outcomes are unclear, thus while timing of exposure is important, the identification of a specific exposure window may potentially miss a critical window, thus studies that examine multiple exposure windows are preferred.

For the considered studies, of specific concern, is the adequateness of the questionnaire tools used for most studies to capture specificities of exposure. This includes, the ability to distinguish GBH from other herbicides or chemicals, the ability to capture timing, duration, and frequency of use. If a study is capturing occupational exposure, how well does the study capture information on occupation? For studies that use questionnaires to calculate an exposure using a job-exposure matrix – how well does it calculate and calculate exposure duration, frequency, and timing?

Of concern for studies that include occupational exposures in a non-occupational specific study population, is differential recall. It is hypothesized that for farmworkers, given their role and responsibility for most aspects that would be involved in chemical treatment (not only application, but also purchasing/obtaining supplies, mixing chemicals, storing chemicals), that recall of specific chemical use by questionnaire would be improved by all the specific tasks involved beyond the application itself (Blair and Zahm 1990). If a study includes both farmworkers and general population participant, there may be differential accuracy in recall for the questionnaires used for exposure assessment.

For some studies, proxy respondents were used to complete questionnaires for deceased individuals. This might be of concern as there is likely a greater amount of exposure measurement error in estimates from proxy respondents compared to self-report. There is some evidence that proxy respondents might lead to misclassification, however that it would be non-differential with the potential to bias towards the null (Blair and Zahm 1990). If a study includes proxy respondents, ideally, they should provide some evidence of the validity or comparability of these responses to self-reported data. If they do not provide this, they should account for the use of proxy measures in the analysis to account for potential differences in exposure measurement. Childhood cancer studies are mostly dependent on parental exposure as a proxy, which would be a source of measurement-error, though likely non-differential for prospective cohort studies, for case-control and retrospective cohort studies there is the possibility this would be non-differential measurement error.

Table 5. Exposure Measurement Bias: Questions and Responses

Signaling question	Guidance	Response options
Exposure estimation Is there a concern that the exposure estimation did not adequately represent the exposure for the outcome of interest? If yes, was this true for all exposure metrics, or a particular metric?	Any evaluation of exposure assessment methods includes consideration of how well the data are collected and how the data are used to classify participants into exposure groups. This is particularly important for studies in which the exposure contrasts in the population are small. For childhood studies estimation of exposure may be more indirect, with parental exposure as a proxy.	**No/minimal concern:** The exposure estimate (estimation of GBH exposure) used in the study closely approximates the exposure of interest. Exposure groups are adequately separated (for categorical exposures).
Exposure measurement Is there concern about error in measurement of the exposure of interest or exposure proxy? Is there concern about the use of the collected data to classify exposure groups? If yes to either, is there concern that measurement error resulted in inadequate separation of groups with respect to exposure? • Did any misclassification vary by exposure category? • Is there concern that the exposure classification did not capture the variability of exposure? Is there concern regarding the timing of exposure estimation and the estimated exposure window?	Ideally, the exposure would be consistently assessed across exposure groups through the use of established or validated methods, with any measurement error being small in relation to between-individual variation compared with differences between groups (e.g., unexposed vs. exposed, high vs. low exposure). Exposure measurement may vary depending on the timing of exposure. In studies with large proportions of exposed individuals, attention to the definition of "unexposed" is important, as these individuals may not be completely unexposed, potentially introducing bias toward the null. In the US ~80% of the population is estimated to have some exposure according to NHANES, likely leading to some GBH exposure in "unexposed" or low exposed groups. This would likely bias towards the null. Most available studies assessed exposure via questionnaire. This is a reasonable exposure estimation tool; however, it may not accurately capture frequency, duration, or the amount of exposure, which may be more relevant for evaluating causality; however the questionnaire instruments themselves, and the use of the data to develop metrics can vary.	Any measurement error is non-differential and small in relation to between-individual variation compared to differences between groups. Timing of exposure estimation is prior to diagnosis. There is no evidence of differential recall. **Some concern:** The study allows for good discrimination between exposed and non-exposed, and potentially between exposure categories. Any measurement error is non-differential. **Moderate/major concern:** The exposure estimation does not specifically, or inadequately captures GBH well, or the estimation is not extensive enough to be confident that exposed and non- or low- exposed groups and/or exposure categories do not overlap somewhat. Minimal additional exposure metrics are available. Timing of exposure estimation is unclear or not stated.

Signaling question	Guidance	Response options
	Ideally, information from questionnaire data should be able to: • Distinguish GBH use from other pesticides, insecticides, herbicides, and other chemicals (e.g. were GBH products specifically assessed in the questionnaire) • Provide information on frequency of use • Provide information on timing of use • Provide information on occupational use vs household use Did the exposure metrics developed adequately capture timing and duration of use and provide useful exposure contrasts? Differing exposure metrics may have been calculated (ever/ never, categories of usage frequency, categories of usage duration). Also to consider in case control studies, is if the exposure estimation tool was consistent in both groups, or were controls asked different questions than cases. If the estimation tool differed between case and control groups, this would bias results away from the null due to differential measurement error. If proxies are used for cases and controls, or for prospective cohort studies, this might result in non-differential recall bias (bias towards the null) or in differential recall bias (bias in either direction). For exposure windows – consider if they are specified and justified for the study. For retrospective cohort studies, did the exposure estimation tool estimate exposure for a relevant time point prior to diagnosis? For case-control studies, was exposure estimated for a time point prior to diagnosis in the cases? Was this adequately matched to a similar exposure window in controls? For childhood cancers, was exposure estimated in utero and/or during early childhood?	There is a possibility of differential recall bias, but it is unlikely to be substantial. **Critical concern:** Exposure assessment is not at the individual level and /or the exposure assessment does not approximate the exposure of interest and there is strong evidence that it is unable to differential to capture exposure and non- or low-exposed participants. No additional exposure metrics. Timing of exposure estimation is not prior to case diagnosis. Differential recall bias is clearly present and substantial.

Signaling question	Guidance	Response options
Observation and differential recall bias Is there concern that knowledge of the outcome (e.g. observation or recall bias) may be differential and potentially bias the exposure assessment (away from the null?)	There may be differential recall bias if cases remember or report chemical use differently than controls. This might be possible for case control studies, and in retrospective cohort studies. There is less concern for differential recall bias in prospective cohort studies. Recall bias may also be a concern if exposure questionnaire is obtained from proxy respondents. If proxies are only used for cases, or in retrospective cohort studies, this may be result in differential recall bias (bias away from the null).	
Is there concern that presence of the outcome may potentially bias the exposure assessment?	The outcome causing GBH exposure is not a concern in GBH studies.	
Is any misclassification differential or nondifferential and what is the predicted direction or distortion of the effect estimate (if there is adequate information)?	Non-differential exposure misclassification biases towards the null for a dichotomized exposure (e.g. ever/never) and is most likely present in all studies. Differential recall bias might be present in case-control studies. Case recall may be more accurately reported than control recall, thus potentially biasing the results away from the null if suspected.	

1.3.4. Outcome Misclassification

The outcome of interest is incidence of a specific cancer type or subtype. Assessment of the potential for bias due to measurement error or outcome misclassification considers: (1) how well the study outcome represents the outcome of interest; (2) the accuracy of the outcome measurement methods; and (3) the potential for observation bias. Cancer incidence data are considered more informative than mortality data, because sources of incidence data (e.g., cancer registries, hospital records) are more accurate than sources of mortality data (e.g., death certificates) (Jewkes and Wood 1998; Linet et al. 2020; NCHS 2013; Patel et al. 2004) and mortality may not reflect incidence for cancers with high survival rates.

