Partnership for Work, Health and Safety

Occupational Asbestos Exposure and Gastrointestinal Cancers: Systematic Review and Meta-analysis

Report to The Workplace Safety and Insurance Board (WSIB) Ontario

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About the Partnership for Work, Health and Safety

The Partnership for Work, Health and Safety (PWHS) is an innovative research unit in the University of British Columbia School of Population and Public Health that combines rigorous work and health research with effective knowledge translation. PWHS brings together policy-makers, researchers and data resources from national and international organizations to address current and emerging issues of work-related health. Our research is aimed at improving understanding of the causes and consequences of injuries and illness, identifying high-risk industries and occupations, and investigating the effectiveness of interventions that improve worker health, prevent occupational illness and injury, and reduce work-related disability. Our collaboration, based on best practices of knowledge transfer, enables researchers and decision-makers to work together to identify relevant questions, understand data, and produce useful information to effectively inform policy and practice.

Disclosures

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Executive Summary

The purpose of the systematic review and meta-analyses was to summarize the epidemiological evidence on the association between occupational exposure to asbestos and the risk of gastrointestinal (GI) cancers.

Primary Research Questions

- Does occupational asbestos exposure increase the risk of esophageal, stomach or colorectal cancer?
- Is there an exposure-response relationship for studies with detailed exposure assessment characteristics (e.g., high/low exposure categories)?
- Is there an exposure-response relationship for sub-groups of workers by occupation/industry?
- Does the risk of esophageal, stomach or colorectal cancer co-vary with the risk of other asbestos-related cancers, specifically lung cancer?

Secondary Research Questions

- Is the risk of esophageal, stomach or colorectal cancer associated with specific types of asbestos?
- Is there a synergistic or antagonistic effect of asbestos exposure with other GI cancer risk factors (smoking, alcohol consumption)?

Methods

The systematic review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO). Eligible scientific studies were identified using a search strategy developed by the investigators with expertise in occupational hygiene, exposure assessment, cancer epidemiology, and systematic review methods, and a university health librarian. The search strategy was applied to the MEDLINE, Web of Science, Embase, CINAHL and Scopus databases.

The review was restricted to cohort and case-control studies. Studies were included if they reported on a statistical association between occupational asbestos exposure and the risk of GI cancers. Searches were not limited by publication year, country/region, or language.

Reported effect estimates (e.g., ORs, HRs, SIRs, and SMRs) were assumed to be equivalent to RRs and presented in forest plots with corresponding meta-risk estimates using established statistical methods. A random effects model was used to account for study heterogeneity. Sensitivity analyses were performed to investigate the robustness of the findings to methodological and analytical decisions.

Two full investigator meetings were convened to refine methods and analyses, to review and interpret the meta-risk estimates, and to seek consensus on the evidence.

Results

We found evidence of an elevated risk of esophageal cancer, stomach cancer and colorectal cancer with occupational exposure to asbestos. There was consistency and higher meta risk estimates in the analyses of studies where there was better exposure assessment and increased confidence in the categorization of high asbestos exposure, including among workers in exposure-response studies (high/low contrast); among workers with a history of significant exposure as a result of the nature of their work (e.g., insulators and insulating manufacturing workers); and among workers in cohorts where there was also a two-fold or greater increased risk of asbestos-related lung cancer as an strong indicator of high exposures. There was heterogeneity in the studies included in the review, although results from sensitivity analyses indicate that there was minimal influence from any one study on the overall meta-estimates or from publication bias. Unexplained heterogeneity was reduced, and the strength of association increased, in the sub-group analyses of studies where there was better asbestos exposure assessment and increased confidence in the categorization of high exposed workers. Further, the consistency of an increased risk of GI cancers with occupational asbestos exposure was robust to multiple sensitivity analyses that investigated the impact of the systematic review and meta-analyses methods. Further research is needed on GI cancers and asbestos fibre type and on effect modification of the association by other occupational and non-occupational factors.

Conclusions

The evidence synthesis, as summarized above, supports a causal link between occupational asbestos exposure and esophageal, stomach and colorectal cancer.

Lay Summary

A systematic review of epidemiology studies found evidence of an increased risk of esophageal cancer, stomach cancer and colorectal cancer with occupational exposure to asbestos. There was stronger evidence of this relationship where there was better exposure assessment and increased confidence in the categorization of significant asbestos exposure in studies, including among the highest exposed workers; among workers with a history of significant exposure as a result of the nature of their work (e.g., asbestos-related insulation); and among workers in cohorts where there was also a two-fold or greater increased risk of asbestos-related lung cancer as a strong indicator of exposure.

Background and Rationale

Historically, Canada was a major producer and exporter of asbestos. Most recently, approximately 410,000 tonnes of asbestos were produced between 2008 and 2010, accounting for approximately 6% of the total global production during that period (1). Today, following the 2018 federal ban on the manufacture of asbestos, the majority of occupational exposure to asbestos is associated with the use or maintenance of products that contain asbestos (e.g., automotive brake repair, ship repair); or the renovation, remediation and abatement of buildings and building materials that contain asbestos (i.e., asbestos was used in over 3,000 different building materials such as stucco, flooring, roof shingles, and insulation) (1,2). In 2016, CAREX Canada estimated that approximately 235,000 Canadians were exposed to asbestos in the workplace as a result of contact with asbestos-containing products (3). In Canada today, the largest exposed industrial groups are specialty trade contractors, followed by building construction, automotive repair, and shipbuilding (3).

Asbestos is known to cause lung cancer and mesothelioma, and the Burden of Occupational Cancer in Canada project (4), including work by the investigators, estimated that historical occupational exposure to asbestos leads to approximately 1,900 lung cancers and 430 mesothelioma cases each year (5). Most of these asbestos-related cancers occur among workers who had been employed in the manufacturing and construction sectors (4, 5). Epidemiological evidence for occupational asbestos exposure as a cause for other cancer sites is viewed as limited compared to that of lung cancer and mesothelioma.

Two Authoritative Reviews of the Evidence on Asbestos and Cancer

The Monograph on Asbestos by the World Health Organization's International Agency for Research on Cancer (IARC) (2009) (6), and a prior systematic review by the US National Academy of Sciences' Institute of Medicine (IOM) Committee on Asbestos (2006) (7) are the two most authoritative summaries of evidence on asbestos disease epidemiology. A summary of the evaluations of the IOM and IARC is provided below (Table 1).

The IOM Committee that met in 2006 completed a systematic review and conducted meta-analyses on five cancer sites: larynx, pharynx, stomach, colorectal, and esophageal. The Committee did not review mesothelioma, lung cancer, or ovarian cancer. The IOM Committee concluded in 2006 that there was sufficient evidence for asbestos exposure as a cause of laryngeal cancer, suggestive evidence for stomach and colorectal cancer, and inadequate evidence for esophageal cancer (7).

The IARC Epidemiological Working Group on asbestos, which met in 2009, systematically reviewed the evidence for a broad range of cancer sites (but did not conduct meta-analyses). The IARC Working Group concluded that there was sufficient evidence for the carcinogenicity of all forms of asbestos with mesothelioma, and with cancer of the lung, larynx and ovary (6). In regards to GI cancers, the IARC Working Group found that 'positive associations have been observed between exposure to all forms of asbestos and

Table 1 | Evidence for other asbestos-related cancers, including gastrointestinal sites

Cancer Site	IOM* Systematic Review Meta Analyses	IARC** Systematic Review
Larynx	Sufficient	Sufficient
Pharynx	Suggestive	Limited
Stomach	Suggestive	Limited
Colorectal	Suggestive	Limited
Esophagus	Inadequate	Inadequate
Ovary	Not reviewed	Sufficient

**U.S. Institute of Medicine. Asbestos: Selected Cancers, 2006.*
*** International Agency for Research on Cancer, Monograph 100C Working Group, 2009.*

cancer of the pharynx, stomach, and colorectum' (6), including associations between prolonged and heavy asbestos exposure and cancer of the stomach (8-12) and the colorectum (13-16), and exposure-response relationships for stomach cancer in cohort studies with high quality exposure assessments methods (17-20). The IARC Working Group "was evenly divided as to whether the evidence was strong enough to warrant classification as sufficient" for colorectal cancer (6). In sum, the IARC Working Group concluded in 2009 that there was limited evidence for asbestos exposure as a cause for stomach and colorectal cancers, and inadequate evidence for esophageal cancer.

Additional Evidence and Systematic Reviews

The Finnish Institute for Occupational Health (FIOH), in collaboration with the International Commission on Occupational Health, convened an international group of experts in 2014 to update the Helsinki Criteria for Diagnosis and Attribution of Asbestos, Asbestosis and Cancer. This work focused on updating CT screening, pathology and biomarkers for diagnosing asbestos-related disease and for the follow-up of asbestos-exposed workers for non-malignant asbestos disease; but also included an update of the evidence for asbestos-related disease since the IARC review in 2009 (21). In terms of GI cancers, the FIOH consensus was in accordance with that of the IARC review, that studies generally provide consistency of evidence of an increased risk of stomach and colorectal cancers with asbestos exposure, especially for heavy and long duration exposures, but that the evidence was not definitive for a causal relationship (21).

In 2015, Peng and colleagues published a systematic review and meta-analysis (22) of 32 cohort studies of stomach cancer mortality and concluded that there was an overall elevated risk associated with asbestos exposure (meta-risk estimate (mRE)=1.19 (95% CI 1.06-1.34)). Also in 2015, Fortunato and Rushton (23) published a meta-analysis of 40 mortality and 15 incidence cohort studies of stomach cancer and found that there was an overall elevated risk associated with occupational asbestos exposure (mRE=1.15 (95% CI 1.03-1.27)), with stronger associations in sub-group-analyses of studies with an increased risk of asbestos-related lung cancer (mRE=1.46 (95% CI 1.22-1.77)), and among generic asbestos workers

(mRE=1.41 (95% CI 1.10-1.82) and insulators (mRE=1.27 (95% CI 1.05-1.53)). In 2019, Kwak and colleagues published a systematic review and meta-analysis (24) of 46 studies of colorectal cancer mortality and concluded that there was an overall increased risk associated with asbestos exposure (mRE=1.16 (95% CI 1.05-1.29)), and stronger associations in sub-group-analyses of studies in which there was an increased risk of asbestos-related lung cancer (mRE=1.44 (95% CI 1.29-1.60)) and of workers in the insulation industry (mRE=1.49 (95% CI 1.26-1.75)). Finally in 2021, Wu and colleagues published a systematic review and meta-analysis (25) of 34 cohort studies of esophageal cancer and concluded that there was an overall elevated risk associated with asbestos exposure (mRE= 1.28 (95% CI 1.19-1.38)), and stronger associations for the highest asbestos exposure groups (mRE =1.84 (95% CI 1.27–2.69)), and for asbestos-related textile (mRE=1.45 (95% CI 1.13, 1.86) and shipyard workers (mRE=1.39 (95% CI 1.15–1.68).

The Rationale for a Systematic Review/Meta-Analysis

A scan of the scientific literature provided the rationale for an updated systematic review of the evidence for asbestos exposure and GI cancers given the publication of many more relevant papers since the time of the IARC and IOM reviews approximately 12 and 15 years ago, respectively; as well as the additional evidence from the systematic reviews published subsequently by Peng, Fortunato and Rushton, Kwak, and Wu on colorectal, stomach and esophageal cancer, respectively. The prior authoritative IARC and IOM reviews were not able to draw strong conclusions and, in one case, no consensus on the level of evidence for esophageal cancer.

Further to the need to update the evidence with the most recent studies, this systematic review and meta-analyses adopted a comprehensive search strategy that was inclusive of all epidemiological studies to maximize the data points available for the research questions on occupational exposure to asbestos and GI cancers. The current review and meta-analyses also included a critical exposure assessment approach by investigators with training and expertise in occupational (cancer) epidemiology and hygiene/exposure assessment, and included studies in sub-group analyses based on confidence in significant exposure to asbestos, similar to that of the IARC review (6) and as advocated for by experts in cancer epidemiology (26). Prior systematic reviews have excluded or downgraded many relevant studies on the basis of inappropriate methodological quality assessments (27), including for exposure methods (26, 28, 29). Traditional quality assessment approaches, developed for randomized controlled studies, pay little attention to exposure assessment methods that are a key component in observational studies and for reviews of evidence focused on occupational asbestos exposure (26, 28). The current approach of an expert-informed assessment of exposure methods is consistent with calls that increased weight be given to exposure assessment and study ‘informativeness’ in reviews of evidence of exposures and cancers (30).

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Methods

Research Team

The research team was comprised of 11 subject-matter experts in the areas of occupational (cancer) epidemiology and occupational hygiene/exposure assessment. Team members had additional expertise in systematic review and meta-analyses methods. All of the investigators have published studies on occupational exposure to asbestos and/or occupational cancer risks, and several have participated in the IARC Monographs Program, as well as on the IOM panel and FIOH report. Several investigators are part of international working groups looking at a critical review of risk of bias approaches in epidemiological studies, including enhanced consideration of the quality of occupational exposure assessments, and on the improvement of the criteria for the inclusion of relevant studies in evidence reviews.

Protocol and Registration

The systematic review and meta-analyses of the association between occupational asbestos exposure and GI cancers was conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (31, 32), and according to the best practices of the Cochrane Collaboration that publishes meta-analyses on health and medical topics (33). The review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO) (#CRD42022282524 – Review Ongoing (34)). PROSPERO is an open access, online database of health-related protocols where key methodological elements of the review are recorded and transparent (35).

Search Strategy

Eligible scientific studies on occupational asbestos exposure and risk of GI cancers were identified using search strategies developed by the investigators in collaboration with the health librarian at the University of British Columbia (36), and reviewed with representatives of the Workplace Safety and Insurance Board's research and policy units (2021-10-13).

Four search strategies were developed as follows:

1. Cohort studies search strategy included asbestos AND cohort study design AND cancer keyword terms. Specific GI cancer sites were not included as keywords in this search strategy as these sites are not always mentioned in the titles, abstracts or subject headings for occupational cohort studies.
2. Case-control studies search strategy included occupation exposure(s) AND case-control study design AND GI cancer keyword terms. Asbestos-related keywords were not included in this search strategy as specific exposures are not always mentioned in the titles, abstracts or subject headings of occupational case-control studies.
3. Additional search strategy included asbestos AND GI cancers keyword terms. Specific study designs were not included in this search strategy as they are not always mentioned in the titles, abstracts or

subject headings of occupational epidemiological studies. However, studies tagged as case reports, editorials and reviews in the subject heading field were excluded.

4. References lists for studies included in the current review and for the IOM 2006 (7), IARC 2009 (6), FIOH 2014 (7) and other published systematic reviews (22-25) were reviewed for additional eligible studies and to ensure all relevant papers were captured.

Search Terms and Databases

Appendix I provides a full list of search terms. The following is a list of terms (keywords and MeSH subject headings) included in the search strategy for MEDLINE via OVID as an example. Equivalent searches were also conducted with search terms tailored to Web of Science, Embase, CINAHL and Scopus databases.

Asbestos

- asbestos OR crocidolite* OR amosite* OR chrysotile* OR tremolite* OR actinolite* OR anthophyllite* (this line identifies asbestos as defined by keyword terms) OR
- exp asbestos/ (this line identifies asbestos as indexed by subject headings)

GI cancer

- exp gastrointestinal neoplasms/ (this line identifies GI cancers as indexed by subject headings) OR
- (exp neoplasms/ OR cancer* OR malignan* OR tumo?r* OR neoplas* OR (other variations)) AND (esophagus OR oesophagus OR stomach OR colon OR rectal OR colorectal OR (other variations)) (this line identifies GI cancers using a combination of cancer AND GI body part search terms)

Occupational exposures

- exp occupational exposure/ OR
- exp occupational diseases/ OR
- exp manufacturing industry/ OR
- exp construction industry/ OR
- ((work or worker* or working or job or employ* or occupation*) ADJ (exposure* or related* or environment? or site? or place? or population? or cohort? or sample?))
- construction* ADJ5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)

Cohort studies

- cohort studies/ OR
- cohort* OR longitudinal* OR follow up

Case-control studies

- case-control studies/ OR
- (case* ADJ10 control*) OR
- (case* ADJ10 referent*) OR
- (case* ADJ cohort*) OR
- (case* ADJ3 nested*)

Screening and Inclusion Criteria

All unique citations identified across the four search strategies and databases were randomly assigned to pairs of investigators with epidemiology and hygiene/exposure assessment expertise for title/abstract and full-text screening. Both title/abstract and full-text screenings were completed using Covidence. Covidence is a web-based software platform that supports standardized procedures for citation screening, as well as for extraction of study characteristics, and the export of data points and references (37). Investigators were provided with a Covidence training tutorial including a sample screening assignment with inclusion criteria, followed by a meeting to reconcile procedures and conflicts. Reviewer disagreements on the inclusion of a citation in the systematic review were resolved by re-review and consensus.

Non-English citations (n=84) were screened for inclusion by investigators with French, German, Italian, Spanish and Japanese language proficiencies, or translated to English using Google Translate. A total of 28 non-English studies were included in the systematic review and meta-analyses (56 excluded—36 did not meet the inclusion criteria, 14 were duplicate records and 6 had no full-text paper available/retrievable). An English version of a non-English citation was also often published (n=18 of 30). Independent of publication language, the most informative effect estimate was extracted for meta-risk analyses following specified criteria (see criteria below).

Study Inclusion Criteria

- Original, epidemiological study investigating association(s) between occupational asbestos exposure and risk of GI cancers;
- Case-control or cohort study design (including nested case-control and SIR/SMR/PMR studies), without restriction to publication year, country/region, or language;
    - » Cohort studies had to include workers with asbestos exposure and case-control studies a measure (or metric) of asbestos exposure;
- Quantitative result(s) (e.g., relative risk, odds ratio, hazard ratio, standardized mortality ratio, standardized incidence ratio) of the association between measure(s) of occupational asbestos exposure and GI cancer outcomes among humans.

Data Extraction and Analytic Variables

A data extraction template was developed based on the WHO International Agency for Research on Cancer (IARC) Table Builder (38) and refined in consultation with the investigators for a systematic review specific to occupational asbestos exposure and GI cancers. The Table Builder is designed to assist with the construction of evidence tables for drawing conclusions on the risk factors that increase the risk of human cancer.

Data was extracted from the included studies by two highly qualified research personnel with graduate-level training in epidemiology and hygiene, respectively: first author, publication year, country, study design, sample size, observed/expected cases, comparison or reference population, follow-up period, occupational asbestos exposure characteristics (measurement methods, any/none, high/low, occupation/industry, fibre type), GI cancer characteristics (data sources, incidence/mortality, type of GI cancer), measures of association with confidence intervals (risk ratios (RRs), odds ratios (ORs), hazard ratios (HRs), standardized mortality ratios (SMRs), standardized incidence ratios (SIRs), population mortality risks (PMRs), measures of association for asbestos-related lung cancer, and confounder adjustments. Appendix II provides a summary table of studies included in the meta-risk analyses by GI cancer site.

GI cancer risk and significant asbestos exposure was analyzed in a sub-group meta-analysis of cohort studies with reported risk estimates for workers with a history of asbestos-related insulation, mining, and cement occupations/industries (plus a category for all other occupations/industries groupings included in cohort studies).

GI cancer risk and significant asbestos exposure was analyzed in a sub-group meta-analyses of cohort and case-control studies with exposure-response risk estimates for the highest exposed workers within the exposed population (internal comparisons). Highest exposure across studies represented workers in categories with the longest duration, greatest probability, highest intensity or highest cumulative levels of asbestos exposure. These studies also tended to be based on more detailed exposure assessment methods, including some combination of employment records/work history, direct measurements, job exposure matrices and/or expert opinions (see Appendix III for exposure-response studies included in analyses of esophageal cancer by assessment characteristics, for example). Highest asbestos exposure categories were independently defined within a study. Highest asbestos exposure was not defined or derived by the current investigators using specific cut-points for a single exposure characteristic.

GI cancer risk and significant asbestos exposure was analyzed in a sub-group meta-analyses of cohort studies that also reported asbestos-related lung cancer risk estimates in the same cohort. A two-fold or higher risk of asbestos-related lung cancer was defined as a strong indicator of significant asbestos exposure within the same cohort of workers for an established exposure-response relationship.

Categorization of asbestos fibre type was derived from information reported in cohort studies and/or by retrieving historical information from prior publications on the same cohort. The categorization (amphibole, chrysotile, mixed amphibole and chrysotile, unknown/unspecified fibre type) was reviewed by the investigators based on their knowledge of the geographic location and the period of occupational exposure for the study, cohort and/or mine.

The outcomes were categorized as esophageal, stomach and colorectal cancer. The majority of included studies reported risk estimates for colon and rectal cancers combined, often because these two cancer sites were assumed to have similar risk profiles, or because of partial misclassification between the two sites and relatively rare rectal cancers.

Statistical Analyses

All analyses were conducted using Stata statistical software, version 17.0 (39) and in accordance with best practices for meta-analyses by the Cochrane Organization (40). Extracted risk estimates (ORs, HRs, SIRs, and SMRs) were assumed to be equivalent to RRs (for relatively rare events) and natural log transformed for the meta-risk analyses (41). Overall and sub-group meta-risk estimates, with associated 95% confidence intervals (CIs), were obtained using random-effects models to account for study heterogeneity in the estimates and a restricted maximum likelihood (REML) method to estimate the variance component parameters (40, 42, 43).

Missing relative risk estimates for studies were computed using the reported observed and expected count data. Stratified estimates (e.g., by occupation) were combined into an overall risk estimate by summing the observed and expected counts and re-calculating the estimate with confidence intervals. Missing confidence intervals were computed using the -eclpci- command in Stata that assumes a Poisson distribution. For stratified estimates that could not be combined, both estimates were included in the meta-analyses. Two studies reported zero exposed cases for esophageal cancer (Peto, 1985; Anderson 1993). Sensitivity analyses were conducted by including these studies with a case count of '1'.

Meta-risk estimates were generated for the overall association between occupational asbestos exposure (any/none) and esophageal, stomach and colorectal cancer. Sub-group meta-analyses were conducted with increasing information and confidence in significant asbestos exposure defined by a) work history in asbestos-related insulation, mining or cement occupations and/or industries; b) a two-fold or higher risk of asbestos-related lung cancer in the same cohort; and c) highest exposure-response comparisons.

Tests for heterogeneity (40, 44) were performed to quantify the degree of inconsistency between study results (Q, T^2 and I^2 statistics). The Q statistic represents the ratio of observed variation to the within study error but the calculation is sensitive to the number of studies. The T^2 statistic is the Q statistic but without

the number of studies in the calculation. The I^2 is the primary measure used in epidemiology and health-related sciences for assessing heterogeneity in meta-analyses.[1]

The I^2 statistic is derived from the Q statistic but expressed as a proportion of observed to true variance (44). It is not a value on an absolute scale and it does not provide a measure of the dispersion of effects.[1] An I^2 value near 0% means that most of the observed variance is random, based on the extent of the overlap of confidence intervals around the study estimates (not that the effect estimates are within a narrow range). An I^2 value near 75% conversely tells us that 75% of the variance in observed effects reflects variance in the true effects. Guidance by the Cochrane Organization (44) suggest that values of 0% to 40% indicate little variance in the observed to true estimates, 30% to 60% moderate variance, 50% to 90% substantial variance, and 75% to 100% considerable variance.

Sensitivity analyses were conducted to investigate bias; and to investigate the robustness of the meta-risk estimates to the inclusion of any one study, the addition of studies over time, and the inclusion of different data points when multiple points were available within a study or across a study over time.

Evidence Synthesis Approach

A two-day meeting of the investigators and collaborators was convened to a) review the database of included studies for comprehensiveness (i.e. potential errors/omissions/duplicates), b) review preliminary meta-risk-estimates to identify analytical areas for refinement, c) develop decisions rules for the selection of a preferred risk estimate as the most informative for the overall and sub-group meta-analyses when multiple estimates were available within a study or for a cohort/study over time, and d) seek consensus on a hierarchy of evidence based on the informativeness of the exposure assessment methods.

Only one independent data point per study or cohort, or per strata for sub-group analyses, was included in each meta-risk analysis. Decision rules for the inclusion of one risk estimate as the most informative to a meta-risk analysis included preference for the following:

- the risk estimates for a specific cancer site (esophageal, stomach, colorectal) versus all GI sites combined;
- the incidence verses mortality relative risk estimate;
- the risk estimate based on the longest follow-up period;
- the risk estimate based on censoring at the last known date alive versus a cut-off date;
- the SMR or SIR estimates based on regional versus national population reference rates;
- the mortality risk estimate based on death certificates versus other data sources;
- a collapsed or stratified SMR/SIR estimate(s) if no overall estimate of association was provided;

[1] Bornstein M (2022). Commentary. In a meta-analysis, the I-squared statistic does not tell us how much the effect size varies Journal of Clinical Epidemiology, 2022; In press, Available on-line Oct 9, 2022: https://doi.org/10.1016/j.jclinepi.2022.10.003

- the risk estimate based on the longest latency period (providing studies measured latency and stratified estimates by latency periods).
    - » True latency is the period of time between when the cancer occurs (or is initiated) and when it is detected (diagnosis for incidence studies, death for mortality studies). For chronic diseases, such as cancer, it is not possible to directly measure true latency. Traditionally, years since first exposure has been used in occupational cancer studies as a measure of latency but includes both the time period that an exposure to a carcinogen has its effect (the induction period) and the latency period. A modern approach to estimate latency is to lag exposure—that is to assume a latency interval and ignore exposure that occurs during that period. For example, if a study lags by 10 years, only asbestos exposure that occurs at least 10 years earlier is counted in assessing dose-response.