Lymphohematopoietic cancers (LHCs) are a heterogeneous grouping of blood- or lymph-based tumors encompassing leukemias and lymphomas. Leukemia originates in the bone barrow and blood, while lymphomas originate in the lymphatic system. Lymphomas include: Hodgkin Lymphoma (HL), Multiple Myeloma (MM), Non-Hodgkin Lymphoma (NHL).

Adult and childhood LHC will be considered separately in the evaluation. Each subtype below will be considered separately when feasible.

Cancer statistics and information:

***Hodgkin Lymphoma* (HL):**

In the U.S. 2018-2022 SEER data, Hodgkin Lymphoma has an age-adjusted incidence of 2.5 per 100,000, with an incidence of 2.8 in males, and 2.2 in females (SEER 2026). The highest percentage of new cases are diagnosed at a relatively young age, with 31.3% of new cases diagnosed at ages 20-34. The overall 5-year survival rate is 89% (2015-2021 SEER data). Globally the age-adjusted incidence of HL is 1.2 in males and 0.8 in females per 100,000 (Sung et al. 2021).

***Multiple Myeloma* (MM):**

In the U.S. MM has an age-adjusted incidence of 7.3 per 100,000 with slightly higher rates in males (8.8 per 100,000) than females (6.1 per 100,000) and high rates in the non-Hispanic Black (17.2 in men, 13.2 in women per 100,000) compared to other race/ethnicity groups (SEER 2018-2022). The overall 5-year survival rate for myeloma in the U.S. is 62.4% (2015-2021 SEER data). New cases have a median age of diagnosis of age 69, with 32.4 percent of cases diagnosed at 65-74. Globally the age-standardized incidence of MM is 2.2 in males and 1.1 in females per 100,000 (Sung et al. 2021).

Non-Hodgkin Lymphoma:

NHL is made up of two main groups categorized by their cellular origins; B cell lymphomas which make up the majority of NHL cancers (85%) and Natural killer/T-cell lymphomas (~15%) (LRF 2024; 2026) B cell lymphomas are further broken down into subtypes: Diffuse large B-cell lymphoma (DLBCL), Chronic lymphocytic leukemia (CLL)/Small lymphocytic lymphoma (CLL/SLL), follicular lymphoma (FL), and hairy cell lymphoma (HCL). The risk factors for NHL largely unknown, though potential risk factors include: oxidative stress, immunotoxicity, infectious agents, and autoimmune disorders (Sapkota and Shaikh 2025). There is evidence that there is heterogeneity for risk factors and etiology across subtypes, studies that are able to, should examine associations by NHL subtype (Morton et al. 2006).

The diagnostic categories for NHL have changed over time. In 1994 a revised classification system was introduced, the Revised European-American (REAL) classification system which was incorporated into ICD-O-2 and aligns with ICD-O-3 and generally adopted in the U.S. and Europe (Al-Hamadani et al. 2015; Harris et al. 1994). These diagnostic criteria were incorporated into WHO classifications in 2001 (Morton et al. 2006). Changes included the clarification and specification of subtype based on morphology, molecular features, clinical behavior, and pathogenesis and incorporates cellular origins of NHL subtypes (Morton et al). Cancer surveillance systems, such as SEER, include conversion factors to account for these changes in diagnostic practices. The majority of included studies include cases after these criteria were adopted. For older studies, including cases diagnosed before the mid-1990s, the broader diagnostic criteria of NHL remained consistent. Studies pooling data from older and newer studies without incorporating available conversions for NHL subtypes, or controlling for differences in diagnostic definitions in some way, might be subject to outcome misclassification.

In the U.S. 2018-2022 SEER data, NHL age-adjusted incidence rates were 22.4 and 15.7 per 100,000 for males and females, respectively. In the U.S. NHL is the 8th most common type of cancer with a median age of diagnosis is age 68 (28.5% of cases diagnosed between ages 65-74). The overall 5-year survival rate in the U.S. is 74.2% (2015-2021 SEER data).

Worldwide in 2020 the age-standardized incidence of NHL was 6.9 and 4.8 per 100,000 person years for males and females, respectively (GCO 2024; Sung et al. 2021).

***Leukemia*:**

Leukemia is comprised of several subtypes including Acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), and chronic myeloid leukemia (CML). In the U.S. SEER 2018-2022 data, leukemia has an age-adjusted incidence of 14.4 per 100,000 with higher rates in men compared to women (18.2 and 11.3 per 100,000, respectively). New cases of leukemia have a median age of diagnosis of 68, though of note, ALL differs from other subtypes with diagnosis primarily at younger ages, with a median age of diagnosis at age 17 and over 50% diagnosed at <20 years old. The overall 5-year survival rate in the U.S. for leukemia is 67.8%, of the subtypes, AML has by far the lowest rate (32.9%) (2015-2021 SEER data). Given that ALL is diagnosed at younger ages, and that AML has markedly different survival rates, consideration of leukemia subtypes is of importance. Globally, leukemia has an age-standardized incidence of 6.3 in males and 4.5 in females per 100,000 person years (Sung et al. 2021).

Given the relatively high survival rates of most LHCs, cancer incidence data are considered more accurate than mortality data as mortality will likely miss a large proportion of cases. Given the potential heterogeneity, specifically in NHL and leukemia, ideally subtypes should be considered.

Childhood lymphohematopoietic cancers:

Leukemia is the most common pediatric cancer with an age-adjusted incidence of 48.0 per 1 million and a 87.3% 5-year survival rate, followed by lymphomas with an incidence of 29.0 per 1 million and an 95.2% 5-year survival rate (U.S., ages <20, 2018-2022). Leukemias are most common in the 1-4 year old age group with an incidence of 88.9 compared to incidence rates of

34-44 per 1 million in other age groups (<1, 5-9, 10-14, and 15-19 years old) (National Childhood Cancer Registry 2025).

For leukemia, the lymphoid leukemias, including ALL and CLL, are the most common with an incidence of 31.1 in females and 39.7 in males per 1 million ages <20 in the U.S. (2018-2022). AML has an incidence of 7.8 in females and 8.2 in males per 1 million ages <20. In the 1-4 year old age group, the incidence is 75.6 compared to rates of 17-36 per 1 million in other age groups.

Within lymphoma, NHL has an incidence of 7.9 in females and 12.9 in males and HL has an incidence of 11.5 in females and 13.0 in males per 1 million ages <20 in the U.S. (2018-2022). Both NHL and HL are most common in the 15-19 year age group, with an incidence of 17.0 for NHL and 32.3 for HL compared to an incidence of 11.2 for NHL and 12.3 for HL in the group 10-14 year age group and even lower in younger ages groups (National Childhood Cancer Registry 2025).

Given the difference in incidence by age, leukemias are more of a concern for younger ages, where lymphomas are more of a concern for adolescents. When studying childhood cancers, study populations for leukemias and lymphomas should take this into consideration.

Core question: Is there concern that the outcome measure did not reliably distinguish between the presence or absence (or degrees of severity) of the outcome?

Table 6. Outcome Misclassification: Questions and Responses

Signaling question	Guidance	Response Options
Is there concern that the method of measuring outcome did not represent the outcome of interest? If mortality data are used, do they adequately reflect incidence?	Because survival is relatively high for most LHCs, incidence data is considered the most accurate and mortality data is not useful; mortality data can be considered useful specifically for AML which has low survival rates. Ideally, cancer subtypes should be measured to add specificity to the LHC outcome of interest.	**No/minimal concern** Outcome methods clearly distinguish between participants diagnosed with a specific cancer type, including cancer subtypes, and participants not diagnosed with that cancer. Follow-up and diagnoses are conducted independent of exposure status. Cancers are

Signaling question	Guidance	Response Options
Is there concern that the disease was not accurately diagnosed? For example: • Does misclassification of outcome v ary across exposure groups or levels of exposure? • If so, were there methods to adjust for any potential bias? • Is there concern that the control group may have cancer?	Ideally, cases of cancer should be histologically confirmed and/or undergo independent pathology review (e.g., on a subset of the cases) by the study investigator. Incidence data from population-based cancer registry sources or hospital pathology data are generally more detailed and accurate than death certificates, as their sources are medical records and cancer registry data. Cancer incidence registries, especially population-based registries, are generally preferred; however, these registries are not consistently available or reliable in LMICs and will miss cases. Hospital and medical records should be used for studies conducted in such countries. Self-reports (or a large percentage of them) should be medically confirmed. Most studies used hospital records, registry, or death records. In absence of medical confirmation, studies that use self-report, should have a validation study to confirm the validity of self-report of outcomes. Proportion of cases ascertained by method(s) other than histological confirmation, and the potential for this to be related to exposure, should be noted.	histologically/cytologically verified (documented in hospital/personal medical records or cancer registry). **Some concern** Cancer diagnoses, or a substantial proportion of cancer diagnoses, are histologically/cytologically verified. Some diagnoses may be verified only clinically but criteria are well documented. Major LHC categories are distinguished, but not all subtypes are assessed. **Moderate/major concern** Cancer diagnosis and type are self-reported, and neither are verified by cancer registry or medical/hospital records, and/or have no or low validation. **Critical concern** There is strong evidence that follow-up and cancer diagnoses are likely related to exposure status or that the methods do not discriminate between diseased and non-diseased participants (unlikely in most cancer studies). LHC is considered as one broad cancer outcome.
Is there concern about observer bias?	Ideally, the outcome assessors do not have knowledge of the participant's exposure status, nor are they influenced by exposure status, which could result in differential bias. This is not likely to be a concern in GBH studies.	
Is any misclassification differential or nondifferential, and what is the predicted direction or distortion of the effect estimate (if there is adequate information)?	Nondifferential outcome misclassification is possible, but very unlikely, and will bias results toward the null.	

1.3.5. Confounding Bias

The evaluation of confounding is a multi-step process that requires consideration of both study methods and study findings. This section discusses: (1) potential confounders that should ideally be considered in studies of lymphohematopoietic (LHC) cancers and exposure to glyphosate or glyphosate-based herbicides; and (2) approaches for evaluating how study authors assessed confounding and/or provided information relevant to its evaluation. Methods for assessing the impact of potential confounders on study findings are described in Section 1.4. Control of

variables that may reduce precision but are unlikely to bias the effect estimate (i.e., variables that are not true confounders) is addressed in the analysis domain.

Potential Confounders

Major potential confounders are defined as factors that are likely to be associated with exposure and associated with disease, are not part of the causal pathway, and are not highly correlated with other risk factors. Although empirical data describing the relationship between GBH exposure and many potential confounders are limited, several exposures were considered potential confounders when a plausible association with GBH exposure could reasonably be expected.

For the LHC cancers under consideration (i.e., non-Hodgkin lymphoma, Hodgkin lymphoma, and leukemias), candidate potential confounders identified across studies are listed in Table 7. Factors established as known risk factors for the cancer of interest (e.g., identified by authoritative sources such as IARC, the Report on Carcinogens [RoC], and the World Cancer Research Fund) are presented in Column 2. Factors likely to be associated with GBH exposure (see Column 3) are considered critical potential confounders and are presented in Column 4.

Confounding must be evaluated on a study-specific basis because the relationship between a cancer risk factor (e.g., animal production) and GBH exposure may vary across populations. Accordingly, it may not be possible to define a uniform set of confounders applicable to all studies. In addition, some risk factors may be population-specific (e.g., obesity) and associated with socioeconomic status (SES); adjustment for SES may therefore only partially account for these factors.

Confounders or effect modifiers across cancer types

Age, gender, race/ethnicity: Ideally, age, gender, and race/ethnicity should be evaluated as potential effect modifiers through stratified analyses, in addition to being controlled for in overall analyses.

Socioeconomic status (or socioeconomic position (SEP)). Socioeconomic factors (e.g. area level SES index, household income, participant education level) are considered a risk factor for NHL and HL survival, but not necessarily incidence, therefore not a confounder (Clarke et al. 2005; Hermann et al. 2010). SES should ideally be evaluated as potential effect modifiers through stratified analyses. Consideration of SES may partially account, in some populations, for personal behaviors such as smoking. SES may be relatively homogeneous in cohort studies of applicators or farm workers, as unexposed workers may be similar to other applicators or farmers. SES might be a concern in case-control studies where the reference group is drawn from the general population.

Non-neoplastic diseases: Autoimmune or immunosuppression diseases were not considered to be confounders because they may potentially lie in the casual pathway. Exposure to GBH was associated with autoimmune diseases (e.g., rheumatoid arthritis, celiac disease) (Parks et al. 2026) and autoimmune diseases (exposure agnostic) is a strong risk factor for NHL (Ekström Smedby et al. 2008; Parks et al. 2016; Yang et al. 2025).

Animal Production: Several studies have reported that substantial occupational contact with animals (e.g., farmers raising livestock) or close residential proximity to large-scale animal production facilities is associated with an increased risk of lymphomas. However, in at least one

study, there is a low reported correlation and variation in GBH use and proximity to animal facilities (Fisher et al. 2020). Thus, proximity to animal production facilities may not be a strong confounder.

Family history of LHC cancer: Family history is unlikely a confounder. Genetics is associated with the outcome, but not the exposure. Shared environment, that might be captured by family history of LHC cancer, is not likely linked to risk factors that would be independently associated with exposure.

Table 7. Potential Confounders for Lymphohematopoietic Cancers and Exposure to Glyphosate-based Herbicides.

Cancer type	Risk factors	Comments	Potential confounders
NHL	Lifestyle: Obesity, tobacco smoking (follicular cell lymphomas) Medical: Viruses, immunosuppression drugs, autoimmune or immunosuppressive diseases Chemicals: Certain solvents (1-3 butadiene[b], styrene[b], benzene, methylene chloride, TCE), PCB, PCP, ethylene oxide Pesticides: Lindane, chlorophenoxy, DDT, Malathion, diazinon Other occupational: Animal production, radiation	Other chemicals: Solvents and medical-related factors not likely to be linked to GBH exposure and are not considered to be key confounders. Adjustment for socioeconomic status (SES) may partially account for related factors such as obesity or differential risk of viral infections, to the extent that these are socially patterned. However, SES may not fully capture these factors, and residual confounding could remain. Immunosuppression or autoimmune disease may be part of the proposed causal pathway rather than confounders. Animal production and pesticide exposure may be population dependent. Animal production may not be associated with GBH in all populations. Some pesticides have been banned and depending on the calendar year of the study, individuals may have had little or no exposure.	Age, gender *Key confounders*: Co-exposure to pesticides *Minor confounder:* tobacco smoking for follicular cell lymphomas. Population or study dependent: Race/ethnicity[a], SES[a], study site (or calendar year), pesticides strongly correlated with GBH, Animal production could be an effect modifier, especially if it potentially cause NHL by causal pathways as GBH.
Multiple myeloma	Chemicals: Solvents (1,3, butadiene, styrene, benzene), automobile gasoline, ethylene oxide, PCP Other occupational: Animal production, radiation	PCP was banned in 1979: its used in GBH studies may not be limited.	Age, gender Minor confounder or effect modifier: Animal production Population dependent: Race/ethnicity[a], SES[a]