Three included papers (Ferrante 2017, Luberto 2019, Magnani 2020) were based on 43 pooled Italian cohorts. Our systematic review identified 22 papers that analyzed data from eight of these underlying 43 Italian cohorts. Estimates based on the pooled Italian cohorts were preferred for meta-analyses because they represented more workers/larger sample sizes with updated methods/data including with follow-up to at least 2010 and an observation period of 40 or more years. Estimates were preferred from the underlying cohorts (22 papers) if they provided data for sub-group analyses not otherwise available in pooled cohort papers.

Evidence of an association between occupational asbestos exposure measurement and GI cancers was defined by the investigators based on a hierarchy of study informativeness with increased confidence in exposure assessment methods and significant asbestos exposure from 1) any/none asbestos exposure, 2) significant asbestos exposure characterized by major occupation and/or industry of employment, 3) two-fold or greater risk of asbestos-related lung cancer within the same cohort, and 4) exposure-response relationships with comparison of the highest versus lowest exposed group. Evidence of an association was also defined by positive meta-risk estimates above '1', stronger meta-risk estimates and the precision of the 95% confidence intervals (the range of effect estimates) with increased study informativeness, consistency of evidence across the meta-analyses, and the robustness of the findings to sensitivity analyses investigating the impact of any one analytic decision or any one study on the meta-risk estimates.

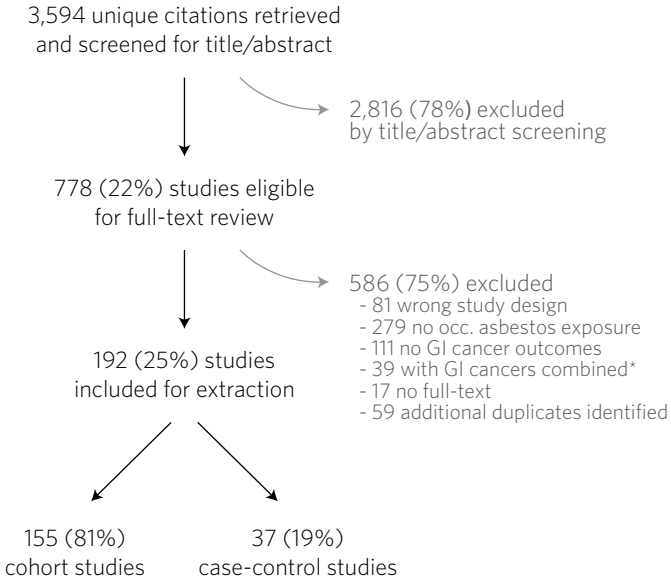
Consensus on the interpretation of the meta-risk estimates was sought during a second full meeting of the investigators and collaborators. The investigators adopted an approach consistent with that of prior authoritative evaluations (6,7) and as advocated for by leading occupational epidemiologists (45) that gives weight to the consistency of the findings and the increase in the estimated risks with study informativeness related to confidence in the exposure assessment; and that gives less weight to measures of statistical significance (arbitrary cut-points) for the meta-risk estimates in consensus decisions when evaluating serious hazards.

Results

Included Studies

Figure 1 provides the PRISMA flow chart for the inclusion of studies in the systematic review and meta-analyses on occupational asbestos exposure and GI cancer. A total of 3,594 unique citations were retrieved across the combined search strategies and databases. Based on title and abstract review a total of 778 studies were eligible for full text screening. After exclusions a total of 192 publications/studies were included in the systematic review.

Figure 1 | Flow chart for inclusion of studies in the systematic review and meta-analyses on the association of occupational asbestos exposure and GI cancers



**39 studies were excluded from the meta-analyses because they reported estimates for combined GI cancers or combined with other cancers. Of the 39 studnues, 21 (54%) have a subsequent paper published by the same authors or with the same cohort that was included in the systematic review with estimates by specific GI cancer site. See the reference list for the systematic review on page 78.*

Summary of Studies

Of the 192 studies included in the systematic review, 155 (81%) were cohort designs and 37 (19%) case-control designs. A total of 82 studies (43%) investigated esophageal cancer, 153 studies (80%) stomach cancer, and 144 studies (75%) colorectal cancer (70 studies (37%) investigated all three GI cancers and 47 (25%) investigated two of the three GI cancers). After the selection of preferred effect estimates as the most informative given multiple estimates within the same study or across the same cohort over time, a total of 56 studies contributed independent effect estimates to meta-analyses for esophageal cancer (68% of 82 eligible studies), 90 studies contributed independent effect estimates to meta-analyses for stomach cancer (59% of 153 eligible), and 82 studies contributed independent effect estimates to meta-analyses for colorectal cancer (57% of 144 eligible).

The publication years of the 192 studies ranged from 1964 to 2022 (1% in 1960s, 4% in 1970s, 21% in 1980s, 29% in 1990s, 23% in 2000s, 17% in 2010s, and 4% from 2020-2022). Studies were published on workers around the world, including in Italy (20%) and the United States (19%), followed by Sweden and Canada (8% respectively), Poland (7%), the United Kingdom and China (6% respectively), Australia and Germany (5% respectively), Japan (4%), France (3%), Austria and Denmark and Finland and Norway and Taiwan (2% respectively); and Belgium, Brazil, Israel, Lithuania, the Netherlands, New Zealand, Russia, Spain and Ukraine (6% combined).

Among the 56 studies that contributed independent effect estimates to meta-analyses for esophageal cancer, 50 (89%) were cohort studies (including four PMR studies) and six (11%) case-control studies. These studies were published from 1979 to 2020 (23% in 1980s, 11% in 1990s, 38% in 2000s, and 25% in 2010s), and spanned 18 different countries (e.g., 21% in the USA; 18% in Italy; 13% in the UK; and 7% in Canada, China and Sweden, respectively). Among the 90 studies that contributed independent effect estimates to meta-analyses for stomach cancer, 76 (84%) were cohort studies (including three PMR studies) and 14 (16%) case control studies (including one nested case-control study). These studies were published from 1976 to 2022 (18% in the 1980s, 29% in 1990s, 24% in 2000s, 18% in 2010s), and spanned 23 countries (e.g., 17% in the USA; 16% in Italy; 10% in China; 9% in the UK and Sweden, respectively; 8% in Japan; and 6% in Poland and Canada, respectively). Among the 82 studies that contributed independent effect estimates to meta-analyses for colorectal cancer, 66 (80%) were cohort studies (including four PMR studies) and 16 (20%) case control studies. These studies were published from 1979 to 2022 (20% in the 1980s, 24% in 1990s, 24% in 2000s, 23% in 2010s), and spanned 21 countries (e.g., 21% in the USA, 16% in Italy, 11% in Sweden, 7% in Canada, 7% in the UK and 6% in China).

Meta-risk Estimates

The overall meta-risk estimate (mREs) for occupational asbestos exposure (any/none) and the risk of GI cancers was 1.17 (95% CI 1.07-1.29) for esophageal cancer, 1.14 (95% CI 1.05-1.23) for stomach cancer, and 1.16 (95% CI 1.08-1.24) for colorectal cancer (Table 2).

Figures 2a to 2c provide forest plots displaying the studies with risk estimates and 95% CIs that were pooled for the overall meta-analyses of esophageal, stomach and colorectal cancer, respectively. For all three cancer sites, variability in the individual study risk estimates and confidence intervals was observed. However, more heterogeneity[2] was observed among the results for stomach (I^2=79.1%) and colorectal (I^2=69.9%) cancer than for esophageal cancer (I^2=27.7%).

[2] I^2 describes the extent of the overlap of confidence intervals around study estimates as a measure of heterogeneity. I^2 is a proportion not a value on an absolute scale. It does not indicate how much the true effects vary. For example, an I^2 of 75% tells us that 75% of the variance in observed effects reflects variance in true effects (Bornstein, 2022).

Table 2 | Meta-risk estimates (mREs) for the association between occupational asbestos exposure and gastrointestinal cancers by site, overall and sub-group analyses defined by asbestos exposure

Asbestos Exposure Categorization	Esophageal Cancer mREs [95% CIs]	Stomach Cancer mREs [95% CIs]	Colorectal Cancer mREs [95% CIs]
Overall			
Any versus None Exposed	1.17 [1.07 – 1.29]	1.14 [1.05 – 1.23]	1.16 [1.08 – 1.24]
Major Occupations/Industries			
Asbestos-related Insulation Workers	1.68 [1.19 – 2.36]	1.53 [0.93 – 2.51]	1.59 [1.14 – 2.23]
Asbestos-related Cement Workers	1.12 [0.84 – 1.47]	1.14 [0.99 – 1.32]	1.21 [1.06 – 1.38]
Asbestos-related Miners	1.13 [0.78 – 1.63]	1.30 [1.14 – 1.49]	1.15 [0.82 – 1.63]
All Other Occupations/Industries	1.16 [1.02 – 1.32]	1.01 [0.91 – 1.13]	1.07 [0.99 – 1.16]
Asbestos-related Lung Cancer Risk			
Risk Ratios < 1.00	0.53 [0.15 – 1.89]	0.81 [0.62 – 1.08]	1.05 [0.80 – 1.39]
Risk Ratios 1.00-1.99	1.15 [1.02 – 1.29]	1.07 [0.95 – 1.20]	1.02 [0.93 – 1.11]
Risk Ratios >= 2.00	1.40 [1.14 – 1.71]	1.33 [1.14 – 1.56]	1.48 [1.35 – 1.63]
Asbestos Exposure-Response			
Highest versus Lowest Exposed	1.63 [1.29 – 2.06]	1.28 [1.09 – 1.52]	1.29 [1.09 – 1.53]
Asbestos Fibre Type			
Chrysotile	1.17 [0.89 – 1.53]	1.09 [0.88 – 1.35]	1.05 [0.88 – 1.26]
Amphibole	1.16 [1.02 – 1.31]	1.35 [1.12 – 1.63]	1.38 [1.27 – 1.49]
Chrysotile and Amphibole Mix	1.44 [1.20 – 1.73]	1.21 [1.04 – 1.41]	1.23 [1.09 – 1.38]
Unclear/Unknown	1.05 [0.85 – 1.30]	0.94 [0.84 – 1.06]	0.99 [0.89 – 1.10]

Asbestos Exposure by Major Industry/Occupation Groups

Among workers with established asbestos exposures defined by their occupation and/or industry of employment, the highest elevated risks were observed for esophageal (mRE=1.68 (95% CI 1.19-2.36)), stomach (mRE=1.53 (95% CI 0.93-2.51)) and colorectal cancer (mRE=1.59 (95% CI 1.14-2.23)) among asbestos insulators/insulation workers (Table 2). Elevated meta-risk estimates were also observed for stomach cancer among asbestos miners (mREs=1.30 (95% CI 1.14-1.49)) and colorectal cancer among asbestos cement workers (mREs=1.21 (95% CI 1.06-1.38)). The remaining mREs by cancer site and industry/occupational exposure classification ranged from 1.12 to 1.15.

Figures 3a to 3c provide forest plots displaying the studies contributing risk estimates for the meta-analyses of esophageal, stomach and colorectal cancer, respectively, by major occupation/industry exposed groups. For all three GI cancer sites, variability in the individual study risk estimates and confidence intervals was observed. However, more variability was observed among the studies for stomach cancer (I^2 ranging from 0% to 75.2% for the three asbestos exposure groups) and colorectal cancer (I^2 from 20.8% to 66.7%), than for esophageal cancer (I^2 from 0% to 30.3%).

Asbestos Exposure by Asbestos-related Lung Cancer Risk

In the sub-group analyses of cohort studies that also investigated lung cancer, there was consistency of increased meta-risk estimates for esophageal (mRE=1.40 (95% CI 1.14-1.71)), stomach (mRE=1.33 (95% CI 1.14-1.56)) and colorectal cancer (mRE=1.47 (95% CI 1.34-1.61)) (Table 2) among workers in cohorts where there was also a two-fold or greater risk of asbestos-related lung cancer.

Figures 4a to 4c provide forest plots displaying the studies contributing risk estimates in the meta-analyses of esophageal, stomach and colorectal cancer, respectively, by risk of asbestos-related lung cancer. For all three cancer sites, variability in individual study risk estimates and confidence intervals was observed in the sub-group of studies where there was a two-fold or greater risk of asbestos-related lung cancer. However, more heterogeneity was observed among the results for stomach cancer (I^2=39.4%) than for esophageal (I^2=23.0%) or colorectal (I^2=0%) cancer.

These results are further detailed in scatter plots and meta regression (correlation) analyses (Figures 4d to 4f) of the relationship between GI and asbestos-related lung cancer estimates within the same cohort. The linear regression results indicate that as the relative risk of asbestos-related lung cancer increases (as a strong indicator of asbestos exposure) so does the risk of each GI cancer. The relationship was strongest for colorectal cancer (regression coefficient (log-scale) β=0.37 (95% CI: 0.25-0.49), followed by stomach (β=0.33 (95% CI: 0.21-0.46) and esophageal (β=0.24 (95% CI: -0.02-0.50)) cancers. In other words, for every 1% increase in asbestos-related lung cancer there was a 0.37%, 0.33% and 0.24% increase in the risk of colorectal, stomach and esophageal cancer, respectively.

Exposure-Response—Highest Asbestos Exposed Workers

In the sub-group analysis of studies that reported an exposure-response relationship (Table 2), there was consistency of increased meta-risk estimates for esophageal (mRE=1.63 (95% CI 1.29-2.06)), stomach (mRE=1.28 (95% CI 1.09-1.52)) and colorectal cancer (mRE=1.29 (95% CI 1.09-1.53)) among workers with the highest asbestos exposures compared to those with the lowest exposures.

Figures 5a to 5c provide forest plots displaying the studies contributing risk estimates in the meta-analyses for esophageal, stomach and colorectal cancer, respectively, for the highest exposed workers. For all three cancer sites, variability in the study risk estimates and confidence intervals was observed. However, more variability was observed among the results for stomach cancer (I^2=72.9%) than for colorectal (I^2=19.0%) or esophageal (I^2=8.8%) cancer.

Appendix III provides a summary of the studies included in the exposure-response meta-risk analyses for esophageal cancer, as illustrative of the detailed exposure assessment characteristics and methods used in these studies to quantify and compare high exposure levels. These studies included detailed exposure assessment methods defined by a combination of exposure characteristics (e.g., intensity, duration, frequency, cumulative exposure) and exposure assessment methods (e.g., employment records/work history, direct measurements, job exposure matrix, expert opinion).

Secondary Research Questions—Fibre Type, Effect Modification

The meta-risk estimates for the association between occupational asbestos exposure and GI cancers was elevated among sub-groups of workers exposed to amphibole, chrysotile and a combination of the two asbestos fibres. Variability around the estimates by fibre-type was greater than that observed for the overall and other sub-group meta-risk estimates due to a smaller number of studies within strata (Table 2). The highest meta-risk estimates were observed for exposure to mixed fibres for esophageal cancer (mRE=1.44 (95% CI 1.20-1.73)), and for exposure to amphibole-only fibres for stomach (mRE=1.35 (95% CI 1.12-1.63)) and colorectal (mRE=1.38 (95% CI 1.27-1.49)) cancer. Lower meta-risk estimates were observed for exposure to chrysotile-only fibres for colorectal (mRE=1.05 (95% CI 0.88-1.26)), stomach (mRE=1.09 (95% CI 0.88-1.35)), and esophageal (mRE=1.17 (95% CI 0.89-1.53)) cancers. For all three GI cancer sites, the lowest meta-risk estimates were observed for studies where the fibre type was not specified or unclear (ranging from 0.94 to 1.05).

Figures 6a to 6c provide forest plots displaying the studies contributing risk estimates in the meta-analyses of esophageal, stomach and colorectal cancer, respectively, by fibre type. For all three GI cancer sites, variability in the study risk estimates and confidence intervals was observed by fibre-type categories. However, more variability was observed among the estimates based on exposure to chrysotile-only (I^2 range 46.0% to 71.5% across cancer sites) and mixed fibres (I^2 range 12.4% to 62.5%) than for exposure to unknown or unspecified asbestos (I^2 range 23.6% to 44.3%) or amphibole-only fibres (0% to 34.1%).

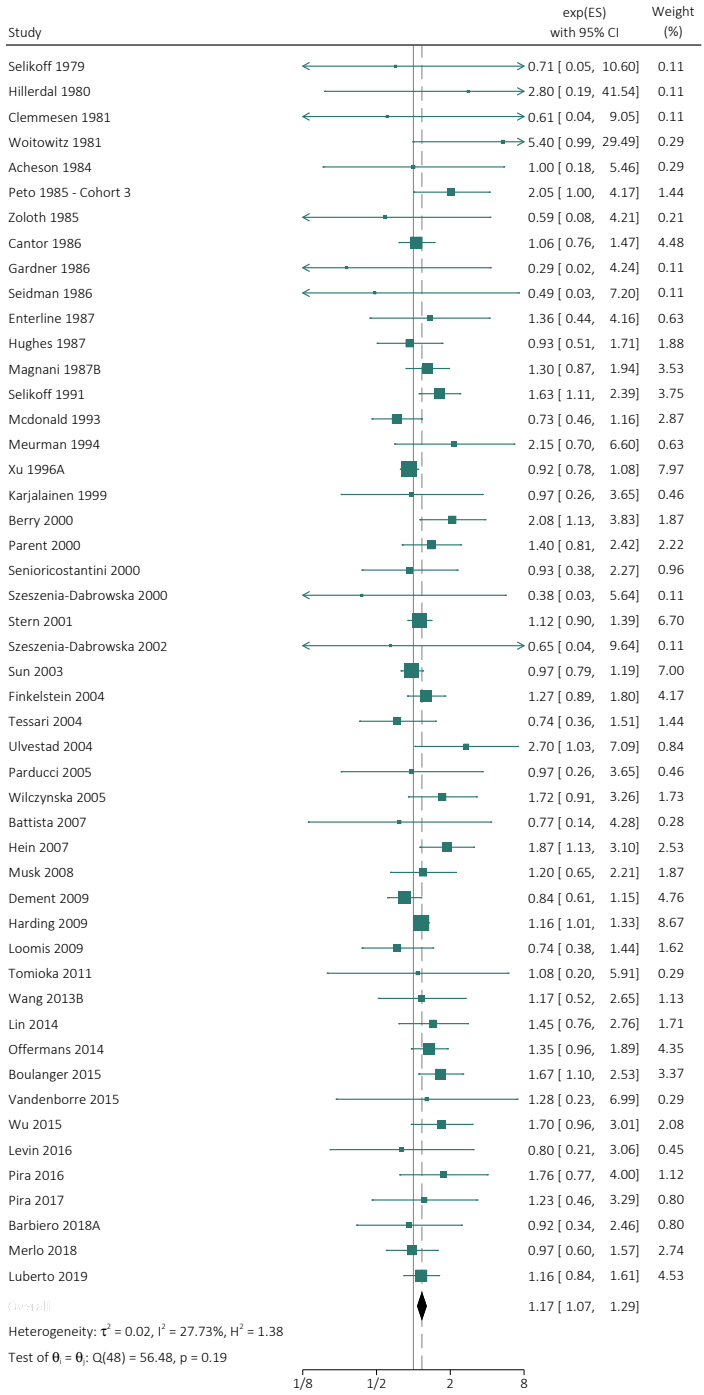
There were insufficient studies providing adequate data to investigate synergistic or antagonistic effects of asbestos with other known risk factors for GI cancers, specifically smoking and alcohol. Most studies included in the current review did not collect data on smoking (61%) or alcohol consumption (97%). Among those that collected smoking or alcohol data, this data was not always used in analyses (incomplete/missing), or results based on this data were not reported or reported in a manner that could be used to investigate synergistic or antagonist effects. See also the detailed discussion section below on confounding specific to smoking and alcohol consumption.

Supplemental Research Questions

The systematic review identified nine studies (eight cohort and one case-control) with independent measures of association on occupational asbestos exposure and small bowel (intestine) cancers. All of the study risk estimates were elevated but with wide confidence intervals (CIs). The risk estimates (with CIs) ranged from 1.25 (95% CI 0.73-2.15) to 7.69 (95% CI 0.52-114.11)—the latter estimate based on one observed case, and most based on small case counts. A meta-risk analysis of these eight studies failed to converge using the preferred restricted maximum likelihood (REML) approach to estimate the variance components. A meta-analysis converged using the maximum likelihood (ML) random effects model, although considered less than ideal for estimating the variance components. The ML model produced a pooled meta-risk estimate of 2.64 (95% CI 1.51-4.62) for occupational asbestos exposure (any/none) and small bowel cancer.

The systematic review identified five studies that measured time since first asbestos exposure to cancer diagnosis as a measure of latency (noting that this measure includes both the induction period and the latency period). Of these five studies, one investigated colorectal cancer (Gerhardsson de Verdier), two investigated stomach cancer (Harding, Raffn (1989)), and two investigated both stomach and colorectal cancer (Smailyte, Sanden). Methods varied across these studies with the investigation of a 0-19 year, 15-year, 20-year and 25-year period between time since first exposure and GI cancer diagnosis; and one study investigating 10-year periods after 20 years since first exposure. Two additional studies used a 10-year exposure lag as a method to account for latency, including one study investigating the three GI cancer sites (Lin) and one investigating stomach cancer (Straif). In sum, a meta-analysis investigating latency for GI cancers was precluded by insufficient evidence/few studies and methodological variability in the measurement and definition of latency for any one GI cancer site.