Cancer type	Risk factors	Comments	Potential confounders
HL	Virus	Viral infection unlikely to correlate with GBH exposure.	Age, gender, SES[a]
Leukemia	Lifestyle: tobacco smoking (myeloid) Medical: Chemotherapeutic drugs (mostly AML) Chemicals: Solvents (benzene), formaldehyde (myeloid), gasoline (CLL and myelodysplastic) Pesticides: Diazinon	Chemotherapeutic drugs and solvent exposure are unlikely to be correlated with exposure to GBH.	Age, gender Key confounders: tobacco smoking (myeloid), and possibly diazinon Population dependent: SES[a], family history
Childhood leukemia	Lifestyle: Parental (pre and post-natal) tobacco smoking Medical: Chemotherapeutic drugs (mostly AML) Chemicals: Solvents (benzene), formaldehyde (myeloid), gasoline (CLL and myelodysplastic) Traffic related air pollution (TRAP) Radiation Pesticides Insecticides	Medical, radiation, TRAP, benzene, and formaldehyde unlikely to be correlated with glyphosate exposure. Consideration of parental/household SES may partially account for confounding by parental smoking (due to the correlation of smoking and SES).	Age, family history of leukemia, environmental tobacco smoke, prenatal maternal smoking, Population dependent: other pesticides or insecticides, gender, SES

Sources: IARC 2017; NTP 2025
Abbreviations: AML = acute myeloid leukemia, CLL = chronic lymphocytic leukemia, DDT = dichloro-diphenyl-trichloroethane, GBH = glyphosate based herbicides, HL = Hodgkin lymphoma, NHL = Non-Hodgkin lymphoma, PCB = polychlorinated biphenyls, PCP = pentachlorophenol, SES = socioeconomic status, TCE = trichloroethylene, TRAP = traffic related air pollution.
[a]Ideally, age, gender, SES, race/ethnicity should be considered in the analysis as effect modifiers.
[b]Risk factor for all lymphomas

Core question: Is there concern that the effect estimate may be confounded, e.g., potential confounding is either not adequately addressed by the methods or there is inadequate information to allow for its evaluation?[a]

Table 8. Potential Confounding: Questions and Responses

Signaling Questions	Guidance	Response Options
Is there concern about the measurement of co-exposures or personal behaviors in the study? If no data are provided about confounders, are surrogate data on potential confounders available?	Ideally, quantitative information on personal behaviors and other likely confounders should be assessed by in-person interview conducted by interviewers blinded to the case status of the respondent, rather than via proxy respondents. Residual confounding is more likely when only limited qualitative information on a given risk factor (e.g., only "yes/no") is available. Studies should provide data on the distribution of potential confounders by exposure and disease status. Data may be available on potential confounders in subsamples, which can help provide interpretation of the prevalence of the potential confounders in the exposed and unexposed or cases and controls. In addition, data on diseases associated with exposure may provide indirect information about risk factors for specific cancer end points of concern.	**Minimal or some concern** The study measured key potential confounders and/or used appropriate analyses or designs to address them. **Some or major concern** These are evaluation-specific and will be defined in the protocol. **Critical concern** There is strong evidence that the effects of the exposure cannot be distinguished from the effects of potential confounders.
Is there concern that the design or analysis did not adequately address important confounding through matching, stratification, multivariable analysis, or other approaches? Is there additional information available with which to evaluate potential confounding or conduct sensitivity analyses (indirect adjustment)? Is there concern that controlling for one or more variables (such as those in the causal pathway) could cause bias? Is there concern that not adjusting for one or more confounders would be expected to differentially favor outcomes in those with higher or lower levels of exposure?	Ideally, confounders should be controlled for in the design or analysis phase. Not controlling for unaccounted potential confounders is likely to bias the results. If there is no information on confounders, determine whether external information (e.g., smoking rates in population, strength of the risk factor for the outcome) can inform whether confounding is an issue. Comparing minimally adjusted (e.g., by age) to fully adjusted models can help inform whether a factor is a confounder. Some potential confounders could also be effect modifiers. Ideally, studies should stratify by these variables to indicate whether a variable is an effect modifier. Considering a variable only as a confounder may obscure possible effects in high-	

Signaling Questions	Guidance	Response Options
	risk or vulnerable subgroup(s) within the study population. Statistical models over-adjusted for factors that do not meet the definition of a confounder may introduce bias and may result in a loss of precision. If collinearity from highly correlated co-exposures is likely to introduce confounding, determine whether additional analyses (e.g., correlation matrices, stratified models, multipollutant models, mixtures methods) can identify the individual effects while controlling for co-exposures.	
What is the predicted direction or distortion of the effect estimate (if there is adequate information)?	Confounding can lead to an over- or underestimation of the risk estimate. When there is enough information to assess the predicted direction and/or distortion of confounding, this assessment should be used in making the domain-level judgment for potential bias.	

[a]The healthy worker effect is both a special type of confounding and a type of selection bias (see Selection and Attrition Bias section).

1.3.6. Analysis Bias

The assessment of analysis bias considers the appropriateness of data assumptions, statistical models and methods used in the statistical analysis to evaluate the overall findings. Bias can occur when adjusting for variables in the causal pathway between exposure and disease. Overadjustment refers to adjusting for additional variables in statistical models that are not considered to be true confounders (see Confounding section 1.3.5). This would decrease precision but does not bias the results.

When adequate data are available, studies should evaluate exposure-response relationships, periods of susceptibility, and latency, or conduct subgroup analyses (especially for subgroups exposed at higher levels for longer durations). This assessment overlaps somewhat with study sensitivity. In some studies, such as case-control studies evaluating exposure to numerous substances without clear hypotheses, appropriate consideration should be given when interpreting multiple comparisons. Some LHC have shorter latency period than solid tumors and the latency is unknown. Ideally, studies should evaluate different lag periods and unlagged data.

Data assumptions for laboratory measured exposures are not relevant for most studies as they assessed exposure by questionnaire.

Core question: Is there concern that the data assumptions and analysis were not adequate or that the study did not conduct relevant analysis of the available data?