Figure 2a | Forest plot for meta-risk analyses of occupational asbestos exposure (any/none) and esophageal cancer



Random-effects REML model

Figure 2b | Forest plot for meta-risk analyses of occupational asbestos exposure (any/none) and stomach cancer

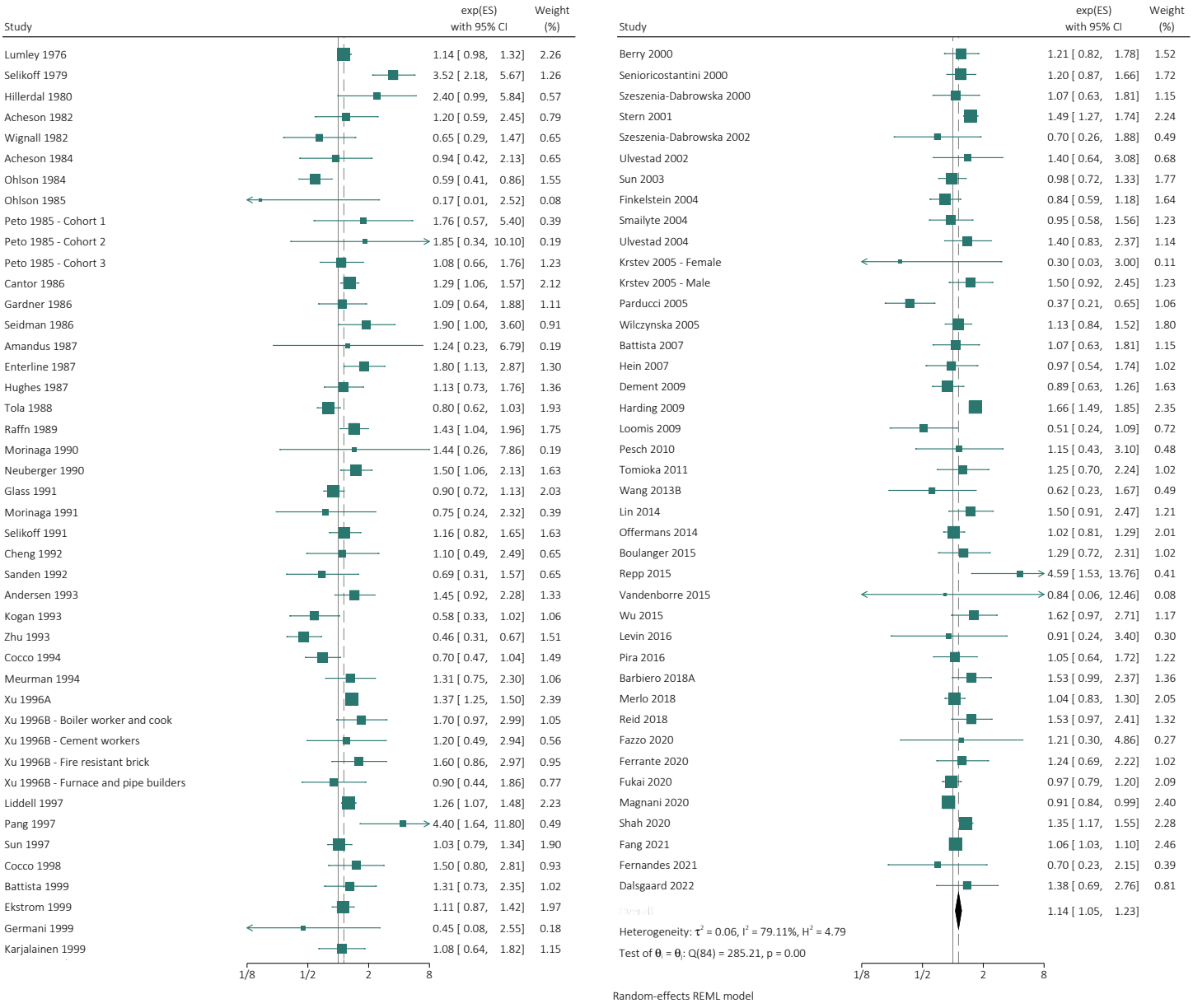


Figure 2c | Forest plot for meta-risk analyses of occupational asbestos exposure (any/none) and colorectal cancer

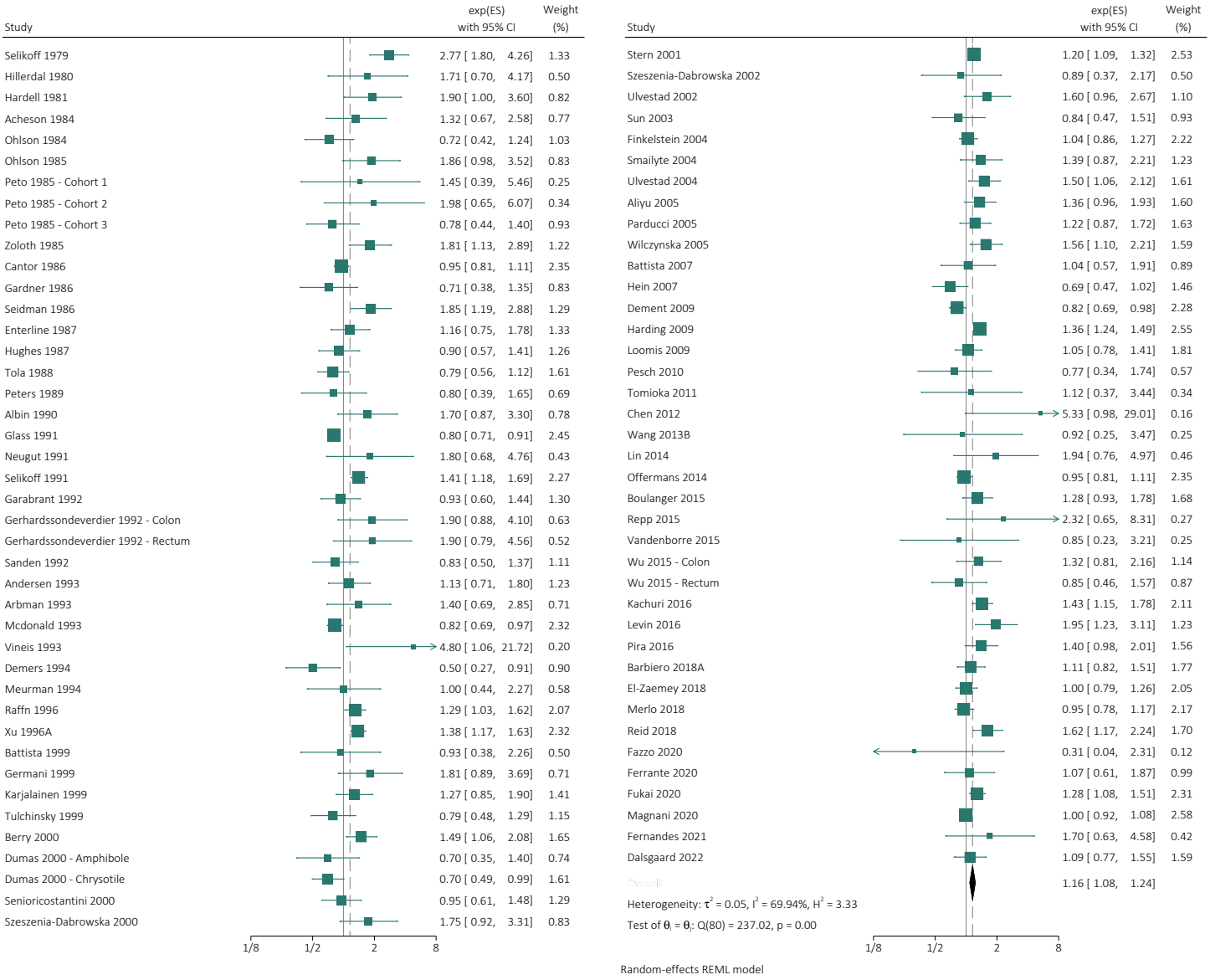


Figure 3a | Forest plot for meta-risk analyses of asbestos exposure and esophageal cancer by industry/occupation groups

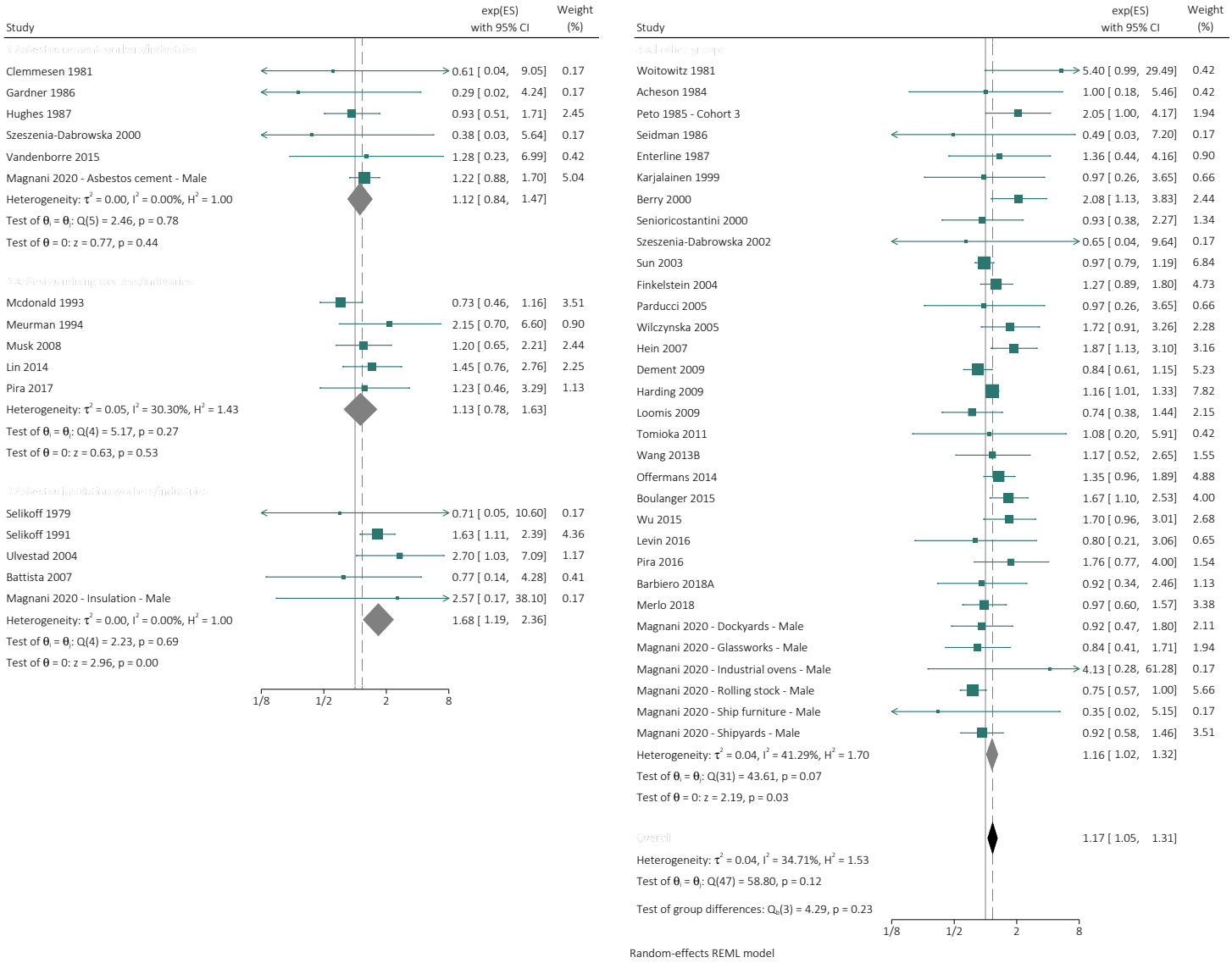


Figure 3b | Forest plot for meta-risk analyses of asbestos exposure and stomach cancer by industry/occupation groups

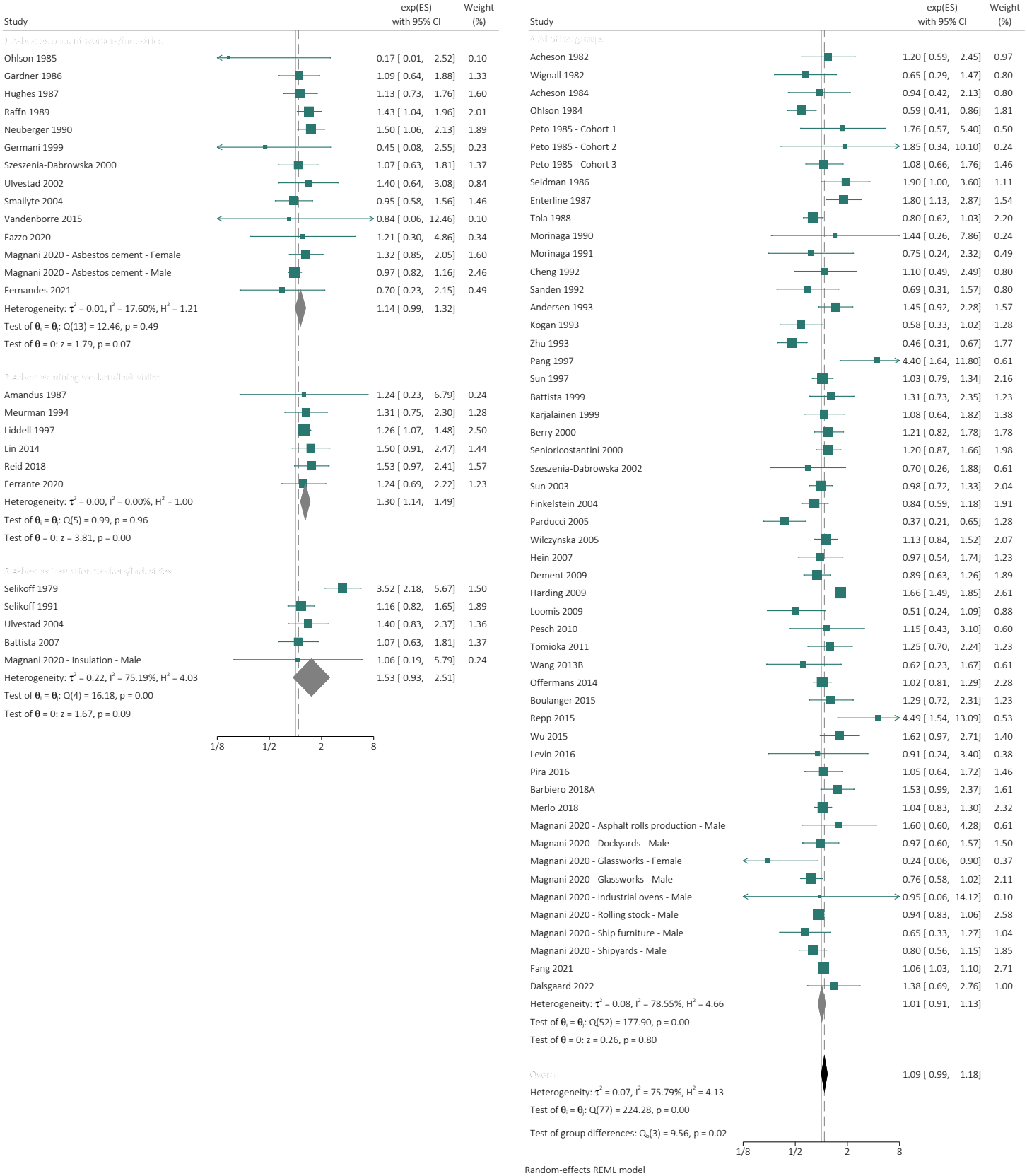


Figure 3c | Forest plot for meta-risk analyses of asbestos exposure and colorectal cancer by industry/occupation groups

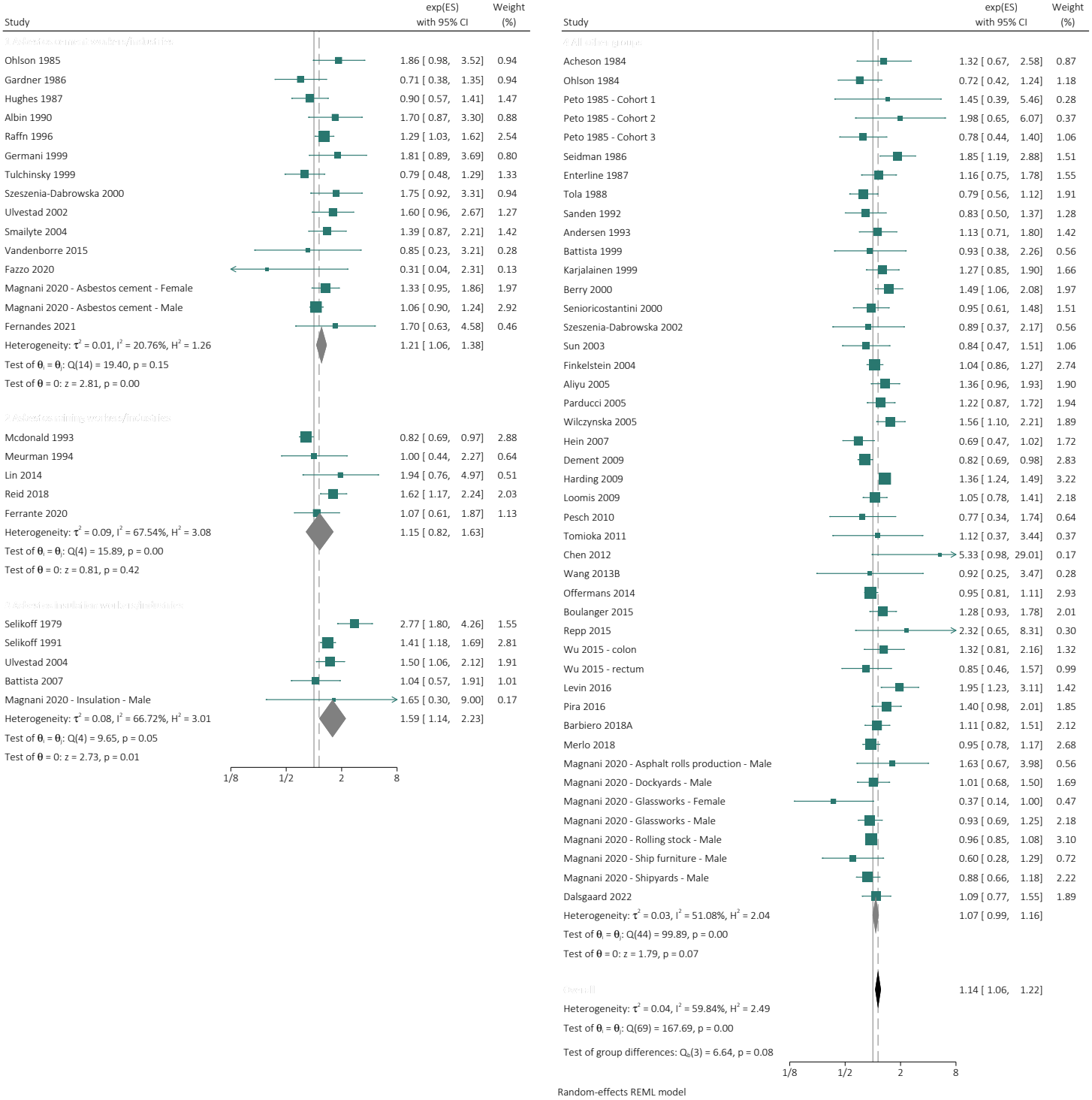
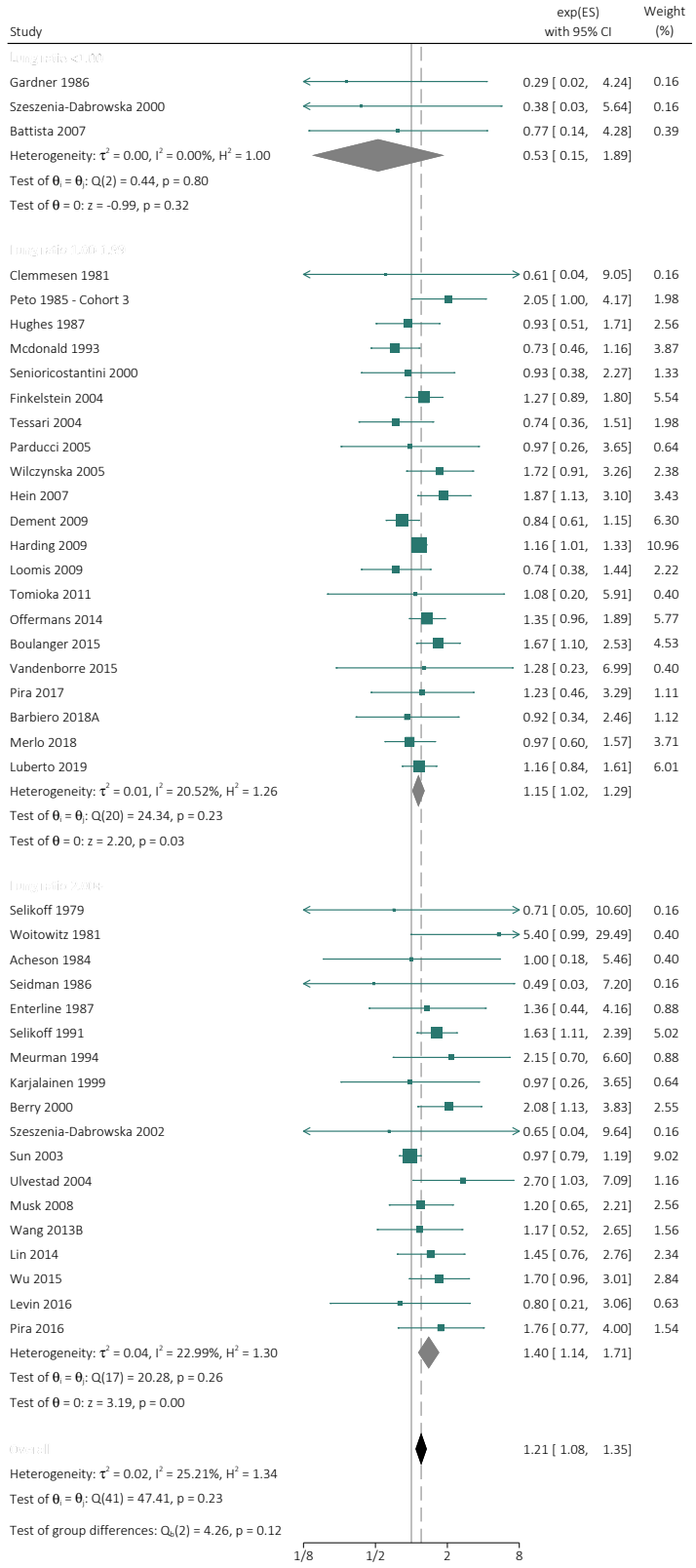


Figure 4a | Forest plot for meta-risk analyses of asbestos exposure and esophageal cancer by asbestos-related lung cancer risks (studies are ordered by year within categories of risk)



Random-effects ML model

Figure 4b | Forest plot for meta-risk analyses of asbestos exposure and stomach cancer by asbestos-related lung cancer risks (studies are ordered by year within categories of risk)

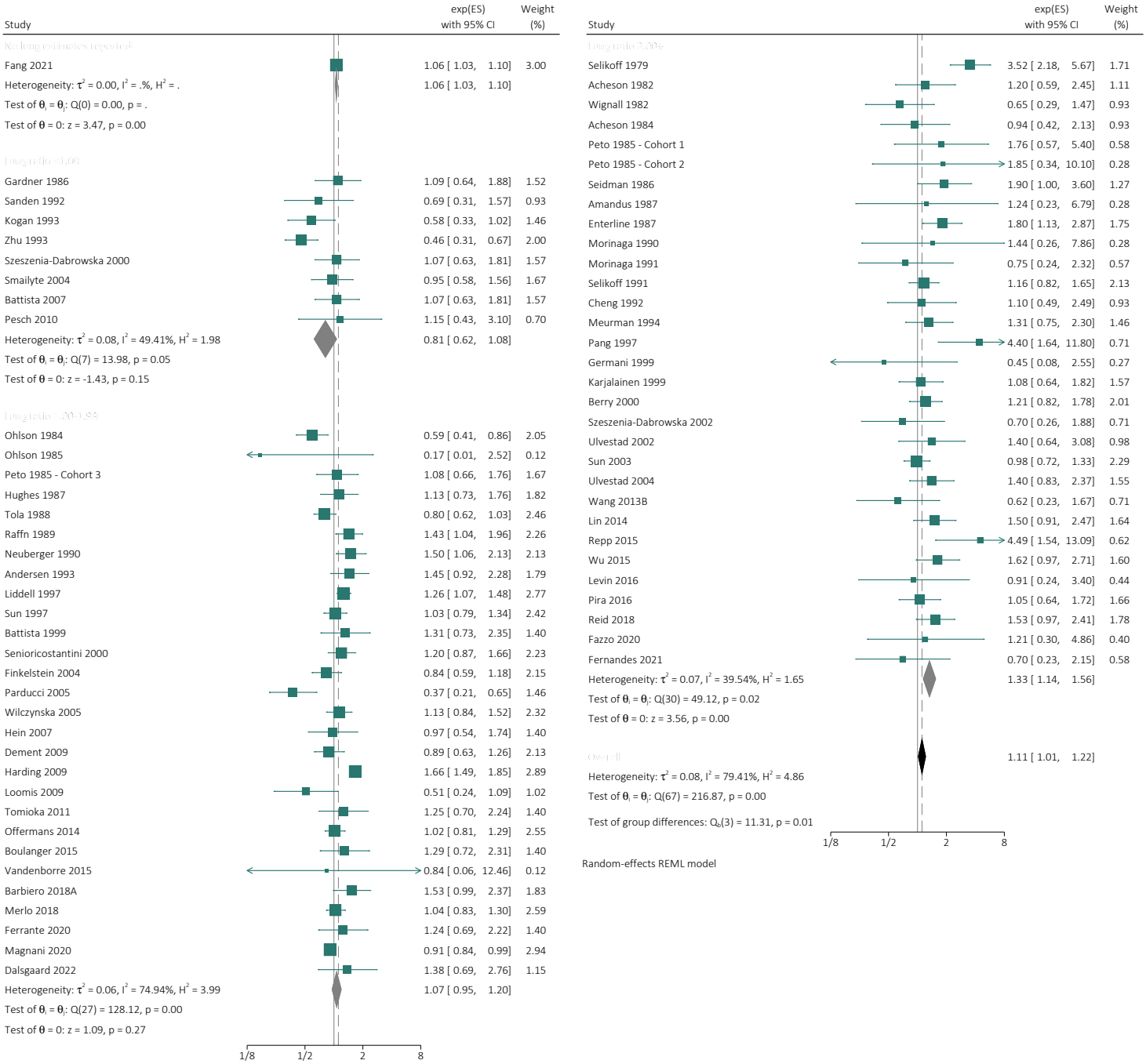


Figure 4c | Forest plot for meta-risk analyses of asbestos exposure and colorectal cancer by asbestos-related lung cancer risks (studies are ordered by year within categories of risk)

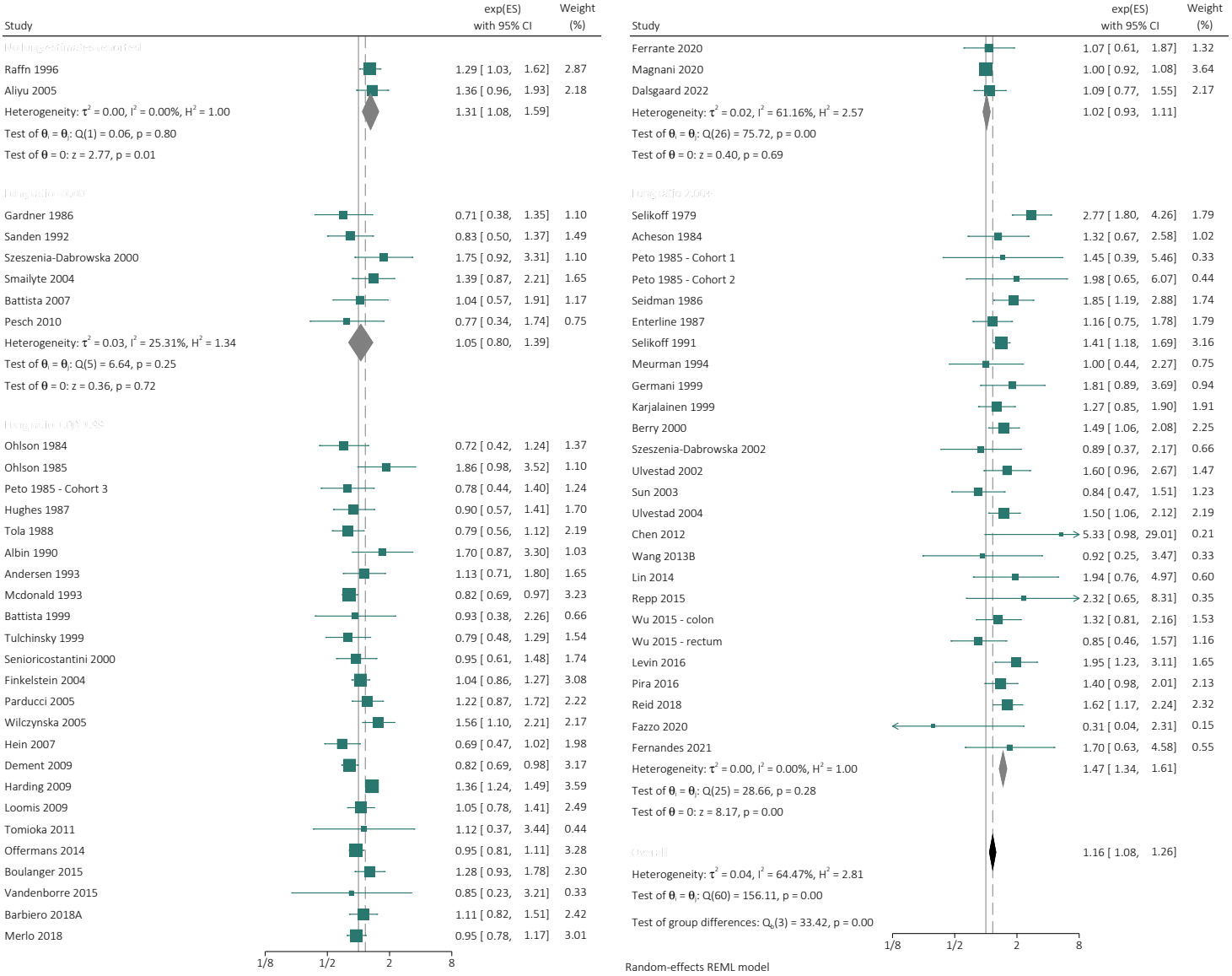
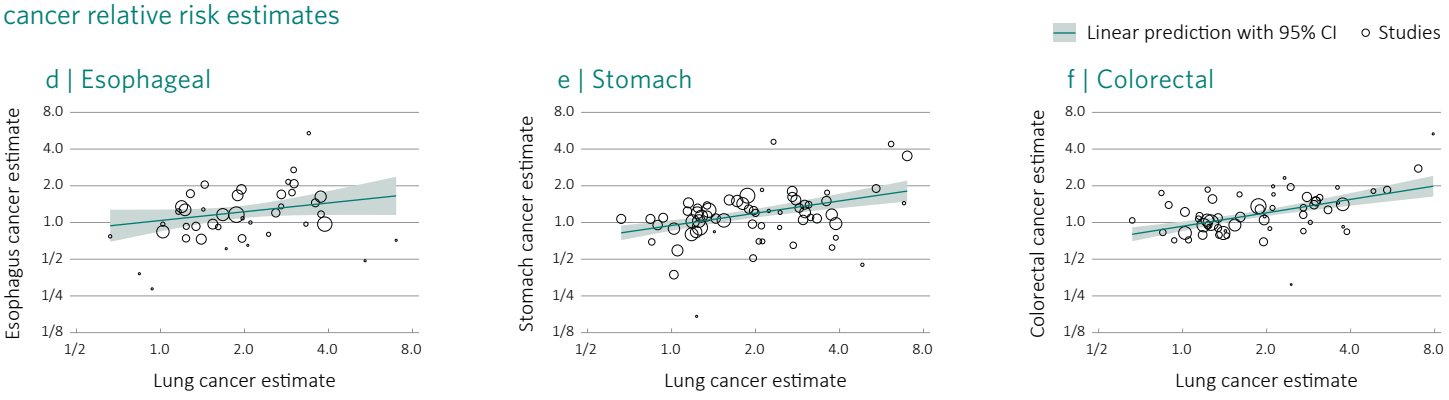
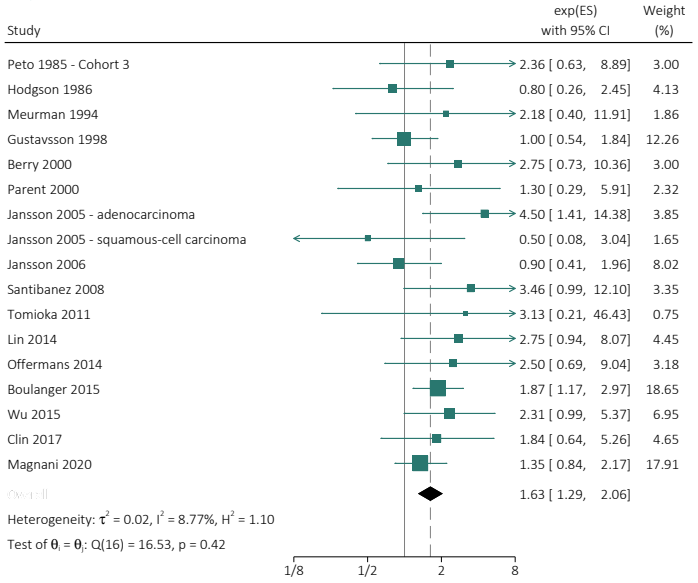


Figure 4d-f | Scatter plots of asbestos-related lung cancer relative risk estimates by esophageal, stomach and colorectal cancer relative risk estimates



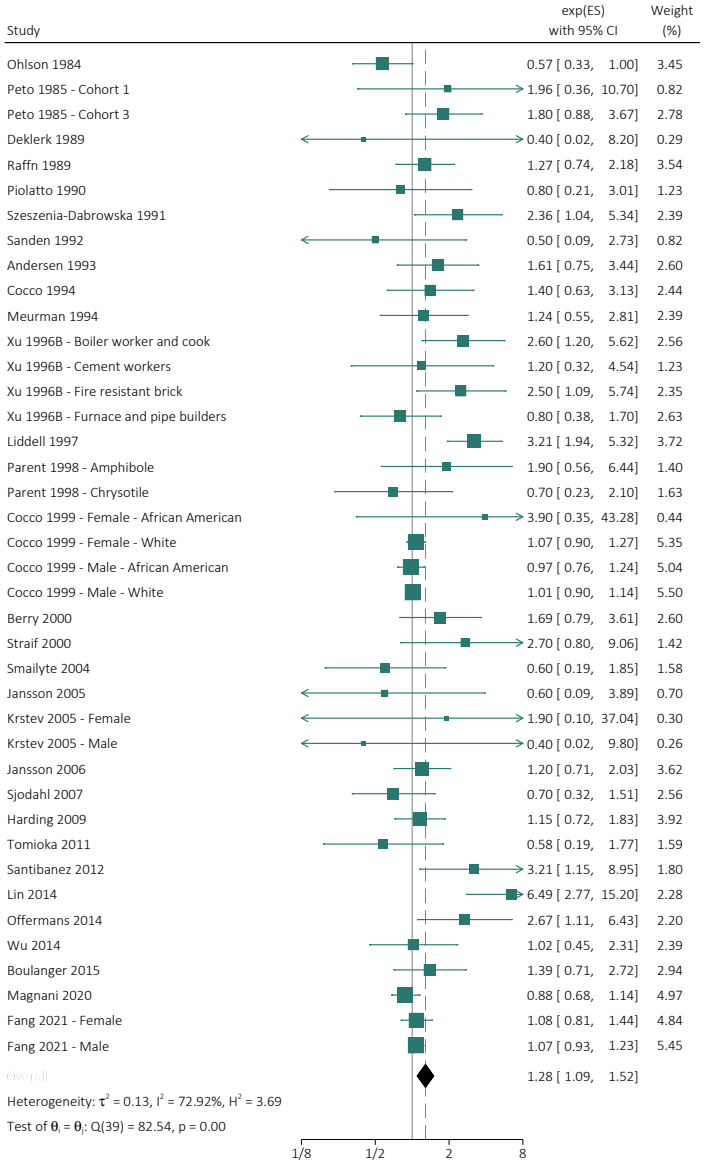
Weights: Random effects. Linear regression line and bubbles are weighted by inverse-variance.
Estimates were analyzed on the log-scale and then exponentiated to relative risks for the figure.

Figure 5a | Forest plot for meta-risk analyses of occupational asbestos exposure and esophageal cancer among highest exposed workers



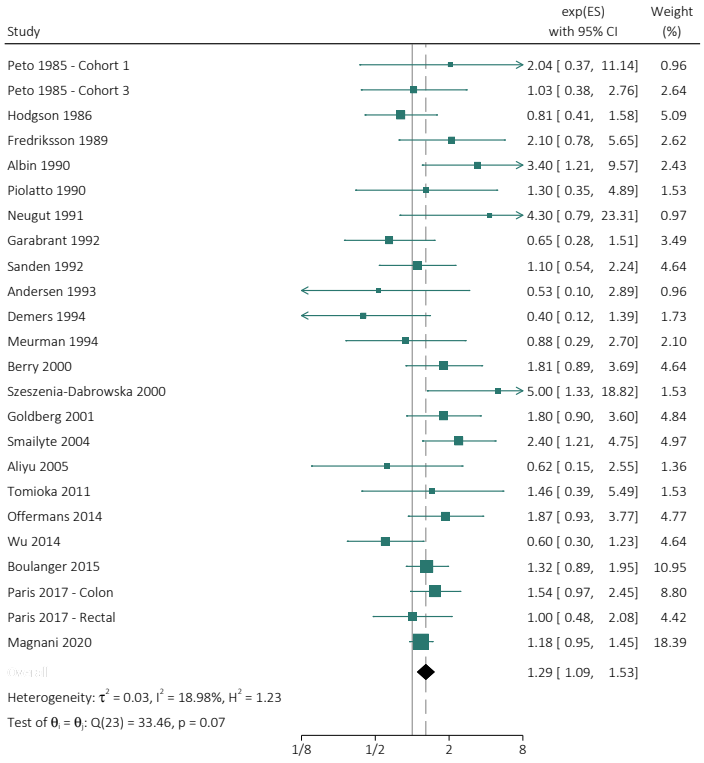
Random-effects REML model

Figure 5b | Forest plot for meta-risk analyses of occupational asbestos exposure and stomach cancer among highest exposed workers



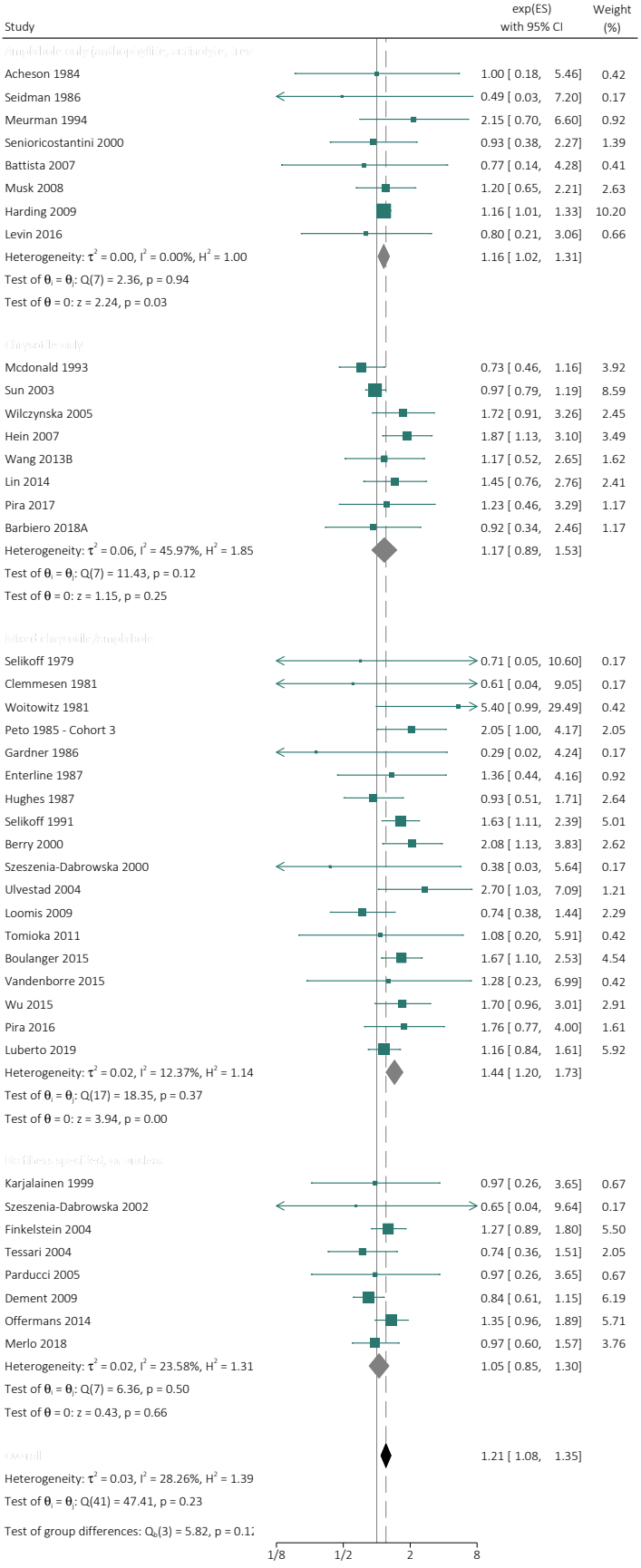
Random-effects REML model

Figure 5c | Forest plot for meta-risk analyses of occupational asbestos exposure and colorectal cancer among highest exposed workers



Random-effects REML model

Figure 6a | Forest plot for meta-risk analyses of occupational asbestos exposure and esophageal cancer by asbestos fibre type characterization



Random-effects REML model

Figure 6b | Forest plot for meta-risk analyses of occupational asbestos exposure and stomach cancer by asbestos fibre type characterization

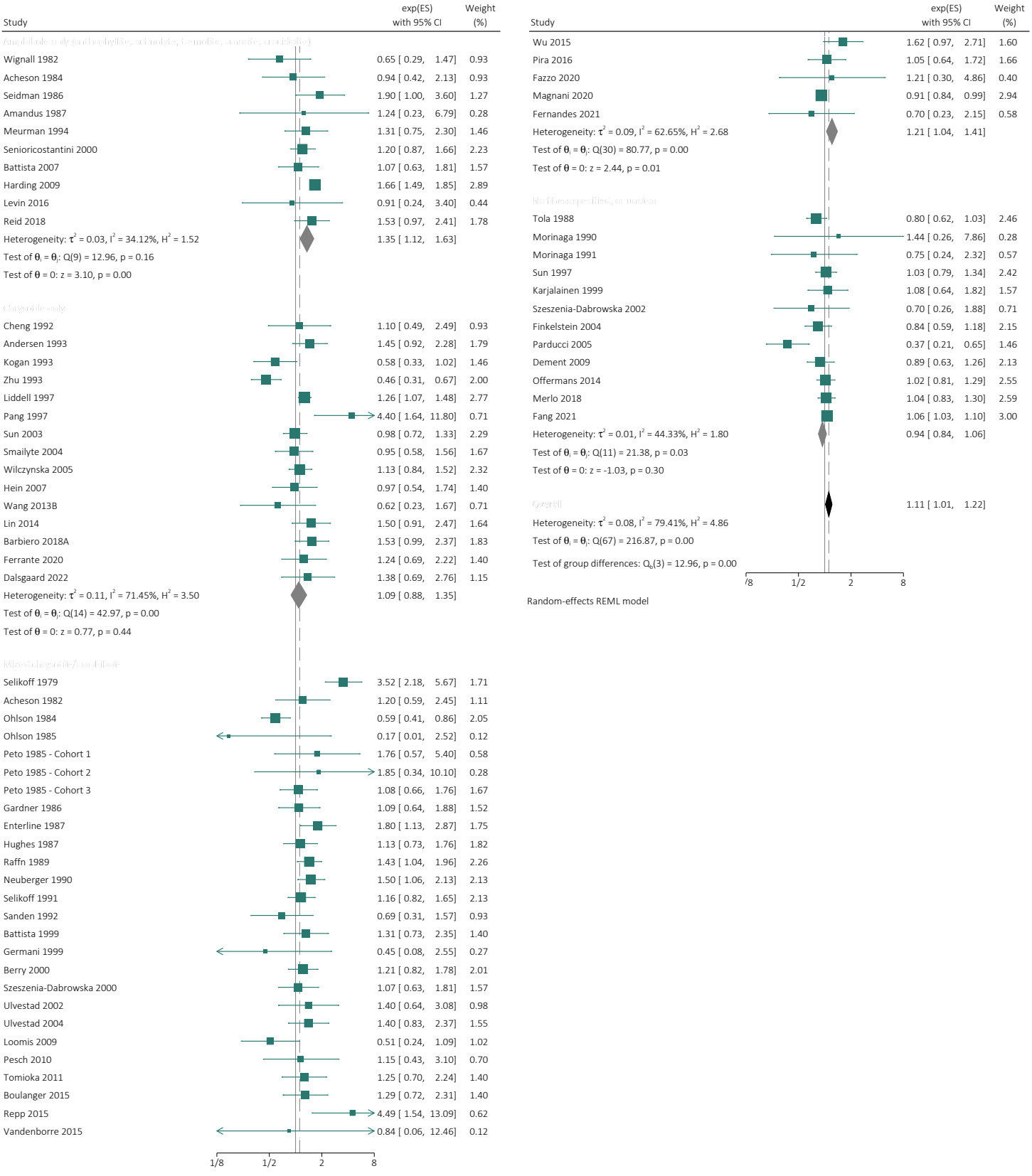


Figure 6c | Forest plot for meta-risk analyses of occupational asbestos exposure and colorectal cancer by asbestos fibre type characterization

Sensitivity Analyses

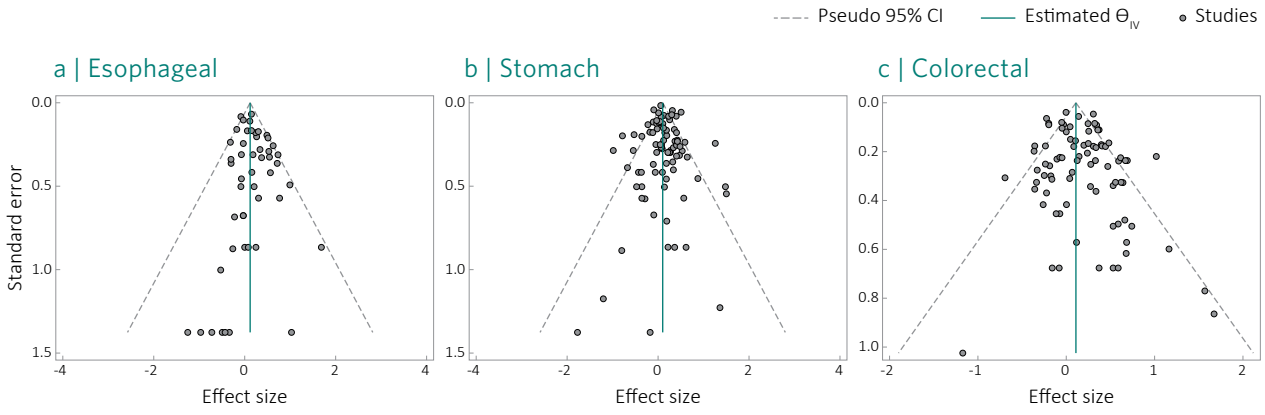
Funnel plots (Figures 7a to 7c) were inspected to assess the degree of publication bias. The plots were symmetric and the tests for asymmetry indicated minimal presence of small-study effects (46).

Sensitivity analyses were performed by removing one study at a time and re-running the meta-analyses (see Appendix IV for forest plots for leave-one-study-out sensitivity analyses for overall meta-risk estimates). Despite the observed heterogeneity, there was consistency of increased meta-risk estimates in the leave-one-out analyses, suggesting minimal influence from any one study on the overall and sub-group findings. For example, in the sub-group meta risk analysis for the highest versus lowest exposure comparisons, the estimates in the leave-one-out analyses for esophageal cancer ranged from 1.55 to 1.72 (compared to 1.63 for the main finding), for stomach cancer from 1.21 to 1.31 (compared to 1.28), and for colorectal cancer from 1.24 to 1.32 (compared to 1.29).

Sensitivity analyses were conducted to investigate the impact of the inclusion or exclusion of different effect estimates from studies. The meta-risk estimates were robust to investigator decisions on a preferred effect estimate as the most informative given multiple estimates within a study or multiple estimates for a study over time, including, for example, consistency of increased meta-risk estimates using data points from the underlying Italian cohort studies verses from the pooled Italian cohort studies.

Sensitivity analyses were conducted for cohort studies versus case-control studies. The positive association remained in the analysis of cohort only studies for which there is typically stronger assessment of asbestos exposure, and in the analysis of case-control only studies for which there is typically a more comprehensive adjustment for potential confounders. Although, there was increased variability around these sub-group estimates by study design given a small number of studies in each stratum (Table 3).

Figure 7a-c | Funnel plots for studies investigating association of occupational asbestos exposure and risk of GI cancer by cancer site



Regression-based Egger test for funnel plot asymmetry: p-value = 0.635

Regression-based Egger test for funnel plot asymmetry: p-value = 0.986

Regression-based Egger test for funnel plot asymmetry: p-value = 0.090

Sensitivity analyses were conducted to investigate the impact of new studies since the previous IARC authoritative evidence synthesis on the overall meta-risk estimates. The analysis included effect estimates from 16 new studies for esophageal cancer, 25 new studies for stomach cancer and 27 new studies for colorectal cancer. The cumulative meta-analysis incrementally re-calculates the estimates with the addition of each study over time. For all three GI cancer sites, the addition of new studies resulted in more consistent meta-risk estimates with less variability over time (see Appendix V for the forest plots). This consistency and reduced variability is due in part to more studies contributing to the meta-estimates over time, but also to the consistency of estimates with less variability from post 2009 studies with improved study designs and better exposure assessment methods.

Table 3 | Meta-risk estimates (mREs) for the association between occupational asbestos exposure and gastrointestinal cancers, by sensitivity analyses

	Esophageal Cancer mREs [95% CIs]	Stomach Cancer mREs [95% CIs]	Colorectal Cancer mREs [95% CIs]
Overall Meta-Risk Results	1.17 [1.07 – 1.29] n=49	1.14 [1.05 – 1.23] n=85	1.16 [1.08 – 1.24] n=81
Study Design			
Cohort	1.21 [1.08 – 1.35] n=42	1.11 [1.01 – 1.22] n=68	1.16 [1.08 – 1.26] n=61
Case-Control	1.35 [0.98 – 1.86] n=3	1.15 [0.97 – 1.35] n=13	1.10 [0.90 – 1.35] n=16
PMR Studies	1.01 [0.87 – 1.16] n=4	1.32 [1.18 – 1.47] n=4	1.23 [0.99 – 1.53] n=4

Discussion

This study, to our knowledge, represents the most comprehensive review yet of the epidemiological evidence of occupational asbestos exposure and GI cancers. The search strategy was designed to be inclusive of all peer-reviewed and published studies to date, including cohort and case-control study designs, mortality and morbidity cancer outcomes, English and non-English language studies, and new studies published since prior authoritative evaluations by IOM and IARC (2009-2022). Studies on the same cohort over time were included in the systematic review database to ensure the selection of preferred effect estimates across all available as the most informative and to maximize the effect estimates available for sub-group analyses on specific research questions (noting that no duplicate estimates were include in any one meta-analyses).

Does occupational asbestos exposure increase the risk of GI cancers?

In this systematic review and meta-analyses, there was consistency and increased meta-risk estimates for esophageal, stomach and colorectal cancer with occupational exposure to asbestos. Although there was a high degree of variability in the results, there was consistency of increased meta-risk estimates across the many sub-analyses and sensitivity analyses we performed.