Table 9. Analysis: Questions and Guidelines

Signaling Questions	Guidance	Response Options
Data assumptions Is there concern about whether the data assumptions used in the statistical analysis were adequate?	For example, are data transformation methods (e.g., log transformation) appropriate Or is an assumption of linearity appropriate? Were outliers removed? (Ideally, outliers should not be removed without strong justification.	**Minimal or some concern** The study used relevant and appropriate data assumption, analysis methods and handled missing data appropriately
Statistical model and methods Is there concern about the appropriateness of the statistical model for the study design or about the adequacy of the conduct of the analysis?	Examples of models include Cox proportional hazards regression (hazard ratio), Poisson regression, multivariable logistic regression (odds ratio), and conditional logistic regression. Matching should be adequately described and accounted for in the analysis (e.g., for controls described as individually matched to cases, was the ratio of cases to controls included, and were conditional regression techniques applied?).	**Some concern** The study uses accepted methods or data assumptions but not the most sensitive or ideal of sensitive. Imputed data may raise some concerns or missing data not missing at random with complete case analysis. **Major or critical concern** There is strong evidence that the study's analytical methods were so limited that the findings were

Signaling Questions	Guidance	Response Options
If applicable, did the study use appropriate methods to adequately evaluate exposure-response and latency, or to conduct subgroup analyses?	Analyses of subgroups exposed at higher levels or for longer durations should use appropriate methods to delineate groupings. Questions about the adequacy of methods include whether the data were modeled continuously or divided into categories, and whether the analysis used linear tests for trend and appropriately stratified groups.	uninterpretable or were distorted such that no conclusion can be made.
Is there concern about "over-controlling" (i.e., controlling for variables unnecessarily)?	Controlling for variables that are not related to exposure or disease will most likely reduce the precision of the risk estimate.	
Missing data Is there concern that missing data may have biased the findings? • Is there concern that missing data on the outcome or on any potential confounders is substantial? • Is there concern that missing data were not handled by an analytically appropriate method (e.g., sensitivity analysis or imputation)?	Complete subject analysis (i.e., including only those participants with complete data for all variables modeled) is considered less than ideal. Ideally, there should be little or no concern that the data are missing for reasons related to both exposure and disease.	
What is the direction, magnitude, and impact of this bias on the effect estimate?	When there is enough information to assess the predicted direction and/or distortion of analysis bias, this assessment should be used in making the domain-level judgment for potential bias. This may be difficult to ascertain for most analyses.	

1.3.7. Study Sensitivity

Study sensitivity is the ability of a study to detect a true effect or hazard (Cooper et al. 2016) and is analogous to the term "informativeness" used in the preamble to the IARC Monographs (IARC 2019; Samet et al. 2020). Studies with low risk of bias but insensitive may not be informative for reaching public health decisions about a potential causal relationship between exposure and outcome. Consideration of both sensitivity and the potential for bias are needed to identify the most informative studies and to identify those study elements that may help to explain heterogeneity across the body of literature. Failure to consider sensitivity may result in overweighting the results from insensitive studies, or erroneously interpreting evidence as being conflicting (Cooper et al. 2016). Study sensitivity should be evaluated with the same rigor as risk of bias.

Our assessment of study sensitivity includes consideration of (1) study size or the numbers of exposed and non-exposed participants or cases and controls, (2) exposure contrast and window, and (3) latency. The overall sensitivity evaluation requires an integration of these factors.

Core question: Does the study have adequate sensitivity to detect an effect from exposure (if present)?

Table 10. Study Sensitivity: Questions and Guidelines

Signaling Questions	Guidance	Response Options
Statistical power Is there concern that the number of exposed cases were not adequate for detection of an effect in the exposed population or subgroups of the exposed population?	When both exposure and disease are rare, statistical power is determined largely by the number of exposed cases and exposed controls. Pooling of multiple studies (e.g., case-control studies of LHCs) may overcome poor statistical power in some instances; however, care should be taken to evaluate the appropriateness of pooling multiple studies. If data pooling is infeasible based on irreconcilable methods across studies, an evaluation of individual studies may be warranted.	**Minimal concern** The study had an adequate number of exposed participants, with substantial exposure (level, duration, or range) and with adequate duration of follow-up for latency status. **Some or major concern**

Signaling Questions	Guidance	Response Options
Exposure contrast Is there concern that the levels, duration, or range of exposure of the population at risk in cohort and case-control studies was not sufficient or adequate for detection of an effect of exposure? • Did the exposed group include individuals with a low or unknown probability of exposure?	Dilution of risk estimates comparing exposed and referent groups can occur when exposure varies widely within the group(s)/categories defined as exposed. The ability to evaluate exposure-response relationships depends on an adequate range of exposure among the study participants and adequate numbers of participants in each exposure category. Ordinal categorization of exposure levels (intensity, frequency, duration of exposure) may be advantageous to ensuring adequate exposure contrast in questionnaire-based exposure assessment collection methods.	Limited statistical power (e.g., small number of exposed cases), poor exposure contrast, and/or limited latency. **Critical concern** The study had very few exposed subjects that critically impacted the ability to interpret statistical results, and/or the exposure range was so minimal that there is no ability to differentiate between exposure groups in a meaningful way.
Latency Was the elapsed time between exposure and the outcome measurement sufficient to allow for cancer induction?	The latency for lymphoproliferative and hematopoietic cancers can vary substantially by subtype with estimates of 2-10 years for lymphomas, and a range of 1.5-35 years for leukemias (WTC 2015). Given this variability and widespread cumulative exposure to GBH, a questionnaire-based assessment may consider reliance on lag and non-lag models as relevant. Ideally studies will consider at least 10 years of follow up from exposure estimation to cancer assessment.	

1.3.8. Judgement for Study Informativeness and Overall Assessment of Each Individual Study

The overall informativeness of each individual study is based on consideration of both the potential for bias (i.e., internal validity) and study sensitivity. Serious concerns about study quality will result in a lower informativeness judgment. However, a well-designed study with low sensitivity (e.g., having few exposed or expected cases for a specific end point) could be considered as having low informativeness for the cancer hazard assessment. In very rare cases, a study that meets inclusion criteria may ultimately be excluded from evidence integration if they are totally and obviously uninformative (such as documented evidence of severe exposure misclassification).

Box 3-2. Study Informativeness-Level Judgement

High: no or minimal concerns about most potential biases, high or moderate sensitivity.

Moderate: low, minimal, or some concerns about most potential biases.

Low: major concerns about several biases, sensitivity rating varies. Depending on the direction and distortion of the potential biases, the study may still be informative for the cancer hazard evaluation and can help explain heterogeneity of the findings.

Inadequate (very rare): critical concern about any bias; sensitivity rating varies.

For transparency, the study informativeness assessments are included in the monograph, and the findings may be briefly summarized, or evaluated in sensitivity analysis. When adequate information is available for a study, a judgment is made on the direction and distortion of known or possible overall biases or whether it has low sensitivity to detect an effect (see Box 3-2 from the RoC Handbook 2025).

The goal of the evaluation is to consider all the evidence and triangulate across the body of evidence, rather than exclude studies. Studies raising critical concern about bias (which is very rare) in at least one domain may be evaluated in the sensitivity analysis. The overall judgment of study informativeness is not meant to be an algorithm that sums up the ratings across domains. Different domains may be given greater weight depending on issues important for the specific candidate substance. For databases in which the quality of the studies varies considerably, informativeness-level categories may be combined, such as “moderate or low”.

1.4. Evidence Evaluation and Interpretation

1.4.1. Evaluation of the Evidence from Individual Studies

The presence of potential bias (such as selection bias or information bias from misclassification of exposure or outcome or confounding) in a study does not necessarily mean that the study will be excluded from the assessment. Conclusions about the evidence from each study will consider the strengths and weaknesses of the study, the direction and distortion of the biases, and the strength of the association between exposure to the substance and the cancer end point.

Bias Impact

We use a three-step approach for evaluating the impact of bias on the body of the literature. First, we assess the potential for bias as described in Section 1.3. Next, we evaluate the impact of bias of the individual study's effect estimate using the methods developed by an IARC working group, Bias Assessment in Case–Control and Cohort Studies for Hazard Identification (Berrington de Gonzalez et al. 2024). Lastly, we evaluate bias across studies (see Section 1.4). We focused our bias impact of individual studies on exposure mismeasurement or misclassification and confounding but will consider other types of biases as needed.