The magnitude of the overall meta-risk estimates observed for asbestos exposure and GI cancers was modest compared to risks observed for asbestos exposure and lung cancer. Overall meta-risk analyses that include studies with greater potential for misclassification of occupational exposure tend to bias estimates towards the null on average, assuming that the error is random and non-differential with respect to the outcome (45). Our overall meta-risk estimates may be underestimates of the underlying association—indeed, the magnitude of the meta-risk estimates was stronger in the sub-analyses of studies where there was increased confidence in the classification of high and substantial occupational asbestos exposure, and where there was less observed heterogeneity.

Is there an exposure-response relationship by exposure characteristics and for sub-groups of workers?

Relatively few of the included studies conducted exposure-response analyses (29%, 44% and 30% of eligible esophageal, stomach and colorectal cancer studies, respectively) and even fewer provided directly comparable results with similar exposure category cut-points. However, the exposure-response studies were considered the most informative using detailed exposure metrics of intensity, duration and/or frequency; and using a combination of assessment methods including occupational history/employment records, direct exposure measurements, job-exposure matrices, and/or expert opinion (e.g., Appendix III for esophageal cancer). While the assessment of 'high' and 'low' asbestos exposure categories was defined relative to individual studies and their methods, it provides an acceptable measure of exposure-response

relationships across studies. Among workers in the highest exposure categories, there was consistency of increased meta-risk estimates for all three GI cancers, demonstrating a positive exposure-response relationship.

To assess the risk of GI cancers by industry and/or occupation, three groups with recognized asbestos exposure were investigated: insulators and insulating manufacturing workers, asbestos cement workers, and asbestos miners. The highest risk of all three GI cancers was observed among insulators and insulating manufacturing workers, an established high-risk group for asbestos-related disease (47, 48). Assessing the risk in these sub-groups yielded some variability, likely because exposure defined solely on industry/occupation does not reflect elements of intensity or duration of exposure, or other elements of study design. Smaller, but still elevated, risks were observed for both cement workers and miners for all three GI cancer sites.

Observed differences in the meta-risk estimates for asbestos insulation versus cement and mining occupations may be related to the nature of the work and the potential for the release of fibres. Insulation work is more prone to fraying, crumbling or abrading with an increased likelihood of fibres being disturbed and becoming airborne than within asbestos-cement where fibres are relatively bound within the cement matrix. Prior reports indicate higher fibre concentrations for insulation work compared to other types of asbestos work (Monograph Table 1.3 (6)) and higher risks of GI cancers among insulation work compared to other asbestos-related work such as mining (24). Although elevated, risk estimates associated with the mining and cement industries may be attenuated because they represent a mix of occupations with widely varying levels of exposure in comparison to insulation workers who have more consistent high exposures.

Does the risk of GI cancers co-vary with the risk of other asbestos-related cancers?

Most of the cohort studies included results for a wide range of cancer sites, the most common of which was lung cancer. Mesothelioma was also considered, but the dose-response is more variable and its rarity as an outcome would have excluded many studies from analyses with no cases. Lung cancer has an established dose-response relationship with asbestos and the risk of asbestos-related lung cancer within the same cohort of workers is a reasonable surrogate measure of asbestos exposure for investigating the risk with other cancers (e.g., the most recent IARC evaluation). In this systematic review, the relative risks for all three GI cancers were positively correlated with the risk of asbestos-related lung cancer in their respective cohorts. Further, when restricting to studies with a relative risk (generally an SMR or SIR) of two-fold or greater, there was consistency of stronger increased meta-risk estimates for all three GI cancers.

Is the risk of GI cancer associated with specific types of asbestos?

Assessing the risk of gastrointestinal cancer by fibre type proved challenging. Relatively few studies reported exposure to chrysotile or amphibole fibres alone. More studies reported exposure to a mixture of fibres and some studies did not indicate fibre type. For esophageal cancer, studies that reported a mixture of both chrysotile and amphibole fibres had the highest meta-risk estimates, with similar but smaller excess risks observed for the two specific fibre types individually. For both stomach and colorectal, the amphibole-only exposed group had the highest risk, the chrysotile-only exposed group the lowest risk (though still slightly elevated), and the mixed fibre exposure group the intermediate risk. The challenge to the interpretation of the findings by fibre type is that the level of exposure is not evenly distributed between the groups. For example, in the case of colorectal cancer, five of the eight studies (63%) for amphibole exposure, and 13 of the 30 studies (43%) for mixed fibre exposure, had asbestos-related lung cancer RRs greater than two; while only two of 10 studies (20%) for chrysotile exposure, and zero of seven studies with unspecified/unknown asbestos fibre exposure, had similarly elevated lung cancer risks. Interestingly, studies that did not report fibre type, perhaps an indicator of crude exposure assessment, had little or no association between asbestos and any of the three GI cancer sites.

Evidence for different asbestos fibres will remain a challenge as workers in different industries, eras and geographic locations are exposed to different types and sizes of asbestos fibres; with very few studies reporting direct measures of fibre type; and where fibre type on its own is not a measure of dose.

Does the exposure-response relationship vary by other GI cancer risk factors?

The International Agency for Research on Cancer's summary of monographs (48) identifies alcohol, tobacco/smoking and ionizing radiation as carcinogenic agents (sufficient evidence) for esophageal cancer; tobacco/smoking, ionizing radiation, rubber manufacturing and Helicobacter pylori infection as carcinogenic agents for stomach cancer; alcohol, tobacco/smoking, ionizing radiation, and processed meat consumption as carcinogenic agents for colon cancers; and alcohol, tobacco/smoking, and processed meat consumption as carcinogenic agents for rectum cancer.

Many of the aforementioned independent risk factors for specific GI cancers are generally not considered strong potential confounders of the relationship under investigation as neither a determinant of, nor reasonably associated with, occupational asbestos exposure. For example, the evidence that ionizing radiation is associated with GI cancers is primarily based on studies with non-occupational exposure to X- and Gamma-rays from medical devices. Some studies included in the current review collected data on smoking (39% of all studies) and alcohol consumption (3%), but most lacked information on these personal behaviours among workers or used indirect methods of adjustment. Smoking and alcohol consumption may be associated with asbestos exposure as these are known to be unequally distributed by occupation/industry groups and by socio-economic characteristics defined by employment (49), although this is less of a concern in studies investigating differences within an occupation or industry. Alcohol and

smoking consumption may also be related to asbestos exposure via unequal distribution of behaviours among age and sex/gender groups, but the majority of studies included in the current review adjusted for age and sex as independent confounders.

A prior investigation (50) of variation in cancer risks by occupation with adjustment of smoking and alcohol using the Nordic Occupational Cancer database (~15M workers across 54 occupational categories) reported minimal or moderate variation in risk estimates for esophageal, colon and rectal cancers among men and women (i.e. high or low risks persisted with adjustment) (NB: stomach cancer was not analyzed and none of the occupational categorizes were defined by asbestos exposures). In a prior systematic review focused on asbestos and colorectal cancer (24), meta-risk estimates for the sub-group analyses of studies with smoking data were similar to that of all studies except for increased variability around the estimates due to the smaller number of studies. Several studies using indirect adjustment or alternate methods to evaluate potential confounding due to smoking in occupational lung studies (51-59) have observed only modest effects due to differences in smoking among sub-groups of workers with different levels of occupational exposure (and where smoking is a strong confounder of lung cancer risk). Further, occupational epidemiologists who have studied the issue (e.g., Axelson, Steenland) have consistently demonstrated and argued that only substantially different distributions of confounders by exposure groups would fully explain an exposure-response relationship, even for strong confounder associations as in the case for smoking and lung cancer (45, 52-54).

Finally, it is important to note that any potential confounder (including smoking in lung cancer studies) can bias results towards or away from the null, depending upon the specific circumstances in a study (44, 49). As such, the observed meta-risk estimates in the current review could be an over- or underestimation of the relationship between occupational asbestos exposure and GI cancers, although reviewers tend to focus on unmeasured confounding as the explanation for positive studies or for exaggerating positive effect estimates (45).

In sum, there was insufficient studies providing adequate evidence to assess if exposure-response relationships varied among sub-groups of workers defined by other GI cancer risk factors, specifically smoking and alcohol. However, there is also minimal evidence that the observed meta-risk estimates for the association between occupational asbestos exposure and GI cancers are explained, fully or in part, by unmeasured confounding due to smoking or alcohol.

Interpretation of the Meta-Risk Estimates

Magnitude of Meta-Risk Estimates

The overall meta-risk estimates for GI cancers, and even the larger estimates observed for higher exposed sub-groups, were not in the range of that observed for asbestos-related lung cancer (generally two-fold). The ability to detect underlying relationships may be diluted by more diverse and less well understood

determinants of GI cancers, as well as difficulty with the detection and diagnosis (misclassification) of these cancers. Further, while causal inference is more challenging when the strength of association (statistical relationship) is smaller, it does not negate the presence of a causal relationship. Many of the recently established causal relationships are based on small or moderately increased risks (for many valid reasons related to cancers linked to multiple carcinogens). These smaller risk estimates are probably close to the "true" risk and not due to study limitations or incomplete evidence on other risk factors. In sum, the magnitude of the association may not be as important in decision-making as the consistency of risk estimates and the increased strength of the association with higher exposure levels (46).

Exposure Assessment

One of the persistent challenges for systematic reviews of occupational studies is differences in approaches to exposure assessment, follow-up periods, and exposure contrasts. This variability precluded sub-group-analyses by single exposure measurement characteristics or cut points. Investigations of risk by a single exposure characteristic or cut-point (e.g., 20 years of exposure) are misleading as a measure of dose as workers with high concentrations of exposure over a short period of time, or those with low concentrations of exposures over a long period of time, may have equal risks of GI cancer. Meta-analyses of asbestos-related lung cancer studies (60-67), as a parallel evidence base, indicate no threshold exposure level with models demonstrating risk with linear increases in exposure, and an exposure response with even low exposures. To address the issue of variability in exposure assessment across studies in the current review, the investigators adopted a hierarchy of informativeness for sub-group analyses for which there was confidence that the meta-risk estimates represented the highest or significantly exposed workers.

Exposure-response relationships are a crucial element in an evidence synthesis of epidemiologic investigations of disease outcomes such as cancer, and requires reconstruction of exposures over long periods of workers' lives for occupational investigations. All studies included in the high-low exposure sub-group analyses in the present synthesis included dose-response findings based on quantitative exposure assessment methods, that maximized the available exposure determinant information to assess exposure to asbestos (see Appendix III for example of exposure assessment for studies included in the esophageal cancer sub-group analyses). Based on a detailed review by an epidemiologist and a hygienist, the exposure assessment methods and measures varied greatly but adhered to best practices given the historical nature of the exposure. The methods involved a combination of approaches, including detailed work histories/employment/medical records, job exposure matrices, self-assessed or workplace survey of exposures, expert assessments, and/or direct measurements approaches; and the high exposure metric, albeit relative within a study/sample, was almost always defined by a combination of duration, intensity, frequency and/or cumulative exposure. In sum, the investigators are confident that the high exposed groups in the sub-analyses of studies with exposure-response effect estimates represent workers with the highest levels of asbestos exposure within their respective workplaces/occupations/study samples.

Latency

The current systematic review does not draw conclusions on latency for GI cancers, or on a minimum threshold for years since first asbestos exposure, with insufficient evidence to conduct sub-group meta-analyses. Investigators rarely have a strong evidence base upon which to select specific values for a latency period for asbestos-related cancer (68), and selecting a latency period assumes that this period is the same across workers rather than having a population distribution with variability (68). Stated another way, latency is not a measure of exposure dose and workers with higher doses may have shorter induction and/or latency periods, or vice versa. A prior systematic review of the asbestos-related lung cancer literature in 2014 (60), as a parallel evidence base to draw upon, did not identify a minimal latency period. Further, we conclude that the use of a period such as employment duration (as neither a measure of dose nor of latency) may be exclusionary and does not fully recognize the exposure complexity that contributes to risk for workers.

Heterogeneity

Heterogeneity was observed in the current meta-analyses (and in other prior meta-analyses) as defined by the I^2 statistic. This was not unexpected because of the pooling of occupational epidemiological studies that include differences in study samples, controls/comparison groups, duration of the follow-up or latency, case ascertainment and exposure assessments. Differences in exposure measures and methods has been noted by others as sources of heterogeneity in meta-analyses of occupational epidemiological studies (69). Some meta-analysis methodologists (Higgins, 70) argue that methodological diversity always occurs in meta-analyses and that statistical heterogeneity is 'inevitable' (and perhaps more so in occupational epidemiological studies than in randomized control trials, for example).

The impact of heterogeneity can be explored by conducting sub-group meta-analyses by study characteristics that have been defined a priori as potential sources of variability and where there are sufficient studies to do so. In the current analyses, this was investigated by a hierarchy of studies where there was increasing confidence in the exposure assessment. Heterogeneity can also be explored by performing random-effects meta-analyses that adjusts for an estimated measure of the extent of random variation across studies, as was the statistical approach in the current meta-analyses; although this does not account for 'true' unmeasured variation in the effects. Finally, some heterogeneity may be explained by the variability of the choice of the effect estimates, although this is less of a concern in sub-group analyses based on cohort studies and the same risk measures.

Regardless, the investigators contend that the observed heterogeneity in the current meta-analyses is expected due to the methodological diversity of the pooled occupational studies (e.g., different populations, effect estimates, exposure metrics and follow-up periods). Despite the methodological diversity, there was consistency of increased meta-risk estimates for GI cancers with occupational asbestos exposure, including

stronger meta-risk estimates for the analyses of studies where there was confidence the workers were the highest or significantly exposed. The meta-risk estimates for the sub-analyses of studies with high or significant asbestos exposure had some of the lowest observed I^2 values collectively (0%, 8.8%, 19.0%, 23.0%, 39.4%), with the exception of the meta-risk for stomach cancer and the highest exposed (72.9%).

Consistency with Prior Systematic Reviews

The conclusion of a causal elevated risk of stomach and colorectal cancers with occupational asbestos exposure in the current systematic review is consistent with the reviews by IOM (2006) (7), IARC (2009/2012) (6) and FIOH (2014) (8); and the more recent systematic reviews by Peng (2015, stomach) (22), Fortunato (2015, stomach) (23) and Kwak (2019, colorectal) (24). Prior evaluations (see Appendix VI for detailed summary) report overall meta-risk estimates for ever exposed to asbestos from 1.11 to 1.19 for stomach cancer, and from 1.15 to 1.16 for colorectal cancer, consistent with our estimates of 1.14 and 1.16, respectively. Further, prior meta-risk estimates for the highest exposed workers ranged from 1.13 to 1.33 for stomach cancer and 1.24 for colorectal cancer, also consistent with our estimates of 1.28 and 1.29, respectively.

The conclusion of a causal elevated risk of esophageal cancer with occupational asbestos exposure is different to the IOM and IARC reviews in 2006 and 2009/12, respectively; but consistent with the more recent systematic review by Wu in 2021 (25). An elevated risk for esophageal cancer in the current and the recent review by Wu may be as a result of the inclusion of more well-designed studies (i.e., 22 studies published since 2006), given this is the GI cancer that had the fewest included studies/estimates in prior meta-analyses and where the evidence was assessed as inadequate or inconclusive. For esophageal cancer, prior evaluations report overall meta-risk estimates for ever exposed to asbestos from 0.99 to 1.28 (see Appendix VI, including footnote for further insight on the IOM estimate), and for highest exposed from 1.35 to 1.84, with our estimates of 1.17 and 1.63 falling within these ranges for ever and highest exposed, respectively.

In the current analyses, the strongest meta-risk estimates were observed in exposure-response studies among workers in the highest exposed groups, ranging from 1.28 to 1.63; among workers with a history of exposure as asbestos insulators, ranging from 1.53 to 1.68; and among workers where there was a two-fold or greater risk of asbestos-related lung cancer in the same cohort, ranging from 1.33 to 1.47. This parallels prior evaluations that also report increased meta-risk estimates among the highest exposed workers, among workers with a history of exposure as insulators, and among cohorts with a two-fold or greater risk of asbestos-related lung cancer (see Appendix VI for detailed comparison).

Conclusions

We found evidence of an increased risk of esophageal cancer, stomach cancer and colorectal cancer with occupational exposure to asbestos. There was consistency of stronger estimates of the association in meta-analyses of studies where there was better exposure assessment and increased confidence in the categorization of asbestos exposure, including among the highest exposed workers in exposure-response studies; among workers with a history of significant exposure as a result of the nature of their work (e.g., asbestos-related insulation); and among workers in cohorts where there was also a two-fold or greater increased risk of asbestos-related lung cancer as a strong indicator of exposure. There was heterogeneity in the studies included in the review, although results from sensitivity analyses indicate that there was minimal influence from any one study on the overall meta-estimates or from publication bias. Unexplained heterogeneity was reduced, and the strength of association increased, in the sub-group analyses of studies where there was better asbestos exposure assessment. The consistency of an increased risk of GI cancers with occupational asbestos exposure was robust to multiple sensitivity analyses that investigated the impact of meta-analyses methods. Further research is needed to investigate GI cancer risk by asbestos fibre type, and effect modification of the relationship between GI cancers and occupational asbestos exposure by other occupational and non-occupational factors.

The evidence synthesis, as summarized above, supports a causal link between occupational asbestos exposure and esophageal, stomach and colorectal cancer.

Appendices

Appendix I | Search Strategy (March 4, 2022)

Full Description of Key Words and Subject Heading Terms

The following table lists the keywords and subject heading terms for the database search strategy, as executed in the following database: Ovid MEDLINE(R) and Epub Ahead of Print, In-Process, In-Data-Review & Other Non-Indexed Citations, Daily and Versions(R) 1946 to December 1, 2021. Equivalent searches were run in Web of Science, Embase, CINAHL and Scopus. The search terms are organized into concepts defined by the PICO framework (population, intervention, comparator, outcome). Bolded terms represent the main concepts to be combined for the final search results.

PICO concept	#	Searches	Results	Final results	Annotations
Asbestos exposure	1	exp Asbestos, Amosite/ or exp Asbestos, Serpentine/ or exp Asbestos/ or exp Asbestos, Crocidolite/ or exp Asbestos, Amphibole/	9,869	967	
	2	asbestos.ti,ab.	13,223	1,463	
	3	asbestiform*.ti,ab.	173	14	
	4	amphibole*.ti,ab.	813	72	
	5	amosite*.ti,ab.	629	49	
	6	crocidolite*.ti,ab.	1,225	101	
	7	chrysotile*.ti,ab.	2,068	181	
	8	tremolite*.ti,ab.	378	44	
	9	actinolite*.ti,ab.	98	6	
	10	anthophyllite*.ti,ab.	159	12	
	11	1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10	15,133	1,562	Final terms for asbestos
Cancer outcomes (all sub-types)	12	exp Neoplasms/	3,581,453	1,637	
	13	tumo?r*.ti,ab.	1,836,515	356	
	14	oncolog*.ti,ab.	168,318	36	
	15	malignan*.ti,ab.	621,767	632	
	16	(metastat* or metastas* or metastaz* or metastagen*).ti,ab.	543,842	40	
	17	neoplas*.ti,ab.	277,414	164	
	18	carcinoma*.ti,ab.	704,491	153	
	19	cancer*.ti,ab.	1,970,437	1,364	
	20	12 or 13 or 14 or 15 or 16 or 17 or 18 or 19	4,687,490	1,883	Final terms for cancer, to be combined with GI body part keywords
Gastrointestinal body parts	21	exp gastrointestinal tract/	670,928	28	
	22	(esophagu* or oesophagu*).ti,ab.	65,961	78	
	23	(esophageal* or oesophageal*).ti,ab.	126,084	76	
	24	stomach.ti,ab.	112,949	185	
	25	(pylorus or pyloric).ti,ab.	15,693	2	
	26	fundus.ti,ab.	32,164	1	
	27	gastric.ti,ab.	265,677	77	
	28	intestine*.ti,ab.	134,645	27	
	29	intestinal.ti,ab.	294,981	30	
	30	duodenum.ti,ab.	34,796	0	
	31	duodenal.ti,ab.	61,310	4	

PICO concept	#	Searches	Results	Final results	Annotations
	32	(duodenojej* or duodeno-jej*).ti,ab.	1,579	0	
	33	jejunum.ti,ab.	22,734	1	
	34	jejunal.ti,ab.	20,430	1	
	35	ileum.ti,ab.	34,758	1	
	36	ileal.ti,ab.	28,011	0	
	37	diverticul*.ti,ab.	29,484	0	
	38	bowel*.ti,ab.	163,297	24	
	39	colon.ti,ab.	176,453	162	
	40	(colorectal* or colo-rectal*).ti,ab.	166,022	105	
	41	hepatic-flex*.ti,ab.	667	0	
	42	sigmoid-flex*.ti,ab.	102	0	
	43	(cecum or caecum).ti,ab.	15,876	1	
	44	splenic-flexure.ti,ab.	1,585	0	
	45	rectal.ti,ab.	93,455	45	
	46	rectum.ti,ab.	39,423	62	
	47	(rectosigm* or recto-sigm*).ti,ab.	5,301	1	
	48	anorect*.ti,ab.	15,878	1	
	49	digestive.ti,ab.	62,834	74	
	50	(gastrointestin* or gastro-intestin*).ti,ab.	264,793	93	
	51	21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 or 45 or 46 or 47 or 48 or 49 or 50	1,775,453	646	Final terms for GI body part, to be combined with cancer terms
GI cancer outcomes (defined by cancer + body part keywords)	52	20 and 51	642,511	646	GI cancer defined by by keywords
GI cancer outcomes (defined by MeSH terms)	53	exp Gastrointestinal Neoplasms/	407,340	362	GI cancer defined by MeSH terms
GI cancer outcomes (keywords and MeSH)	54	52 or 53	692,491	712	Final terms for GI cancers
Cohort study filters	55	cohort studies/	300,123	440	
	56	follow-up studies/	676,581	248	
	57	longitudinal studies/	153,015	24	
	58	prospective studies/	605,874	141	
	59	cohort*.ti,ab.	710,565	805	
	60	(follow* adj3 stud*).ti,ab.	123,962	63	
	61	follow up.ti,ab.	1,072,892	419	
	62	followed up.ti,ab.	110,469	94	
	63	longitudinal*.ti,ab.	298,160	28	
	64	retrospective*.ti,ab.	871,463	256	
	65	prospective*.ti,ab.	778,557	169	
	66	55 or 56 or 57 or 58 or 59 or 60 or 61 or 62 or 63 or 64 or 65	3,402,716	1,431	Final terms for cohort study filter
Case-control study filters	67	Case-Control Studies/	312,649	326	
	68	(case* adj10 control*).ti,ab.	248,490	401	
	69	(case* adj3 comparison*).ti,ab.	7,677	9	
	70	(case* adj3 comparator*).ti,ab.	111	0	
	71	(case* adj10 referent*).ti,ab.	1,075	48	

PICO concept	#	Searches	Results	Final results	Annotations
	72	(case* adj3 matched*).ti,ab.	18,097	62	
	73	(case* adj3 nested*).ti,ab.	11,604	61	Hybrid study design
	74	(case* adj cohort*).ti,ab.	2,353	9	Hybrid study design
	75	casecontrol*.ti,ab.	176	1	
	76	casereferent.ti,ab.	1	0	
	77	67 or 68 or 69 or 70 or 71 or 72 or 73 or 74 or 75 or 76	462,751	526	Final terms for case-control filter
Occupational exposures	78	exp occupational diseases/	138,013	931	
	79	exp workers' compensation/	7,684	12	
	80	exp occupational exposure/	67,063	696	
	81	exp employment/	93,782	36	
	82	occupations/	23,810	139	
	83	worker?.ti,ab.	201,469	808	
	84	(worksite? or workplace? or jobsite?).ti,ab.	49,203	67	
	85	work* compensation.ti,ab.	4,760	3	
	86	((work or worker* or working or job or employ* or occupation*) adj (exposure* or related* or environment? or site? or place? or population? or cohort? or sample?)).ti,ab.	69,977	439	
	87	(exposure* adj matri*).ti,ab.	1,318	67	
	88	exp manufacturing industry/	93,908	271	
	89	exp construction industry/	1,804	13	
	90	(ship adj5 break*).ti,ab.	49	0	
	91	(ship adj5 build*).ti,ab.	92	2	
	92	(ship adj5 yard*).ti,ab.	47	0	
	93	(ship adj5 dismant*).ti,ab.	8	0	
	94	(ship adj5 recycl*).ti,ab.	19	0	
	95	(ship adj5 scrap*).ti,ab.	17	0	
	96	shipbreak*.ti,ab.	24	3	
	97	shipbuild*.ti,ab.	290	17	
	98	shipyard*.ti,ab.	875	46	
	99	factory.ti,ab.	11,771	101	
	100	factories.ti,ab.	6,926	29	
	101	(textile* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	3,668	62	
	102	(cement* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	1,200	87	
	103	(construction* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	5,788	55	
	104	(pipefitt* or (pipe adj5 (fitt* or worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*))).ti,ab.	184	12	
	105	(steamfitt* or (steam adj5 (fitt* or worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*))).ti,ab.	149	3	
	106	(insulat* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	410	35	
	107	(miners or ((mining* or mine*) adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*))).ti,ab.	10,391	127	