Exposure misclassification or mismeasurement

Misclassification of exposure in cancer epidemiology studies is often nondifferential, which generally biases risk estimates toward the null, although in some circumstances it may bias results beyond the null. Exceptions include situations such as dependent misclassification of both exposure and outcome, Berkson bias, and multi-exposure categories (NTP 2025). Except for the latter, such scenarios are unlikely to be major concerns in the GBH literature. In addition, nondifferential misclassification across multiple exposure categories may attenuate exposure–response relationships. When external validation studies are available, risk estimates can be adjusted using methods that incorporate estimates of sensitivity and specificity (Berrington de Gonzalez et al. 2024). Differential recall bias may be a concern in case-control studies and can lead to bias away from the null. However, Blair and Zahm (1990), in their review of studies of herbicide use and occupational exposure, found little evidence of substantial differential recall bias.

Confounding

We plan to conduct bias-impact assessments for individual studies in which there is concern about uncontrolled confounding, particularly when the expected direction of bias is away from the null or is uncertain and the study reports a positive finding. For each confounder not adequately addressed in the study methods, we will perform indirect adjustments using exposure–confounder prevalence data (when available) or apply quantitative bounding analyses to estimate the lower bound of the effect estimate for that confounder, consistent with guidance from the Berrington de Gonzalez et al. (2024).

Confidence in the evidence

The level of confidence in the evidence from the individual studies (rated as "moderate to strong evidence", "some evidence", "null", or "inconclusive") will be reached by considering the strength of the association, the potential for specific biases or confounding, the expected

directions of distortions, and the impact of these potential biases or unaddressed confounding on the effect estimate, and the sensitivity of the study to detect an effect. In addition to considering the potential of biases in context with strength of the findings (magnitude and exposure response relationships), confidence in a study also considers other factors such as internal consistency.

Guidelines for evaluating the overall confidence in the evidence from each study are as follows:

Moderate to strong evidence: Elevated risk estimates for GBH are found for different exposure metrics of GBH use, or in different subgroup analyses; at least one estimate has confidence intervals that do not include one. The evidence is usually reported in studies with lower potential for bias. However, the evidence may come from studies with a higher potential for bias if the potential bias is towards the null or the impact of the bias is not expected to explain all the excess risk. Evidence of an association can also come from a positive study that has low sensitivity to detect a true effect.

Some evidence: Elevated effect estimates with moderate precision are found for at least one metric of GBH use. Studies with higher potential for bias can provide evidence of an association if the potential for bias is towards the null, the impact of the bias is not expected to explain all the excess risk, or the study is positive despite having low sensitivity.

Null: Studies which are considered "null" show effect estimates of approximately ≤ 1.0.

Inconclusive: Findings vary; the overall direction of potential biases is unknown; potential confounding may explain the findings. Alternatively, studies have very low precision, and the findings may be due to chance.

1.4.2. Evidence Integration: Qualitative Assessment

Application of the RoC listing criteria to the body of studies meeting final PECO criteria on a specific substance involves evaluating (1) whether there is credible evidence for an association between exposure to the substance and cancer, and (2) whether such an observed association can be explained by chance, bias, or confounding[1]. Level-of-evidence conclusions are based on an in-depth and cohesive integration of the body of the evidence that systematically evaluates consistency and the key issues – impact of biases, sensitivity, exposure metrics, and effect modification across the studies (Arroyave et al. 2021). We plan to use triangulation methods that integrate findings from different approaches with potentially different sources of biases to evaluate the evidence. For example, consistent results from studies with different sources of biases can help strengthen the confidence of the conclusions (Lawlor et al. 2016). Finally, additional overall considerations — strength of the association, consistency across studies, evidence of an exposure-response gradient, and temporality of exposure (Bradford Hill 1965) — are used to help guide the evaluation of these questions. However, it should be noted that that these are not criteria; except for temporality, no single element is required to demonstrate causality (Rothman and Greenland 2005).

As mentioned in Section 1.2, lymphohematopoietic cancers (LHC) will be included in the database for conducting a systematic review. Of the categories of LHC, NHL has the largest

[1] The steps related to RoC listing criteria and reaching level-of-evidence conclusions will only be completed for the internal cancer hazard assessment, and will not be carried out for journal publications.

number of studies. If other categories or NHL subtypes are determined to have a sufficient number of studies, they will be considered in a systematic evaluation as well. For LHC subgroups that have a sufficient number of studies, the approach to reaching a level of evidence conclusion is as follows:

- Integrate the overall confidence of evidence judgements for the individual studies (see Section 1.4.1) to evaluate the strength of the findings and consistency for that cancer.
- Systematically evaluate key issues identified to date (e.g., using forest plots and/or text) – overall study informativeness, specific biases and key confounders, exposure metrics, effect modification, and confounders – using triangulation methods (when appropriate) across studies. For example, effect estimates from studies could be stratified by the potential for exposure misclassification or by adjustment for specific confounders.
- Consider other causality considerations (e.g., Bradford Hill guidelines) such as temporality and strength of the association (e.g., magnitude and exposure/duration response).
- Integrate the findings from the quantitative analysis for GBH and LHC cancers.
- Apply the RoC listing criteria.

Finally, for each cancer subtype, the confidence of carcinogenic hazard may be contextualized further based on the evidence.

1.4.3. Evidence Integration: Quantitative Assessment

During the scoping process, there were nine meta-analyses identified on GBH exposure and LHCs. Seven of the meta-analyses report findings on NHL that have discrepancies in findings, adding to the lack of clarity on the potential role of GBH and risk of cancer (Table 11). Several of the meta-analyses are updates or in response to prior meta-analyses. The Boffetta et al (2021) meta-analysis is an update of Donato et al. (2020) after being criticized on their selection criteria and methods in Rana et al. (2020); Rana et al. (2020) includes its own re-analysis of the Donato meta-estimates. Kabat et al. (2021) is an extension of Zhang et al. (2019) including some additional analyses by latency period. Of the more recent meta-analyses, Zhang et al. (2019) is the most comprehensive in terms of reporting and description of their methods and results. Included in the summary table below are the existing meta-analyses reporting on NHL and the meta-analysis reported in the IARC (2017) report. Differences across the meta-analyses stem from study and/or effect estimate selection (e.g. selecting single or pooled studies), model specification (e.g. fixed or random-effects models), and exposure contrast selection (e.g. ever/never, categories). Of the seven meta-analyses, though there are differences in which study was included, of the studies reporting NHL estimates from the cohort and case-control studies identified reporting risk estimates, each study seems to be included in at least one of the meta-analyses.

Due to the existing large number of meta-analyses on NHL and the number of efforts to pool together existing study data, we do not plan to conduct an additional meta-analysis in conjunction with this review.