PICO concept	#	Searches	Results	Final results	Annotations
	108	(warehous* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	180	1	
	109	(manufactur* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	8,452	58	
	110	(railway* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	499	11	
	111	(transport* adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	5,509	5	
	112	((trade or trades) adj5 (worker* or worksite* or workplace* or job* or staff* or personnel* or occupation* or employ* or industr* or sector*)).ti,ab.	1,807	17	
	113	78 or 79 or 80 or 81 or 82 or 83 or 84 or 85 or 86 or 87 or 88 or 89 or 90 or 91 or 92 or 93 or 94 or 95 or 96 or 97 or 98 or 99 or 100 or 101 or 102 or 103 or 104 or 105 or 106 or 107 or 108 or 109 or 110 or 111 or 112	569,113	1,451	Final terms for occupational exposures
Combinations of PICO concepts (search results)	114	11 and 20 and 66	1,683	1,336	Search #1: Cohort (asbestos exposure + general cancer outcomes + cohort studies, without regard for GI cancer sub-type)
	115	54 and 77 and 113	420	364	Search #2: Case-control (GI cancer outcomes + case control studies + occupation exposures, without regard for asbestos exposure)
	116	11 and 54	602	391	Search #3: Asbestos + GI cancer without regard for specific study design
	117	114 or 115 or 116	2,461	1,883	Combined search results
Additional limiters	118	remove duplicates from 117	2,458		Combined search results, excluding duplicates across the strategies
	119	exp animals/ not humans/	4,923,990		Animal studies to exclude
	120	118 not 119	2,416		Combined search results, excluding animal-only studies
	121	limit 120 to "review articles"	304		
	122	limit 120 to "case reports"	159		
	123	limit 120 to "comment"	31		
	124	limit 120 to "editorial"	4		
	125	limit 120 to "letter"	45		
	126	limit 120 to "meta analysis"	63		
	127	limit 120 to "review"	304		
	128	limit 120 to "systematic review"	42		
	129	limit 120 to "systematic reviews"	46		
	130	121 or 122 or 123 or 124 or 125 or 126 or 127 or 128 or 129	533		Studies to exclude based on limiters
	131	120 not 130	1,883		Final combined search results, excluding duplicates, animal studies, and non-relevant publications such as systematic reviews and editorials)

Appendix II | Summary Table of Included Studies in Meta-Risk Analyses by Cancer Site

Esophageal Cancer Studies

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Selikoff	1979	United States	Cohort 1: 632M, Cohort 2: 17800M	1967	1943-1976	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Case-control	Hillerdal	1980	Sweden	482M, 420W exposed/ cases; 1158M, 960W controls	1968-1972		General Work Population	No fibers specified, or unclear	Ratio O/E
Cohort	Clemmesen	1981	Denmark	5686	1943-1976	1944-1976	Asbestos Cement	Mixed chrysotile/amphibole	SIR
PMR	Woitowitz	1981	Germany	2944	1972-1979	1972, 1980	Various Industries Asbestos Dust (Central Register: Industrial Injuries Insurance Institutes)	Mixed chrysotile/amphibole	SPMR
Cohort	Acheson	1984	UK	4820	1947-1979	1980	Insulation Board Manufacturing	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Peto	1985	UK	cohort 1: 145M, cohort 2: 283W, cohort 3: 3211M exposed/cases	1933	1983	Asbestos Textile Factory Workers	Mixed chrysotile/amphibole	SMR
PMR	Zoloth	1985	United States	407	1976-1983		Sheet Metal Workers	No fibers specified, or unclear	PMR
PMR	Cantor	1986	United States	7121	1960-1979		Plumbers And Pipefitters	No fibers specified, or unclear	PMR
Cohort	Gardner	1986	UK	2167	1941-1983	1941-1984	Asbestos Cement Factory Workers	Mixed chrysotile/amphibole	SMR
Cohort	Hodgson	1986	UK	31150	Pre post 1969, 1972	1981	General Work Population, Workplaces Subject To 1969 Asbestos Regulations	No fibers specified, or unclear	
Cohort	Seidman	1986	United States	820	1941-1954	1982	Asbestos Factory Workers	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Enterline	1987	United States	1074	1941-1980	1941-1969; 1941-1973; 1941-1980	Production, Maintenance Employees, The Asbestos Company	Mixed chrysotile/amphibole	SMR
Cohort	Hughes	1987	United States	6931M (5492 men in analytic cohort)	1970	1982	Asbestos Cement Manufacturing	Mixed chrysotile/amphibole	SMR
Case-control	Magnani B	1987	UK	244 cases; 935 controls	1959-1963 1965-1979		Various Occupations, Industries	No fibers specified, or unclear	RR
Cohort	Selikoff	1991	United States	17800	1967	1977-1986	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Cohort	Mcdonald	1993	Canada	5351M	Born 1891-1920, surviving upto 1976	1976-1988	Asbestos Miners, Millers	Chrysotile only	SMR
Cohort	Meurman	1994	Finland	736M, 167F	1953-1967	1953-1991	Anthophyllite Mines	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SIR
Case-control	Xu A	1996	China	8887	1980-1989		Iron And Steel Workers	No fibers specified, or unclear	PMR
Case-control	Gustavsson	1998	Sweden	545 exposed/cases; 641 controls	1988-1990		General Work Population	No fibers specified, or unclear	

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Karjalainen	1999	Finland	1287M men, 89W with asbestosis, 4708M,179W with benign pleural disease	1964-1995	1964-1995	General Work Population (Finnish Registry of Occupational Diseases)	No fibers specified, or unclear	SIR
Cohort	Berry	2000	UK	3000M, 700W, 1400 insulators	1933-1964 men, 1936-1942 women	1951-1980	Asbestos Manufacturing	Mixed chrysotile/amphibole	SMR
Case-control	Parent	2000	Canada	99 exposed/cases; population control=533; other types of cancer=533 controls	1979-1985		Motor Vehicle Mechanics; Welders and Flame Cutters; Stationary Engineers (Table 3)	Chrysotile only	OR
Cohort	Seniori-costantini	2000	Italy	3741	1960-1995	1960-1996	Railway Rolling Factory	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Szeszenia-Dabrowska	2000	Poland	2525M, 591W	1959-1965	1959-1991	Asbestos Cement	Mixed chrysotile/amphibole	SMR
PMR	Stern	2001	United States	12873M	1972-1996		Construction Plasterers, Cement Masons	No fibers specified, or unclear	PMR
Cohort	Szeszenia-Dabrowska	2002	Poland	907M, 490W	1970-1997	1970-1999	Asbestos Processing Plant, Foundry, Shipyard	No fibers specified, or unclear	SMR
Cohort	Sun	2003	China	5681	1960-1980	1960-2000	Chrysotile Asbestos Spinning	Chrysotile only	SMR
Cohort	Finkelstein	2004	Canada	25285	1949	1950-1999	Plumbers And Pipefitters	No fibers specified, or unclear	SMR
Cohort	Tessari	2004	Italy	1621 cohort1, 1190 cohort2	since 1946	1965-2001 and 1970-2001	Railway Rolling Stock Manufacture, Repair	No fibers specified, or unclear	SMR
Cohort	Ulvestad	2004	Norway	1116M	1930-1975	1953-1999	Insulation Workers	Mixed chrysotile/amphibole	SIR
Cohort	Jansson - adenocar-cinoma and squamous cell carcinoma	2005	Sweden	260052M	1971-1993	1971-2000	Construction Workers	Chrysotile only	
Cohort	Parducci	2005	Italy	1585W, 756M	1960-1994	2002	Tobacco Production	No fibers specified, or unclear	SMR
Cohort	Wilczynska	2005	Poland	4187	1945-1980	1945-1999	Asbestos Plant Manufacturing Asbestos Yarn, Cloth, Cords, Packings, Stuffing, Brake Linings, And Asbestos-Natural Rubber Sheets	Chrysotile only	SMR
Case-control	Jansson	2006	Sweden	189 exposed/cases; 262 controls	1995-1997		Various Occupations (Table 1, 2)	No fibers specified, or unclear	
Cohort	Battista	2007	Italy	2301	1978-1988	1962-2003	Asbestos Insulation	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Hein	2007	United States	3072	1916-1977	1940-2001	Asbestos Textile Plant	Chrysotile only	SMR
Cohort	Musk	2008	Australia	6943	1943-1966	1943-2000	Crocidolite Miners And Millers	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Case-control	Santibanez	2008	Spain	185 exposed/cases; 285 controls	1995-1999		Carpenters, Joiners, Miners, Quarry Workers, Manufacturing Labourers And Many Others	No fibers specified, or unclear	

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Dement	2009	United States; Canada	17345	Not reported	1986-2004	Sheet Metal Workers	No fibers specified, or unclear	SMR
Cohort	Harding	2009	UK	98117	1971-2005	1971-2006	General Work Population, Focusing On Workplaces Subject To The 1969 Asbestos Regulations In The Uk	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Loomis	2009	United States	5770	1950-1973	1950-2003	Asbestos Textile Plant	Mixed chrysotile/amphibole	SMR
Cohort	Tomioka	2011	Japan	249	1947-1979	1947-2007	Laggers And Boiler Repairers In A Refitting Shipyard	Mixed chrysotile/amphibole	SMR
Cohort	Wang B	2013	China	586M, 279F	1972	1972-2008	Asbestos Textile	Chrysotile only	SMR
Cohort	Lin	2014	China	1539	Not reported	1981-2006	Asbestos Mining	Chrysotile only	SMR
Cohort	Offermans	2014	Netherlands	58,279	Not reported	1986-1970	General Work Population (From The Netherlands Cohort Study)	No fibers specified, or unclear	HR incidence
Cohort	Boulanger	2015	France	2024	1978	1978-2009	Asbestos Plant Workers	Mixed chrysotile/amphibole	SIR
Cohort	Vandenborre	2015	Belgium	1,397,699 (2056 asbestos workers, 385046 potentially exposed workers, 1010597 reference population)	Not reported	2001-2009	Asbestos Cement, Products Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Wu	2015	Taiwan	4427	1975-1989	1985-2008	Shipbreaking	Mixed chrysotile/amphibole	HR incidence
Cohort	Levin	2016	United States	979	1954-1972	1993-2013	Pipe Insulation Plant (The Tyler Facility)	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Pira	2016	Italy	1083W, 894M	1946-1984	1946-2013	Asbestos Textile	Mixed chrysotile/amphibole	SMR
Cohort	Clin	2017	France	13859 exposed/cases; 656 controls; 14515 overall	2003-2005	Up to 2015	Asbestos-Exposed Workers Covered By French National Health Insurance Fund (Mechanics, Plumbers And Pipefitters, Bricklayers, Electricians, Sheet-Metal Workers, Welders, Mill Workers, Freight Handlers, Insulation Workers; Various Industries: Iron/Steel Manufacturing, Construction Sector, Cargo Handling, Metalworking, Ship Repair)	No fibers specified, or unclear	
Cohort	Pira	2017	Italy	1056 exposed/cases; N/A controls	1930-1990	1946-2014	Asbestos Mining	Chrysotile only	SMR
Cohort	Barbiero A	2018	Italy	2488	1991-2008	1995-2009	Various: Metalworking, Shipbuilding, Electrical Utilities, Insulation	Chrysotile only	SIR
Cohort	Merlo	2018	Italy	3984	1960-1981	1960-2014	Shipyard Workers	No fibers specified, or unclear	SMR
Cohort	Luberto	2019	Italy	12578	Not reported	40+ years of observation	Asbestos Cement Workers	Mixed chrysotile/amphibole	SMR
Cohort	Magnani	2020	Italy	51801 (46060M, 5741W)	Years of first exposure ranged from pre-1949 to 1992	1970 onwards (end year not reported)	Various: Asbestos-Cement, Rolling Stock Construction and Maintenance, Shipyards, Ship Furnishing, Glassworks, Dockyards, Insulation, Asphalt Rolls, Oven Construction, And Asbestos Miners	Mixed chrysotile/amphibole	

Stomach Cancer Studies

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
PMR	Lumley	1976	UK	1377 exposed/cases; 4998 controls	1960-1969		Dockyard	Mixed chrysotile/amphibole	PMR
Cohort	Selikoff	1979	United States	Cohort 1: 632M, Cohort 2: 17800M	1967	1943-1976	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Case-control	Hillerdal	1980	Sweden	482M, 420W exposed/ cases; 1158M, 960W controls	1968-1972		General Work Population	No fibers specified, or unclear	Ratio O/E
Cohort	Acheson	1982	UK	1327	1939	1951-1980	Gas Mask Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Wignall	1982	UK	500	1939-1944	1951-1977	Gas Mask Assembly	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Acheson	1984	UK	4820	1947-1979	1980	Insulation Board Manufacturing	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Ohlson	1984	Sweden	3297	Not reported	1951-1980	Railroad Maintenance Worker	Mixed chrysotile/amphibole	SMR
Cohort	Ohlson	1985	Sweden	1176	1943 to 1976	1951-1982 mortality; 1958-1979 morbidity	Asbestos Cement Workers	Mixed chrysotile/amphibole	SMR
Cohort	Peto - Cohort	1985	UK	cohort 1: 145M, cohort 2: 283W, cohort 3: 3211M exposed/cases	1933	1983	Asbestos Textile Factory Workers	Mixed chrysotile/amphibole	SMR
PMR	Cantor	1986	United States	7121	1960-1979		Plumbers And Pipefitters	No fibers specified, or unclear	PMR
Cohort	Gardner	1986	UK	2167	1941-1983	1941-1984	Asbestos Cement Factory Workers	Mixed chrysotile/amphibole	SMR
Cohort	Seidman	1986	United States	820	1941-1954	1982	Asbestos Factory Workers	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Amandus	1987	United States	575	M hired prior to 1970	1981	Asbestos Miners And Millers	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Enterline	1987	United States	1074	1941-1980	1941-1969;1941-1973;1941-1980	Production, Maintenance Employees: The Asbestos Company	Mixed chrysotile/amphibole	SMR
Cohort	Hughes	1987	United States	6931M (5492M analytic cohort)	1970	1982	Asbestos Cement Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Tola	1988	Finland	12693	1945-1960	1953-1981	Shipyard, Machine Shop (Welders, Platers, Machinists, Pipe Fitters)	No fibers specified, or unclear	SIR
Case-cohort Nested	Deklerk	1989	Australia	92 (lung), 31 (meso), 17 (stomach cancer) exposed/ cases; Matched>20 controls	1943-1946 employed, followed up to 1980		Asbestos Mine	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	
Cohort	Raffn	1989	Denmark	7996M, 584W exposed/ cases; 1% LTFU controls	1928-1984	1943-1984	Asbestos Cement Workers	Mixed chrysotile/amphibole	SIR

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Morinaga	1990	Japan	208 workers (73M and 135F)	1964-1981	1964-1983	Asbestos Workers	No fibers specified, or unclear	SMR
Cohort	Neuberger	1990	Austria	2816	1950-1981	1987	Asbestos Cement	Mixed chrysotile/amphibole	SMR
Cohort	Piolatto	1990	Italy	1048	1946-1987	1987	Asbestos Miners	Chrysotile only	
Case-control	Glass	1991	New Zealand	19904	1980-1984		Various (Machinery Fitters, Welders, Plumbers, Boilermakers, Electricians, Bricklayers)	No fibers specified, or unclear	OR
Cohort	Morinaga	1991	Japan	789 (329M, 460F)	1972-1974	1975-1974	Asbestos Handling Workers	No fibers specified, or unclear	SMR
Cohort	Selikoff	1991	United States	17800	1967	1977-1986	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Cohort	Szeszenia-Dabrowska	1991	Poland	2403	1945-1973	1945-1985	Asbestos Processing Plant	Chrysotile only	
Cohort	Cheng	1992	China	1,172 individuals in the cohort (662M and 510W)	Since 1972	1972-1987	Asbestos Production	Chrysotile only	SMR
Cohort	Sanden	1992	Sweden	3893	1977-1979	1977-1979 to 1987	Shipyard Workers	Mixed chrysotile/amphibole	SMR
Cohort	Andersen	1993	Norway	690	1920-1966	1960-1991	Lighthouse Keepers- Drinking Rain Water from Asbestos Cement Tiled Roofs	Chrysotile only	SIR
Cohort	Kogan	1993	Russia	156 (cohort 1); 2834 (cohort 2)		1966-1985 (C1), 1949-1988 (C2)	Asbestos Friction Product Workers	Chrysotile only	SMR
Cohort	Zhu	1993	China	5893M and W	1972-81	1982-1986	Asbestos Factory Workers	Chrysotile only	SMR
Case-control	Cocco	1994	Italy	640 exposed/cases; 959 controls	1985-1987		General Work Population	No fibers specified, or unclear	OR
Cohort	Meurman	1994	Finland	736M and 167F	1953-1967	1953-1991	Anthophyllite Mines	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SIR
Case-control	Xu A	1996	China	8887	1980-1989		Iron And Steel Workers	No fibers specified, or unclear	PMR
Case-control	Xu B	1996	China	610 (lung cancer); 293 (stomach cancer) exposed/ cases; 959 controls	1989-1993 (lung cancer); 1989-1993 (stomach cancer)		Iron and Steel Workers (Boiler workers cooks; cement workers, resistant brick; furnace and pipe builders)	No fibers specified, or unclear	OR
Cohort	Liddell	1997	Canada	9780M	Born 1891-1920	1904-1992	Asbestos Miners And Millers	Chrysotile only	SMR
Cohort	Pang	1997	China	160M, 370F	Not reported	1972-1994	Asbestos Plant	Chrysotile only	SMR
Cohort	Sun	1997	Japan	17344	Not reported	1973-1993	Construction Workers (Construction Workers' Health Insurance Society Of Mie Prefecture)	No fibers specified, or unclear	SMR
Case-control	Cocco	1998	United States	1056 exposed/cases; 5280 controls	1984-1992		Various Industries	No fibers specified, or unclear	OR
Case-control	Parent - amphibole and chrysotile	1998	Canada	250 exposed/cases; Cancer at other site=2290; population based control= 533 controls	1979-1985		Chrysotile: Motor Vehicle Mechanics, Welders and Flame Cutter, Stationary Engineers; Amphibole: Stationary Engineers, Pipe Fitters, Plumbers, Electricians	Mixed chrysotile/amphibole	OR

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Battista	1999	Italy	734	1945-1969	1970-1997	Railway Carriage Construction And Repair Workers	Mixed chrysotile/amphibole	SMR
Case-control	Cocco	1999	United States	41957 exposed/cases; Two controls for each case controls	1984-1996		General Work Population (Female-African American, Female-White; Male – African American; Male – White)	No fibers specified, or unclear	OR
Case-control	Ekstrom	1999	Sweden	567 exposed/cases; 1165 controls	1989-1995		Various Industries	No fibers specified, or unclear	OR
Cohort	Germani	1999	Italy	631W	1979	1980-1997	Asbestos Cement And Textile Industries	Mixed chrysotile/amphibole	SMR
Cohort	Karjalainen	1999	Finland	1287M + 89W with asbestosis, 4708M + 179W with benign pleural disease	1964-1995	1964-1995	General Work Population (Finnish Registry Occupational Diseases)	No fibers specified, or unclear	SIR
Cohort	Berry	2000	UK	3000M, 700W, 1400 insulators	1933-1964 M, 1936-1942W	1951-1980	Asbestos Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Seniori-costantini	2000	Italy	3741	1960-1995	1960-1996	Railway Rolling Factory	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Straif	2000	Germany	8933	1950-1981	1981-1991	Rubber Workers	No fibers specified, or unclear	
Cohort	Szeszenia-Dabrowska	2000	Poland	2525M, 591W	1959-1965	1959-1991	Asbestos Cement	Mixed chrysotile/amphibole	SMR
PMR	Stern	2001	United States	12873M	1972-1996		Construction Plasterers And Cement Masons	No fibers specified, or unclear	PMR
Cohort	Szeszenia-Dabrowska	2002	Poland	907M and 490W	1970-1997	1970-1999	Asbestos Processing Plant, Foundry, Shipyard	No fibers specified, or unclear	SMR
Cohort	Ulvestad	2002	Norway	541M	1942-1976	1953-1999	Asbestos Cement Production	Mixed chrysotile/amphibole	SIR
Cohort	Sun	2003	China	5681	1960-1980	1960-2000	Chrysotile Asbestos Spinning	Chrysotile only	SMR
Cohort	Finkelstein	2004	Canada	25285	1949	1950-1999	Plumbers And Pipefitters	No fibers specified, or unclear	SMR
Cohort	Smailyte	2004	Lithuania	1887	1956-1985	1978-2000	Asbestos Cement Workers	Chrysotile only	SIR
Cohort	Ulvestad	2004	Norway	1116M	1930-1975	1953-1999	Insulation Workers	Mixed chrysotile/amphibole	SIR
Cohort	Jansson	2005	Sweden	260052M	1971-1993	1971-2000	Construction Workers	Chrysotile only	
Case-control	Krstev	2005	Poland	443 exposed/cases; 479 controls	1994-1996		Various Occupations, Industries	No fibers specified, or unclear	OR
Cohort	Parducci	2005	Italy	2341 workers (1585 women 756 men)	1960-1994	2002	Tobacco Production	No fibers specified, or unclear	SMR
Cohort	Wilczynska	2005	Poland	4187M andW	1945-1980	1945-1999	Asbestos Plant Manufacturing Asbestos Yarn, Cloth, Cords, Packings, Stuffing, Brake Linings, Asbestos-Natural Rubber Sheets	Chrysotile only	SMR
Case-control	Jansson	2006	Sweden	189 exposed/cases; 262 controls	1995-1997		Various Occupations	No fibers specified, or unclear	
Cohort	Battista	2007	Italy	2301	1978-1988	1962-2003	Asbestos Insulation	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Hein	2007	United States	3072M andF	1916-1977	1940-2001	Asbestos Textile Plant	Chrysotile only	SMR
Cohort	Sjodahl	2007	Sweden	256357M	1971-1993	1971-2002	Construction Workers	No fibers specified, or unclear	

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Dement	2009	United States; Canada	17345	Not reported	1986-2004	Sheet Metal Workers	No fibers specified, or unclear	SMR
Cohort	Harding	2009	UK	98117	1971-2005	1971-2006	General Work Population, Workplaces Subject To The 1969 Asbestos Regulations	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Loomis	2009	United States	5770	1950-1973	1950-2003	Asbestos Textile Plant	Mixed chrysotile/amphibole	SMR
Cohort	Pesch	2010	Germany	576M	1993-1997	1993-2007	Various Industries Involving Asbestos (Central Registration Agency For Employees Exposed To Asbestos Dust)	Mixed chrysotile/amphibole	SMR
Cohort	Tomioka	2011	Japan	249	1947-1979	1947-2007	Laggers And Boiler Repairers In A Refitting Shipyard	Mixed chrysotile/amphibole	SMR
Case-control	Santibanez	2012	Japan	241 intestinal and 109 diffuse adenocarcinomas exposed/cases; 455 controls	1995-1999		Various Occupations, Industries	No fibers specified, or unclear	OR
Cohort	Wang B	2013	China	586M, 279F	1972	1972-2008	Asbestos Textile	Chrysotile only	SMR
Cohort	Lin	2014	China	1539	Not reported	1981-2006	Asbestos Mining	Chrysotile only	SMR
Cohort	Offermans	2014	Netherlands	58,279	Not reported	1986-1970	General Work Population (The Netherlands Cohort Study)	No fibers specified, or unclear	HR incidence
Wu 2014	Wu	2014	Taiwan	4155	1975-1986	1985-2008	Shipbreaking Workers	Mixed chrysotile/amphibole	SIR
Cohort	Boulanger	2015	France	2024	1978	1978-2009	Asbestos Plant Workers	Mixed chrysotile/amphibole	SIR
Cohort	Repp	2015	Germany	2072	1997-2001	Median time: 11.3 years	General Work Population (Study Of Health In Pomerania)	Mixed chrysotile/amphibole	RR mortality
Cohort	Vandenborre	2015	Belgium	1,397,699 (2056 asbestos workers, 385046 potentially exposed workers, 1010597 reference)	Not reported	2001-2009	Asbestos Cement And Products Manufacturing	Mixed chrysotile/amphibole	SMR
Wu 2015	Wu	2015	Taiwan	4427	1975-1989	1985-2008	Shipbreaking Workers	Mixed chrysotile/amphibole	HR incidence
Cohort	Levin	2016	United States	979	1954-1972	1993-2013	Pipe Insulation Plant (The Tyler Facility)	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Pira	2016	Italy	1083W and 894M	1946-1984	1946-2013	Asbestos Textile	Mixed chrysotile/amphibole	SMR
Cohort	Barbiero A	2018	Italy	2488	1991-2008	1995-2009	Various Groups: Metalworking, Shipbuilding, Electrical Utilities, Insulation	Chrysotile only	SIR
Cohort	Merlo	2018	Italy	3984	1960-1981	1960-2014	Shipyard Workers	No fibers specified, or unclear	SMR
Cohort	Reid	2018	Australia	6500	1943-1966	1943-2009	Wittenoom Asbestos Mine	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Fazzo	2020	Italy	204 (177M, 27W) for mortality outcomes	1958-1993	1986-2018	Asbestos Cement Workers	Mixed chrysotile/amphibole	SMR
Cohort	Ferrante	2020	Italy	974	1917-1990	2013	Asbestos Mine	Chrysotile only	SMR
Case-control	Fukai	2020	Japan	555254 exposed/cases; 128973 controls	2005-2015		Various Occupations, Industries	No fibers specified, or unclear	OR