Table 11. Findings from Published Meta Analyses on Glyphosate-Based Herbicide Exposure and NHL

Reference	Exposure comparison	N studies	Meta RR (95% CI)
Boffetta et al. 2021	Ever exposed	6	1.05 (0.90-1.24)
Boffetta et al. 2021	High exposure	3	1.15 (0.72-1.83)
Kabat et al. 2021	High exposure, 20 year lag	6	1.41 (1.13-1.76)
Kabat et al. 2021	High exposure, 15 year lag	6	1.25 (1.01-1.25)
Kabat et al. 2021	Ever exposed, no lag	5	1.05 (0.87-1.28)
Donato et al. 2020	Ever vs Never	7	1.03 (0.86-1.21)
Rana et al. 2020	Ever vs Never	7	1.14 (0.94-1.39)
Zhang et al. 2019	Highest cumulative exposure	5	1.41 (1.13-1.75)
Zhang et al. 2019	Ever exposed	6	1.30 (1.03-1.64)
Chang and Delzell 2016	Any use	6	1.3 (1.0-1.6)
Schinasi and Leon 2014	Ever	6	1.5 (1.1-2.0)
IARC 2017	Not specified	6	1.3 (1.03-1.65)

1.5. Reporting

1.5.1. Systematic Extraction of Data from the Epidemiologic Studies

Detailed information regarding study data and methods abstraction from individual studies is described in the (2025) RoC Handbook. The latest published follow-up or update for each of the cohort, nested case-control, and case-control studies are extracted for each cancer type included in the study. Additional relevant information (such as exposure data or re-analyses) from earlier and/or related publications on the same or overlapping study population(s) is also included if these publications provide unique or additional data to inform either the primary cancer study evaluation or the cancer hazard evaluation.

Data will be extracted from individual studies into a standardized data entry (such as Table Builder, Shapiro et al. 2018). The database contains fields that are specific for the various types of extracted information (such as study population characteristics, exposure and outcome assessment, analytical methods, and results). The instructions for data extraction (questions and considerations) describe the specific type of information that should be summarized or entered into each field. The fields from the database are used to populate tables for the monograph. Quality assurance of data extraction and database entry are accomplished by: (1) review of the

data entry by an independent reviewer and (2) resolution of any discrepancies by mutual discussion with reference to the original data source.

1.5.2. Reporting

We plan to include the following elements in our report:

- An overview of study characteristics (e.g., population characteristics, exposure assessment methods, outcomes) included in the review, even if not included in the evidence integration for bias, quality, or other reasons.
- A discussion of biases and limitations for each bias domain across studies, in addition to the rationale for the risk-of-bias at the study level.
- A scientific narrative of the interpretation of study findings including a discussion of the confidence in the evidence of each study, heterogeneity across studies (not limited to potential for biases) and the rationale for the conclusion (e.g., consideration of dose-response relationships, consistency, ruling out chance, bias and confounding).
- Findings from studies – reported in summary tables and graphed in forest plots (if appropriate.
- Preliminary level of evidence conclusions for cancers, Non-Hodkin Lymphoma and other lymphohematopoietic cancers.

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Appendix A. Roles and Responsibilities

A.1. Evaluation Team:

Evaluation teams are composed of federal staff and contractor staff. Procedures are in place to avoid actual or perceived conflicts of interest. Members of the evaluation team have experience or training in conducting literature searches and/or evaluating occupational and environmental epidemiology studies.

A.1.1. NIEHS Core Project Leaders

Develops research concept, rationale, and framework; serves as a researcher

- Ruth Lunn, DrPH [overall project]
- Suril Mehta, DrPH [overall project]
- Sabah Quraishi, PhD [human cancer]
- Matthew Urich, PhD [animal cancer]

A.1.2. Team Members

Responsibilities	Member	Affiliation
Overall		
Project management	Whitney Arroyave, PhD	Inotiv
Develop search strategy, conduct literature searches, and manage literature	Rachael Kalsch, MLIS	Inotiv
Literature management, document preparation, visualization	Tracy Saunders, BS	Inotiv
Investigation, writing, review and editing	Bevin Blake*, PhD	NIEHS
Animal cancer assessment		
Researcher: toxicological expertise, study informativeness assessment, data extraction, writing, critical review	Ezazul Haque, PhD John Bucher, PhD Matthew Urich, PhD	NIEHS
	Danila Cuomo, PhD	Inotiv
Human Cancer Assessment		
Researcher: Epidemiological expertise, study informativeness assessment, data extraction, writing, critical review	Ruth Lunn, DrPH Suril Mehta, DrPH Sabah Quraishi, PhD	NIEHS
	Whitney Arroyave, PhD	Inotiv

*Joined the evaluation team after the protocol was finalized, however was involved in the assessment and report writing.

A.2. Technical Advisors and Reviewers

Expertise or Role	Member	Affiliation
Statistical analyses	Keith Shockley	NIEHS

Expertise or Role	Member	Affiliation
Pathology	Sonika Patial, PhD, DVM	NIEHS
Protocol reviewer (animal cancer assessment)	Chad Blystone, PhD Kristen Ryan, PhD Amy Wang, PhD	NIEHS
Protocol reviewer (human cancer assessment)	Kyla Taylor, PhD Katie O'Brien, PhD	NIEHS

Appendix B. Citation Database Search Terms

Pubmed

((((("glyphosate"[nm] OR "1071-83-6"[tw] OR "(Carboxymethylamino)methylphosphonic acid"[tw] OR "Carboxymethylaminomethanephosphinic acid"[tw] OR "C-K Yuyos FAV"[tw] OR "CP 67573"[tw] OR "Folusen"[tw] OR "Forsat"[tw] OR "Glialka"[tw] OR "Glifoglex"[tw] OR "Glifosan 747"[tw] OR "gliphosate"[tw] OR "Gliz"[tw] OR "Glyfos"[tw] OR "GlyGran"[tw] OR "Glyphodin A"[tw] OR "Glyphomax"[tw] OR "Glyphosate"[tw] OR "Glyphosphate"[tw] OR "Ground Bio"[tw] OR "Herbatop"[tw] OR "HM 2028"[tw] OR "Kickdown"[tw] OR "Lancer herbicide"[tw] OR "MON 2139"[tw] OR "MON 3539"[tw] OR "MON 6000"[tw] OR "N-(Phosphonomethyl)glycine"[tw] OR "N-(phosphonomethyl)-Glycine"[tw] OR "N-Phosphomethylglycine"[tw] OR "NPhosphonomethylglycine"[tw] OR "Phorsat"[tw] OR "Phosphonomethylglycine"[tw] OR "Phosphonomethyliminoacetic acid"[tw] OR "Pondmaster"[tw] OR "Rebel Garden"[tw] OR "Roundup Max"[tw] OR "Safal"[tw] OR "Scout herbicide"[tw] OR "Silglif"[tw] OR "yerbimat"[tw] OR "Roundup"[tw] OR "34494-03-6"[tw] OR "MON 0459"[tw] OR "40465-66-5"[tw] OR "MON 14420"[tw] OR "MON 8750"[tw] OR "Roundup Hi-Load"[tw] OR "Roundup PRODry"[tw] OR "70393-85-0"[tw] OR "MON 8000"[tw] OR "Monsanto 8000"[tw] OR "Polado"[tw] OR "Trisodium hydrogen bis(N-(phosphonatomethyl)aminoacetate"[tw] OR "39600-42-5"[tw] OR "Glyphosate potassium"[tw] OR "Glyphosate monopotassium salt"[tw] OR "Glyphosate potassium"[tw] OR "Glyphosate-potassium"[tw] OR "Monopotassium glyphosate"[tw] OR "Roundup Attack"[tw] OR "Roundup Energy"[tw] OR "Roundup Maxload"[tw] OR "Roundup Original Max"[tw] OR "Roundup Power Max"[tw] OR "Roundup Ultramax II"[tw] OR "Roundup Weathermax"[tw] OR "Touchdown Forte HiTech"[tw] OR "Transorb R"[tw] OR "Weathermax"[tw] OR "Zapp Qi"[tw] OR "70901-12-1"[tw] OR "Glyphosate-potassium"[tw] OR "Potassium glyphosate"[tw] OR "Potassium N-(phosphonomethyl)glycine"[tw] OR "Uragan Forte"[tw] OR "VisionMAX"[tw] OR "N-(phosphonomethyl)glycine potassium salt"[tw] OR "114370-14-8"[tw] OR "Glyphosate ammonium"[tw] OR "N-(phosphonomethyl)glycine ammonium salt"[tw] OR "69254-40-6"[tw] OR "Glyphosate-diammonium"[tw] OR "Diammonium N-(phosphonomethyl)glycine"[tw] OR "N-(phosphonomethyl)glycine diammonium salt"[tw])) OR (("glyphosate, isopropyl amine salt"[nm] OR "N-(phosphonomethyl)glycine trimethylsulfonium salt"[nm] OR "38641-94-0"[tw] OR "Glyphosate-isopropylammonium"[tw] OR "Glyphosate isopropylamine salt"[tw] OR "Azural AT"[tw] OR "CP 70139"[tw] OR "Fosulen"[tw] OR "Glifosato estrella"[tw] OR "Glycel"[tw] OR "Glycine, N-(phosphonomethyl)-, cmpd with 2- propanamine (1:1)"[tw] OR "Glyfos AU"[tw] OR "Glyfos BIO"[tw] OR "Glyphosate isopropylamine salt"[tw] OR "Glyphosate mono(isopropylamine) salt"[tw] OR "Glyphosateisopropylammonium"[tw] OR "Glyphosate-mono(isopropylammonium)"[tw] OR "Landmaster"[tw] OR "MON 139"[tw] OR "MON 39"[tw] OR "N-(Phosphonomethyl)glycine isopropylamine salt"[tw] OR "N-(Phosphonomethyl)glycine isopropylammonium salt"[tw] OR "N-(Phosphonomethyl)glycine monoisopropylamine salt"[tw] OR "Nitosorg"[tw] OR "Rondo"[tw] OR "Utal"[tw] OR "Utal (herbicide)"[tw] OR "Vision (herbicide)"[tw] OR "2- Propanamine, compd, with N-(phosphonomethyl)glycine (1:1)"[tw] OR "Glycine, N-(phosphonomethyl)-, compd. with 2-propanamine (1:1)"[tw] OR "N-(Phosphonomethyl)glycine, compound with 2-propylamine (1:1)"[tw] OR "Isopropylamine glyphosate"[tw] OR "81591-81-