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Magnani	2020	Italy	51801 (46060M, 5741W)	Years of first exposure ranged from pre-1949 to 1992	1970 onwards (end year not reported)	Various Industries, Including Asbestos-Cement, Rolling Stock Construction And Maintenance, Shipyards, Ship Furnishing, Glassworks, Dockyards, Insulation, Asphalt Rolls, Oven Construction, And Asbestos Miners.	Mixed chrysotile/amphibole	SMR
Case-control	Shah	2020	United States; Canada; Italy; China, Russia, Japan, Spain, Brazil	14465 exposed/cases; 34972 controls	1985-2010		General Work Population	No fibers specified, or unclear	OR
Cohort	Fang	2021	Taiwan	1,043,319	Not reported	1950-2015	Asbestos Related Factories	No fibers specified, or unclear	SIR
Cohort	Fernandes	2021	Brazil	988M	1995-2016	1995-2018	Asbestos-Cement Plant	Mixed chrysotile/amphibole	SMR
Cohort	Dalsgaard	2022	Denmark	9685 (10% of reference cohort= 108987)	1945-1994	1968-2015	General Work Population (The Danish Asbestos Cement Plant Eternit Fabrik A/S region)	Chrysotile only	SIR

Colorectal Cancer Studies

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Selikoff	1979	United States	Cohort 1: 632M, Cohort 2: 17800M (including survivors of the 632 cohort)	1967	1943-1976	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Case-control	Hillerdal (colon and rectum)	1980	Sweden	482M, 420W exposed/ cases; 1158M, 960W controls	1968-1972		General Work Population	No fibers specified, or unclear	Ratio O/E
Case-control	Hardell	1981	Sweden	16 exposed/cases; 137 controls	1978-1979		General Work Population	No fibers specified, or unclear	OR
Cohort	Acheson	1984	UK	4820	1947-1979	1980	Insulation Board Manufacturing	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Ohlson	1984	Sweden	3297	Not reported	1951-1980	Railroad Maintenance Worker	Mixed chrysotile/amphibole	SMR
Cohort	Ohlson	1985	Sweden	1176	1943 to 1976	1951-1982 for mortality; 1958-1979 for morbidity	Asbestos Cement Workers	Mixed chrysotile/amphibole	SMR
Cohort	Peto - Cohort	1985	UK	cohort 1: 145M, cohort 2: 283W, cohort 3: 3211M exposed/cases	1933	1983	Asbestos Textile Factory Workers	Mixed chrysotile/amphibole	SMR
PMR	Zoloth	1985	United States	407	1976-1983		Sheet Metal Workers	No fibers specified, or unclear	PMR
PMR	Cantor	1986	United States	7121	1960-1979		Plumbers, Pipefitters	No fibers specified, or unclear	PMR
Cohort	Gardner	1986	UK	2167	1941-1983	1941-1984	Asbestos Cement Factory Workers	Mixed chrysotile/amphibole	SMR
Cohort	Hodgson	1986	UK	31150	Pre/post 1969, 1972	1981	General Work Population, Workplaces Subject To 1969 Asbestos Regulations	No fibers specified, or unclear	
Cohort	Seidman	1986	United States	820	1941-1954	1982	Asbestos Factory Workers	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Enterline	1987	United States	1074	1941-1980	1941-1969;1941-1973;1941-1980	Production, Maintenance Employees-The Asbestos Company	Mixed chrysotile/amphibole	SMR
Cohort	Hughes	1987	United States	6931M (5492M analytic cohort)	1970	1982	Asbestos Cement Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Tola	1988	Finland	12693	1945-1960	1953-1981	Shipyard, Machine Shop Workers (Welders, Platers, Machinists, Pipe Fitters)	No fibers specified, or unclear	SIR
Case-control	Fredriksson	1989	Sweden	329 exposed/cases; 658 controls	1980-1983		Various Occupations	No fibers specified, or unclear	OR
Case-control	Peters	1989	United States	147M exposed/cases; 147M controls	1974-1982		General Work Population	No fibers specified, or unclear	OR
Cohort	Albin	1990	Sweden	2898 exposed/cases; 1552 controls	1907-1977	1986	Asbestos Cement Workers	Mixed chrysotile/amphibole	RR incidence
Cohort	Piolatto	1990	Italy	1048	1946-1987	1987	Asbestos Miners	Chrysotile only	
Case-control	Glass	1991	New Zealand	19904	1980-1984		Various Industries (Machinery Fitters, Welders, Plumbers, Boilermakers, Electricians, Bricklayers)	No fibers specified, or unclear	OR

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Case-control	Neugut	1991	United States	107 exposed/cases; 509 controls	1986-1988		Various Occupations	No fibers specified, or unclear	OR
Cohort	Selikoff	1991	United States	17800	1967	1977-1986	Asbestos Insulation Workers	Mixed chrysotile/amphibole	SMR
Case-control	Garabrant	1992	United States	746 exposed/cases; 746 controls	1983-1986		Various Occupations	No fibers specified, or unclear	OR
Case-control	Gerhards-sondeverdier	1992	Sweden	569 exposed/cases; 512 controls	1986-1988		Various Occupations	No fibers specified, or unclear	RR
Cohort	Sanden	1992	Sweden	3893	1977-1979	1977-1979 to 1987	Shipyard Workers	Mixed chrysotile/amphibole	SMR
Cohort	Andersen	1993	Norway	690	1920-1966	1960-1991	Lighthouse Keepers- Drinking Rain Water off Asbestos Cement Tiled Roofs	Chrysotile only	SIR
Case-control	Arbman	1993	Sweden	colon=98; rectal=79 exposed/cases; hospital control=371; population controls=430	1984-1986		Various Occupations/Industries	No fibers specified, or unclear	OR
Cohort	Mcdonald	1993	Canada	5351M	Born 1891-1920, surviving up to 1976	1976-1988	Asbestos Miners, Millers	Chrysotile only	SMR
Case-control	Vineis	1993	Italy	131 exposed/cases; 463 controls	1990-1991		Pipe Fitters, Boilermakers- Construction Industry	No fibers specified, or unclear	OR
Case-control	Demers	1994	United States	261 exposed/cases; 183 controls	1984-1987		General Work Population, Construction Industry	No fibers specified, or unclear	OR
Cohort	Meurman	1994	Finland	736M and 167F	1953-1967	1953-1991	Anthophyllite Mines	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SIR
Cohort	Raffn	1996	Denmark	8463	1928-1984	End of 1990	Asbestos Cement Workers	Mixed chrysotile/amphibole	SIR
Case-control	Xu A	1996	China	8887	1980-1989		Iron and Steel Workers	No fibers specified, or unclear	PMR
Cohort	Battista	1999	Italy	734	1945-1969	1970-1997	Railway Carriage Construction and Repair Workers	Mixed chrysotile/amphibole	SMR
Cohort	Germani	1999	Italy	631W	1979	1980-1997	Asbestos Cement, Textile Industries	Mixed chrysotile/amphibole	SMR
Cohort	Karjalainen	1999	Finland	1287M + 89W with asbestosis, 4708M + 179W with benign pleural disease	1964-1995	1964-1995	General Work Population (Finnish Registry of Occupational Diseases)	No fibers specified, or unclear	SIR
Cohort	Tulchinsky	1999	Israel	3057M	1953	1953-1992	Asbestos Cement Workers	Mixed chrysotile/amphibole	SIR
Cohort	Berry	2000	UK	3000M, 700W, 1400 insulators	1933-1964M, 1936-1942W	1951-1980	Asbestos Manufacturing	Mixed chrysotile/amphibole	SMR
Case-control	Dumas (amphibole; chrysotile)	2000	Canada	257 exposed/cases; 533 controls	1975-1985		General Occupational Population	Mixed chrysotile/amphibole	OR
Cohort	Seniori-costantini	2000	Italy	3741	1960-1995	1960-1996	Railway Rolling Factory	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Szeszenia-Dabrowska	2000	Poland	2525M, 591W	1959-1965	1959-1991	Asbestos Cement	Mixed chrysotile/amphibole	SMR
Case-control	Goldberg	2001	Canada	497 exposed/cases; 1514 other-disease controls, 533 population- controls	1979-1985		General Occupational Population (98 Occupations, 77 Industries)	No fibers specified, or unclear	

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
PMR	Stern (intestine, rectum)	2001	United States	12873M	1972-1996		Construction Plasterers, Cement Masons	No fibers specified, or unclear	PMR
Cohort	Szeszenia-Dabrowska	2002	Poland	907M and 490W	1970-1997	1970-1999	Asbestos Processing Plant, Foundry, Shipyard	No fibers specified, or unclear	SMR
Cohort	Ulvestad	2002	Norway	541M	1942-1976	1953-1999	Asbestos Cement Production	Mixed chrysotile/amphibole	SIR
Cohort	Sun	2003	China	5681	1960-1980	1960-2000	Chrysotile Asbestos Spinning	Chrysotile only	SMR
Cohort	Finkelstein	2004	Canada	25285	1949	1950-1999	Plumbers, Pipefitters	No fibers specified, or unclear	SMR
Cohort	Smailyte	2004	Lithuania	1887	1956-1985	1978-2000	Asbestos Cement Workers	Chrysotile only	SIR
Cohort	Ulvestad	2004	Norway	1116M	1930-1975	1953-1999	Insulation Workers	Mixed chrysotile/amphibole	SIR
Cohort	Aliyu	2005	United States	1839 exposed/cases; 7924 controls	1989-1993	2003	General Work Population (Specific High-Risk Trades with asbestos Exposure (Insulation, Sheet Metal, Plumbing, Plasterboard, Ship Fitting, Ship Electrical Work, Boiler Making, Ship Scaling))	No fibers specified, or unclear	RR incidence
Cohort	Parducci	2005	Italy	2341 workers (1585 women 756 men)	1960-1994	2002	Tobacco Production	No fibers specified, or unclear	SMR
Cohort	Wilczynska	2005	Poland	4187M and W	1945-1980	1945-1999	Asbestos Plant Manufacturing Asbestos Yarn, Cloth, Cords, Packings, Stuffing, Brake Linings, And Asbestos-Natural Rubber Sheets	Chrysotile only	SMR
Cohort	Battista	2007	Italy	2301	1978-1988	1962-2003	Asbestos Insulation	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Hein	2007	United States	3072M and F	1916-1977	1940-2001	Asbestos Textile Plant	Chrysotile only	SMR
Cohort	Dement	2009	United States; Canada	17345	Not reported	1986-2004	Sheet Metal Workers	No fibers specified, or unclear	SMR
Cohort	Harding	2009	UK	98117	1971-2005	1971-2006	General Work Population, Workplaces Subject To 1969 Asbestos Regulations	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Loomis	2009	United States	5770	1950-1973	1950-2003	Asbestos Textile Plant	Mixed chrysotile/amphibole	SMR
Cohort	Pesch	2010	Germany	576M	1993-1997	1993-2007	Various Industries Involving Asbestos (Central Registration Agency For Employees Exposed to Asbestos Dust)	Mixed chrysotile/amphibole	SMR
Cohort	Tomioka	2011	Japan	249	1947-1979	1947-2007	Laggers And Boiler Repairers, Refitting Shipyard	Mixed chrysotile/amphibole	SMR
Cohort	Chen	2012	China	124	1981-2008	1981-2008	Shipyard, Construction Manufacturing, Mechanical, Mining, Electricity, Other Asbestos Related Work (Including Textile, Fire Fighters, Incineration).	Mixed chrysotile/amphibole	SMR
Cohort	Wang B	2013	China	586M, 279F	1972	1972-2008	Asbestos Textile	Chrysotile only	SMR
Cohort	Lin	2014	China	1539	Not reported	1981-2006	Asbestos Mining	Chrysotile only	SMR
Cohort	Wu	2014	Taiwan	4155	1975-1986	1985-2008	Shipbreaking Workers	Mixed chrysotile/amphibole	SIR
Cohort	Offermans	2014	Netherlands	58,279	Not reported	1986-1970	General Work Population (The Netherlands Cohort Study)	No fibers specified, or unclear	HR incidence

Design	Author	Year	Country	Sample	Start Years	End Years	Occupation Industry	Fibre	Overall Meta-risk Metric
Cohort	Boulanger	2015	France	2024	1978	1978-2009	Asbestos Plant Workers	Mixed chrysotile/amphibole	SIR
Cohort	Repp	2015	Germany	2072	1997-2001	Median time: 11.3 years	General Work Population (Study of Health In Pomerania)	Mixed chrysotile/amphibole	RR mortality
Cohort	Vandenborre	2015	Belgium	1,397,699 (2056 asbestos workers, 385046 potentially exposed workers, 1010597 reference population)	Not reported	2001-2009	Asbestos Cement, Products Manufacturing	Mixed chrysotile/amphibole	SMR
Cohort	Wu (colon, rectum)	2015	Taiwan	4427	1975-1989	1985-2008	Shipbreaking Workers	Mixed chrysotile/amphibole	HR incidence
Case-control	Kachuri	2016	Canada	colon=931; rectal=840 exposed/cases; 1360 controls	1994-1997		Various Occupations/Industries	No fibers specified, or unclear	OR
Cohort	Levin	2016	United States	979	1954-1972	1993-2013	Pipe Insulation Plant (The Tyler Facility)	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Pira	2016	Italy	1083W and 894M	1946-1984	1946-2013	Asbestos Textile	Mixed chrysotile/amphibole	SMR
Cohort	Paris	2017	France	14515	2003-2005	EnrollMt to 2014 (approximately 10 years)	Asbestos-Exposed Workers Covered (French National Health Insurance Fund	No fibers specified, or unclear	
Cohort	Barbiero A	2018	Italy	2488	1991-2008	1995-2009	Various Groups: Metalworking, Shipbuilding, Electrical Utilities, Insulation	Chrysotile only	SIR
Case-control	El-Zaemey	2018	Australia	918 exposed/cases; 1021 controls	2005-2007		Various Occupations/Industries	No fibers specified, or unclear	OR
Cohort	Merlo	2018	Italy	3984	1960-1981	1960-2014	Shipyard Workers	No fibers specified, or unclear	SMR
Cohort	Reid	2018	Australia	6500	1943-1966	1943-2009	Wittenoom Asbestos Mine	Amphibole only (anthophyllite, actinolyte, tremolite, amosite, crocidolite)	SMR
Cohort	Fazzo	2020	Italy	204 (177M, 27W) for mortality outcomes	1958-1993	1986-2018	Asbestos Cement Workers	Mixed chrysotile/amphibole	SMR
Cohort	Ferrante	2020	Italy	974	1917-1990	2013	Asbestos Mine	Chrysotile only	SMR
Case-control	Fukai	2020	Japan	555254 exposed/cases; 128973 controls	2005-2015		Various Occupations/Industries	No fibers specified, or unclear	OR
Cohort	Magnani	2020	Italy	51801 (46060M, 5741W)	Years of first exposure ranged from pre-1949 to 1992	1970 onwards (end year not reported)	Various Industries (Asbestos-Cement, Rolling Stock Construction/ Maintenance, Shipyards, Ship Furnishing, Glassworks, Dockyards, Insulation, Asphalt Rolls, Oven Construction, Asbestos Miners)	Mixed chrysotile/amphibole	SMR
Cohort	Fernandes	2021	Brazil	988M	1995-2016	1995-2018	Asbestos-Cement Plant	Mixed chrysotile/amphibole	SMR
Cohort	Dalsgaard	2022	Denmark	9685 (10% of reference cohort of 108987 individuals)	1945-1994	1968-2015	General Work Population (Danish Asbestos Cement Plant Eternit Fabrik A/S Region)	Chrysotile only	SIR

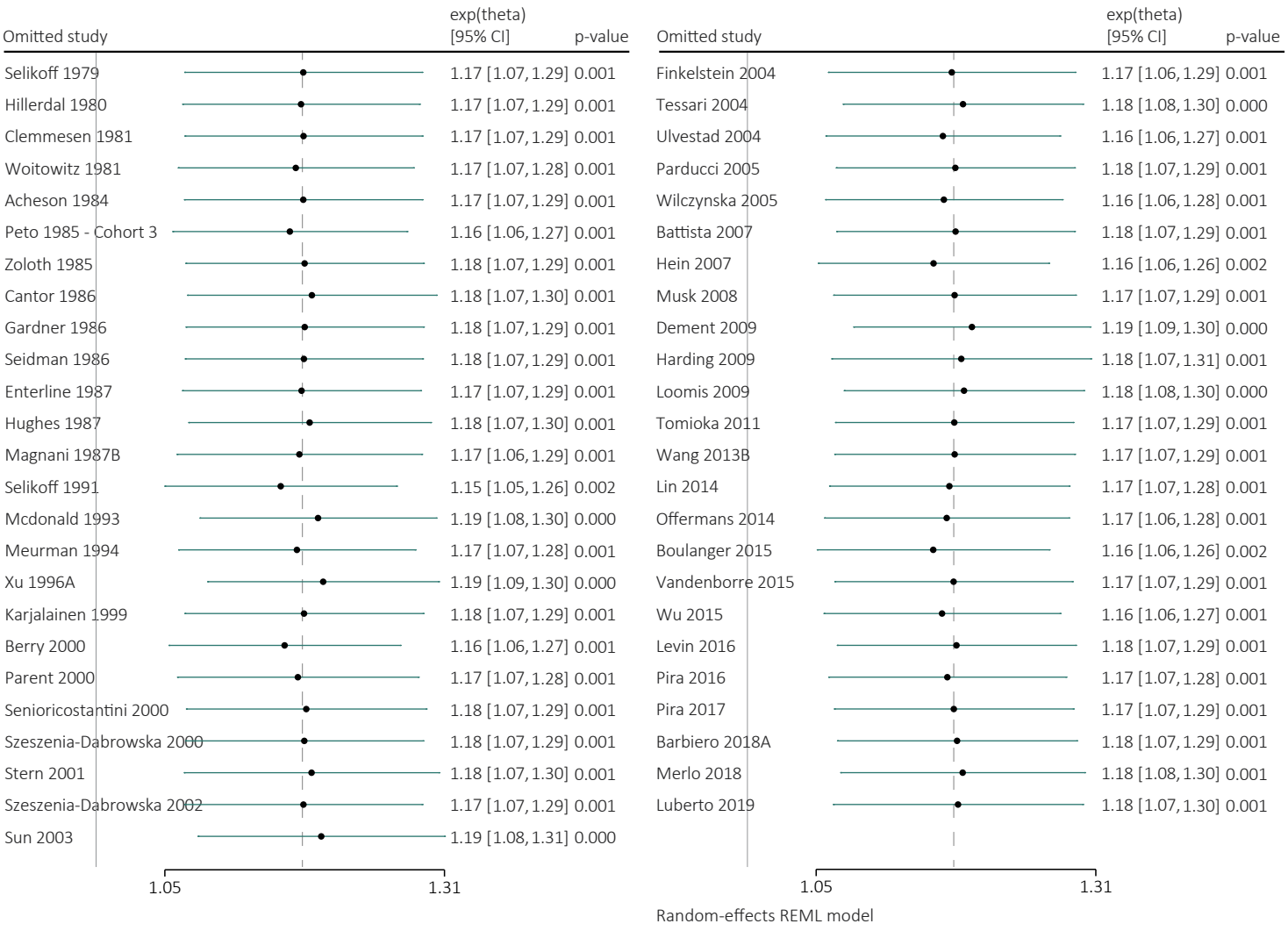
Appendix III | Description of Exposure-Response Studies Included in High/Low Asbestos Exposure Sub-group Analyses by Exposure Characteristics, Esophageal Studies (Illustrative Example)

Study	Description of highest exposure characteristics	Elements of (D)uration (I)ntensity (F)requency	Description of exposure assessment method	Elements of (E)mployment records (W)ork histories (S)urvey assessments, (J)ob Exposure Matrices (D)irect measures (A) Expert assessments (M)edical Records
Peto 1985	Employed 10+ years in asbestos-exposed areas of the plant (scheduled areas), with 20+ years since first employment	D, I, F	Employment records – detailed work history (factory area, occupation, type of work, employment code (unique to job), and scheduled (consistent) areas	E, W
Hodgson 1986	Cumulative exposure >=20 years	D, I, F	Worker survey every 2 years for detailed occupational history and duration of exposure to asbestos (all workers employed in a factory/workplace governed by asbestos regulations (UK)	S, W, M
Meurman 1994	3+ months of heavy exposure	D, I	Employment records, job description and work history (duration, quality and intensity of dusty work), with heavy exposure defined by working in mines or mills	E, W
Gustavsson 1998	High asbestos exposure level vs. control (based on the cut off at the median of cumulative dose among the control group)	D, I, F	Occupational history via interviews, followed by occupational hygienist review to code the intensity and probability of exposure	W, S, A
Berry 2000	Worked 2+ years with severe intensity exposure covered under asbestos regulations in asbestos factory (sectional pipe making, manufacture of insulating material with high asbestos content, textile and mattress sections, openers, disintegrators, disposal of dust)	D, I	Occupational history via employment records, with exposure degree and duration classified using a combination of job length and job characteristics	E, W
Parent 2000	Substantial exposure level (vs. no exposure). Defined as probable or definite confidence, >5 years since first exposure, >=level 4 for concentration by frequency, and >5 years duration of exposure	D, I, F	Questionnaire followed by rating from expert panel to classify exposure groups (defined by confidence of exposure, years since first exposure, concentration X frequency, and duration of exposure)	S, A
Jansson 2005 (esophageal adenocarcinoma and squamous-cell carcinoma)	High exposure level (vs. no exposure).	D, I, F	Job titles, occupational exposure history via on-site surveys by occupational health services, combined with a job exposure matrix developed by industrial hygienist. Exposure levels graded by hygienists based on TLVs	E, S, J, A
Jansson 2006 (esophageal adenocarcinoma)	High cumulative exposure level (vs. no exposure)	D, I, F	Job titles, occupational exposure history via on-site surveys by occupational health services, combined with a job exposure matrix developed by industrial hygienist. Exposure levels graded by hygienists based on TLVs	E, S, J, A
Santibanez 2008	Exposure level high (>0.26 fibres/cm3) vs. low (<= 0.26 fibres/cm3)	D, I, F	Occupational history collected by interview combined with the FINJEM job-exposure matrix to estimate probability X intensity of exposure	S, J
Tomioka 2011	Worked 12+ years (duration of exposure in shipyard by occupation – laggers, boiler repairers)	D, I	Employment records	E
Wu 2015	High (vs. low exposure level within shipbreaking occupations – exposure score >=45.46	D, I, F	Panel of seven experts (hygienists, occupational medicine physicians, risk assessment expert) – scored asbestos exposure intensity and exposure potential by job titles in shipbreaking industry using employment records, gross tonnage of shipbreaking, and exposure scores correlated with direct measures f/cm3 in worksites, to construct high/low exposure categories	E, A, D
Lin 2014	Cumulative exposure level 3 (vs. cumulative exposure level 1)	D, I	Cumulative dust exposures were estimated based on historical dust measurements of different workshops, job titles and employment duration (from employment records)	E, D