3"[tw] OR "Glyphosate-trimesium"[tw] OR "Glyphosphate-trimesium"[tw] OR "Avans 330"[tw] OR "Glyphosate mono(trimethylsulfonium) salt"[tw] OR "Glyphosate trimethylsulfonium salt"[tw] OR "Glyphosate-trimesium"[tw] OR "Medallon"[tw] OR "Ouragan"[tw] OR "R 50224"[tw] OR "SC 0224"[tw] OR "Sulfosate"[tw] OR "Sulphosate"[tw] OR "Touchdown herbicide"[tw] OR "Trimethylsulfonium carboxymethylamino-methylphosphonate"[tw] OR "Trimethylsulfonium glyphosate"[tw] OR "Glycine, N-(phosphonomethyl)-, ion(1-), trimethylsulfonium"[tw] OR "Sulfosate"[tw]))) OR (("Aminomethylphosphonic acid" OR (AMPA and metabolite)) OR ("N-acetyl AMPA") OR ("N-acetyl glyphosate")))

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ts=((((("glyphosate" OR "1071-83-6" OR "(Carboxymethylamino)methylphosphonic acid" OR "Carboxymethylaminomethanephosphinic acid" OR "C-K Yuyos FAV" OR "CP 67573" OR "Folusen" OR "Forsat" OR "Glialka" OR "Glifoglex" OR "Glifosan 747" OR "gliphosate" OR "Gliz" OR "Glyfos" OR "GlyGran" OR "Glyphodin A" OR "Glyphomax" OR "Glyphosate" OR "Glyphosphate" OR "Ground Bio" OR "Herbatop" OR "HM 2028" OR "Kickdown" OR "Lancer herbicide" OR "MON 2139" OR "MON 3539" OR "MON 6000" OR "N-(Phosphonomethyl)glycine" OR "N-(phosphonomethyl)-Glycine" OR "N-Phosphomethylglycine" OR "NPhosphonomethylglycine" OR "Phorsat" OR "Phosphonomethylglycine" OR "Phosphonomethyliminoacetic acid" OR "Pondmaster" OR "Rebel Garden" OR "Roundup Max" OR "Safal" OR "Scout herbicide" OR "Silglif" OR "yerbimat" OR "Roundup" OR "34494-03-6" OR "MON 0459" OR "40465-66-5" OR "MON 14420" OR "MON 8750" OR "Roundup Hi-Load" OR "Roundup PRODry" OR "70393-85-0" OR "MON 8000" OR "Monsanto 8000" OR "Polado" OR "Trisodium hydrogen bis(N-(phosphonatomethyl)aminoacetate" OR "39600-42-5" OR "Glyphosate potassium" OR "Glyphosate monopotassium salt" OR "Glyphosate potassium" OR "Glyphosate-potassium" OR "Monopotassium glyphosate" OR "Roundup Attack" OR "Roundup Energy" OR "Roundup Maxload" OR "Roundup Original Max" OR "Roundup Power Max" OR "Roundup Ultramax II" OR "Roundup Weathermax" OR "Touchdown Forte HiTech" OR "Transorb R" OR "Weathermax" OR "Zapp Qi" OR "70901-12-1" OR "Glyphosate-potassium" OR "Potassium glyphosate" OR "Potassium N-(phosphonomethyl)glycine" OR "Uragan Forte" OR "VisionMAX" OR "N-(phosphonomethyl)glycine potassium salt" OR "114370-14-8" OR "Glyphosate ammonium" OR "N-(phosphonomethyl)glycine ammonium salt" OR "69254-40-6" OR "Glyphosate-diammonium" OR "Diammonium N-(phosphonomethyl)glycine" OR "N-(phosphonomethyl)glycine diammonium salt")) OR (("glyphosate, isopropyl amine salt" OR "N-(phosphonomethyl)glycine trimethylsulfonium salt" OR "38641-94-0" OR "Glyphosate-isopropylammonium" OR "Glyphosate isopropylamine salt" OR "Azural AT" OR "CP 70139" OR "Fosulen" OR "Glifosato estrella" OR "Glycel" OR "Glycine, N-(phosphonomethyl)-, cmpd with 2- propanamine (1:1)" OR "Glyfos AU" OR "Glyfos BIO" OR "Glyphosate isopropylamine salt" OR "Glyphosate mono(isopropylamine) salt" OR "Glyphosateisopropylammonium" OR "Glyphosate-mono(isopropylammonium)" OR "Landmaster" OR "MON 139" OR "MON 39" OR "N-(Phosphonomethyl)glycine isopropylamine salt" OR "N-(Phosphonomethyl)glycine isopropylammonium salt" OR "N-(Phosphonomethyl)glycine monoisopropylamine salt" OR "Nitosorg" OR "Rondo" OR "Utal" OR "Utal (herbicide)" OR "Vision (herbicide)" OR "2- Propanamine, compd, with N-

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Scopus

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