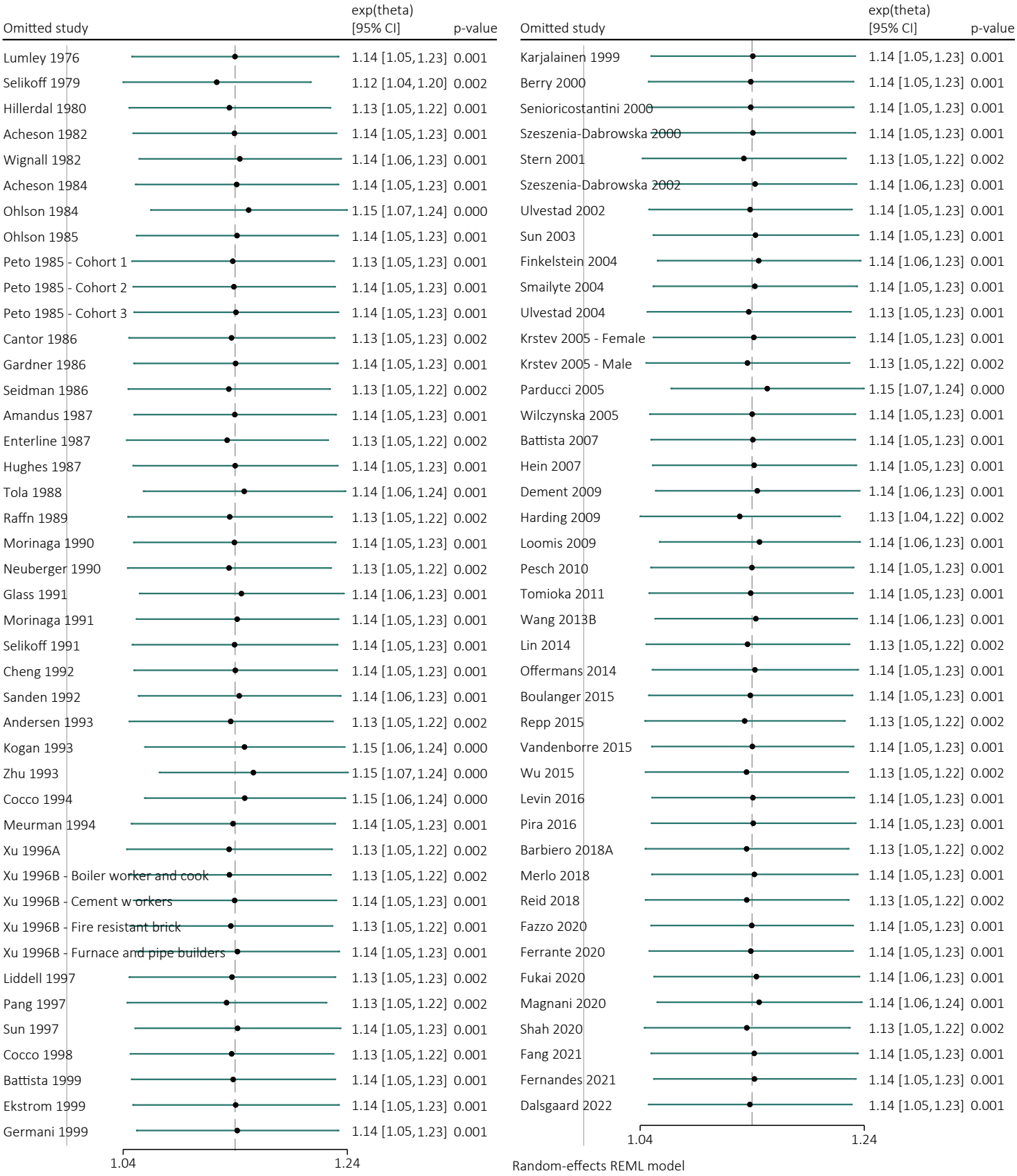
Study	Description of highest exposure characteristics	Elements of (D)uration (I)ntensity (F)requency	Description of exposure assessment method	Elements of (E)mployment records (W)ork histories (S)urvey assessments, (J)ob Exposure Matrices (D)irect measures (A) Expert assessments (M)edical Records
Offermans 2014	Duration of high exposure (probability X intensity of exposure) tertile 3 (vs. tertile 1)	D, I, F	Lifetime occupational history via questionnaire and combined with DOMJEM job-exposure matrix	S, W, J
Boulanger 2015	Cumulative Exposure Index >80 f/mL-year (vs. population of Calvados incidence rates)	D, I, F	Employment and medical records, combined with company specific JEM (date of employment, departure, exposure sector, types of asbestos handled) and annual dust accumulation measurement data in company workshops	E, M, J, D
Clin 2017	Cumulative Exposure Index 64+ f/mL-year (vs. non-exposed reference group)	D, I	Occupational history via questionnaire, followed by industrial hygienist review to calculate a cumulative exposure index for each job (exposure level X duration of employment)	W, S, A
Magnani 2020	Cumulative exposure > 620 f/mL-y (vs. <54 f/mL-y)	D, I, F	Company records combined with industrial hygienist review to estimate the proportion of exposed workers, the percentage of time in asbestos exposing tasks and the minimum and maximum concentrations of asbestos airborne fibres	E, A

Appendix IV | Leave-One-Out Sensitivity Analyses

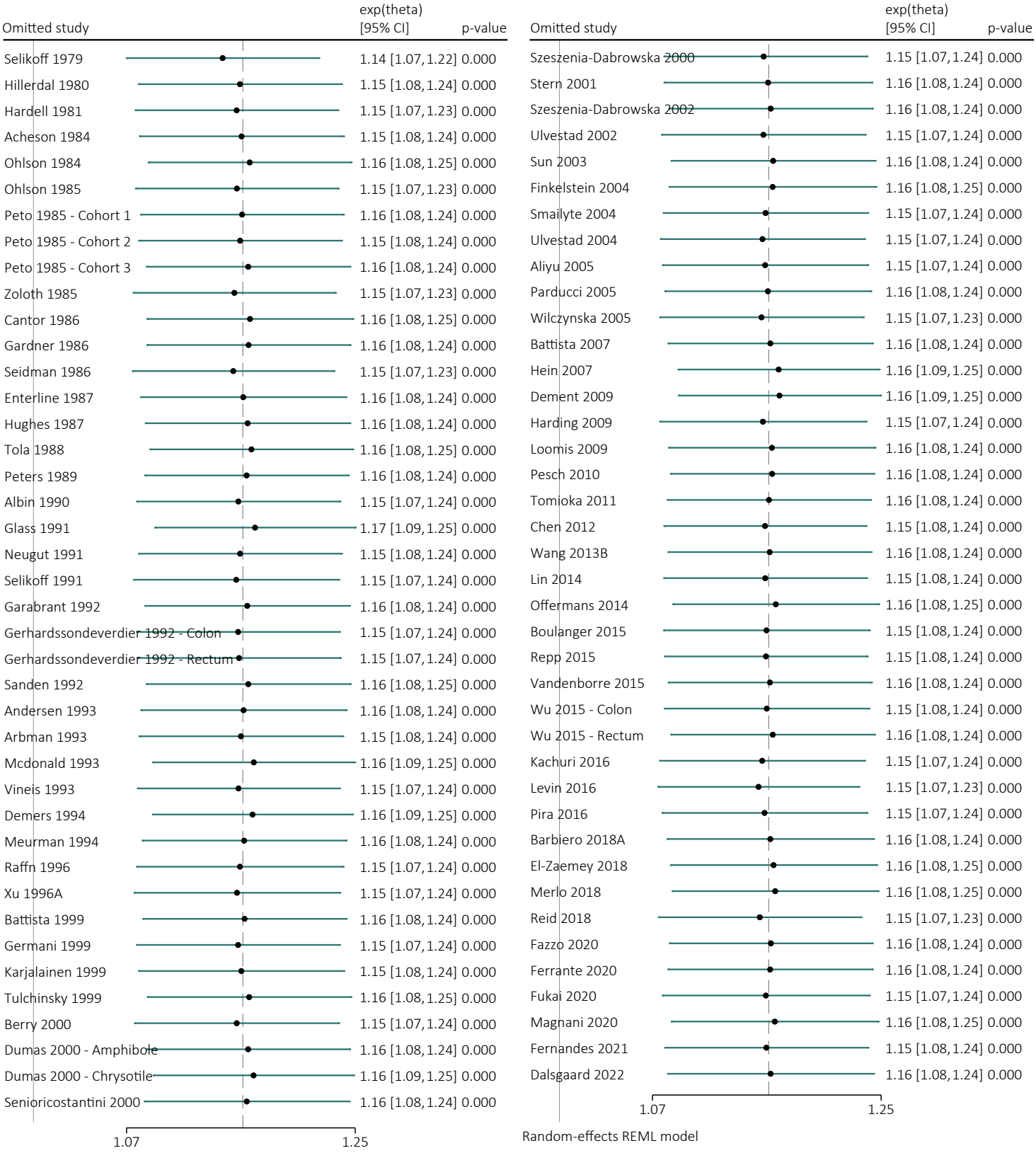
Esophageal Cancer Studies



Stomach Cancer Studies

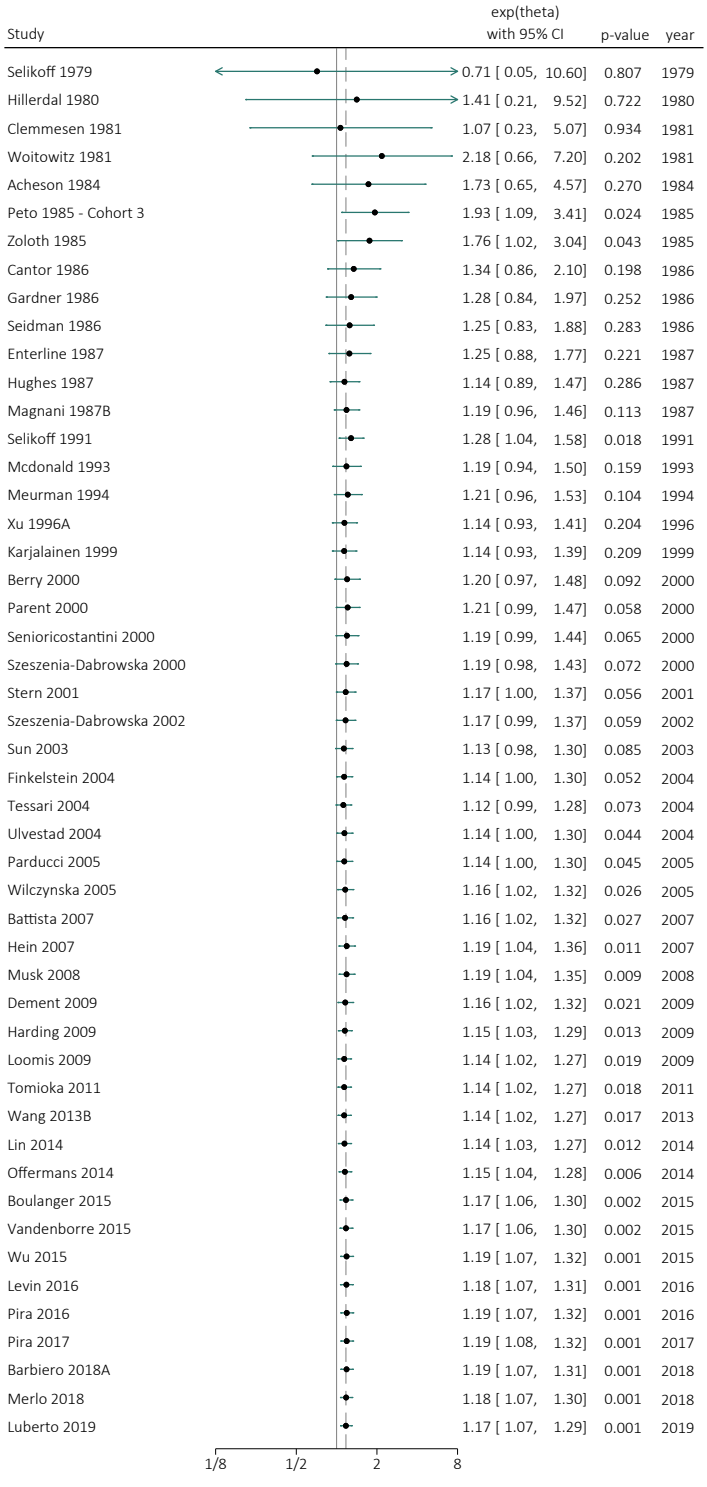


Colorectal Cancer Studies



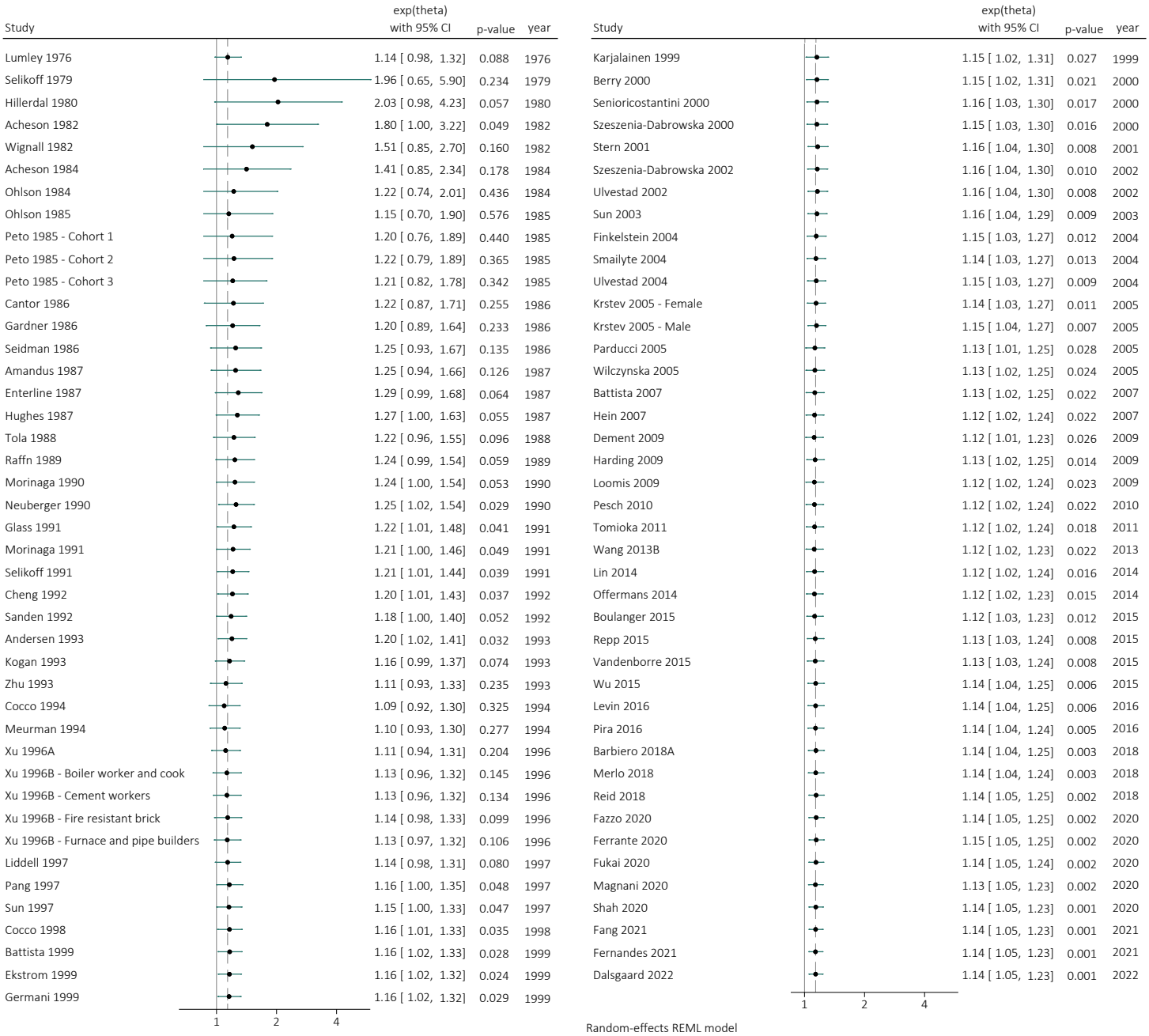
Appendix V | Cumulative Meta-risk Analysis

Esophageal Cancer Studies

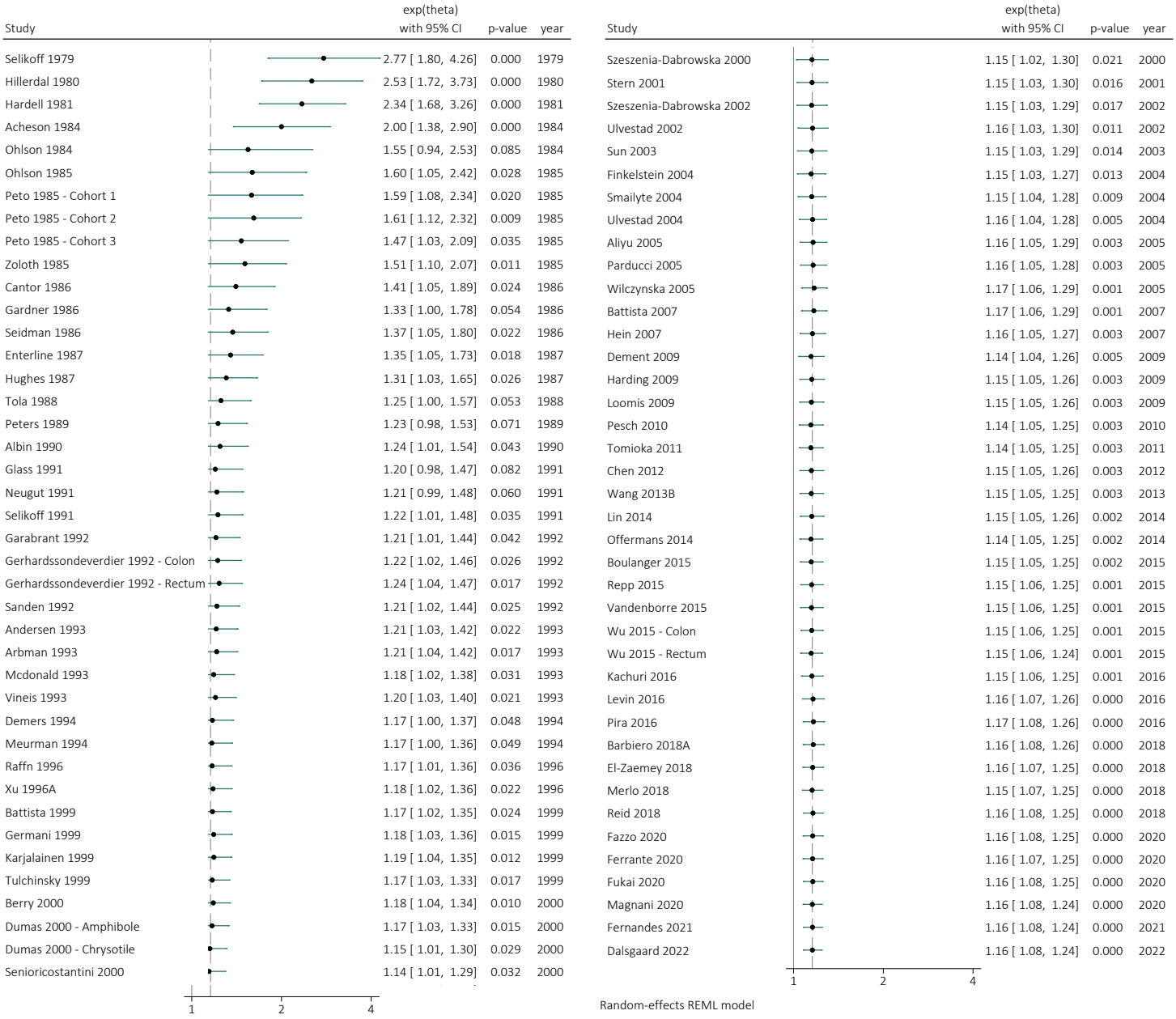


Random-effects REML model

Stomach Cancer Studies



Colorectal Cancer Studies



Appendix VI | Summary of Evidence from Prior Evaluations

Description of Review Publications

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Type	Systematic review and meta-analysis	Systematic/umbrella review (no meta-analysis)	Systematic/umbrella review (no meta-analysis)	Systematic review and meta-analysis	Systematic review and meta-analysis	Systematic review and meta-analysis	Systematic review and meta-analysis	Systematic review and meta-analysis
Inclusion criteria	Cohort and case-control studies	Cohort and case-control studies	Cohort and case-control studies	Cohort studies	Cohort studies	Cohort studies	Cohort studies, plus 2 ecological studies	Cohort and case-control studies
Exposure	Occupational and non-occupational asbestos cohorts	Occupational and non-occupational asbestos cohorts	Occupational and non-occupational asbestos cohorts	Occupational asbestos exposure only	Occupational asbestos exposure only	Occupational asbestos exposure only	Occupational and environmental asbestos exposure	Occupational asbestos exposure only
GI Outcome	Incidence and mortality for esophageal, stomach and colorectal	Incidence and mortality for esophageal, stomach and colorectal	Incidence and mortality for stomach and colorectal	SMR/SIR for stomach cancer only	SMR for stomach cancer only	SMR for colorectal cancer only	SMR for esophageal cancer only	SIR/SMR/OR/HR etc. for esophageal, stomach and colorectal
Language inclusion	English only (reviewed title and abstract of non-English references)	Not explicitly stated	Not explicitly stated	English only	English and Chinese	English only	Not stated	No restrictions on language
Handling of related/ duplicate cohorts in meta-analyses	Removed to avoid double-counting	No meta-analyses conducted	No meta-analyses conducted	"When several publications relating to the same cohort were available, we used the most recent report."	"As some papers on the same cohort study were published several times, only the newest or most informative single article was included."	Re-analysis without duplicates published in a letter to editor.	The review did not remove duplicate cohorts in the meta-analysis (e.g., Frost 2008/Harding 2009, Levin 1998/Levin 2016).	Removed to avoid double-counting
Source of meta risk estimates or conclusions	Esophageal: TABLE 9.1 (TABLE D.3) Stomach: TABLE 10.1 (TABLE D.4) Colorectal: TABLE 11.1 (TABLE D.5)	Page 294 (see Evaluation)	Page 67 (colorectal cancer), Page 72 (stomach cancer)	Figure 1	Figure 2	Figure 2	Figure 2	
Reference	Institute for Occupational Medicine. Asbestos: Selected Cancers. Institute of Medicine of the National Academy of Science. The National Academies Press: Washington DC, 2006.	International Agency for Research on Cancer. Asbestos (chrysotile, amosite, crocidolite, tremolite, actinolite, and anthophyllite). In: Arsenic, metals, fibres, and dusts: A review of human carcinogens. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100C. Geneva, CH: 2012.	Finnish Institute of Occupational Health. Asbestos, asbestosis, and cancer: Helsinki Criteria for Diagnosis and Attribution 2014. Helsinki, FI: 2014.	Fortunato L, Rushton L. Stomach cancer and occupational exposure to asbestos: a meta-analysis of occupational cohort studies. British Journal of Cancer (2015) 112, 1805-1815.	Peng WJ, Jia XJ. Wei BG, Yang LS, Yu Y, Zhang L. Stomach cancer mortality among workers exposed to asbestos: a meta analysis. J Cancer Res Clin Oncol (2015) 141:1141-1149.	Kwak K, Paek D, Zoh KE. Exposure to asbestos and the risk of colorectal cancer mortality: a systematic review and meta-analysis. Occup Environ Med 2019;76:861–871.	Wu CW, Chuang HY, Tsai DL, Kuo TY, Yang CC, Chen HC, Kuo CH. Meta-Analysis of the Association between Asbestos Exposure and Esophageal Cancer. Int. J. Environ. Res. Public Health 2021, 18, 11088.	

Any Versus None Asbestos Exposure Evidence

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Esophageal cancer	Cohort (N=25): 0.99 (0.79 to 1.27)* Case-control (N=2): 1.47 (0.87 to 2.47)	Conclusions not explicitly stated for esophageal cancers	-	-	-	-	1.28 (1.19 to 1.38)	1.17 (1.07 to 1.29)
Stomach cancer	Cohort (N=42): 1.17 (1.07 to 1.28)* Case-control (N=5): 1.11 (0.76 to 1.64)	...positive associations have been observed between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum	...stomach cancer is classified as an entity that is reasonably anticipated to be caused by asbestos (equivalent to IARC Group 2A).	1.15 (1.03 to 1.27)	1.19 (1.06 to 1.34)	-	-	1.14 (1.05 to 1.23)
Colorectal cancer	Cohort (N=41): 1.15 (1.01/1.02-1.31) Case-control (N=13): 1.16 (0.90 to 1.49)	...positive associations have been observed between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum. For cancers of the colorectum, the Working Group was evenly divided as to whether the evidence was strong enough to warrant classification as sufficient.	Ccolorectal cancer is classified as reasonably anticipated to be caused by asbestos (equivalent to IARC Group 2A)…. The magnitude of the reported relative risk varies between studies and is in the range of 1.3 to 5. Assessed study-by-study, SMRs for colorectal cancer tended to be lower than SMRs for lung cancer, indicating that the effect of asbestos, in terms of relative risk, is considerably weaker for colorectal cancer than for lung cancer.	-	-	1.16 (1.05 to 1.29)	-	1.16 (1.08 to 1.24)

* The data point for stomach cancer from the Tola 1988 paper (SMR: 0.80, 95% CI: 0.61-1.02) appears to have been erroneously included in the esophageal cancer analysis and erroneously excluded from the stomach cancer analysis in the IOM evaluation (See Figure 9.1 and Figure 10.1). A possible explanation is that Tola was contacted and provided this esophageal cancer estimate but this was not reported in the IOM 2006 data tables (Table D.3), and does not explain the exclusion of Tola 1988 from the stomach cancer analysis. After reanalyzing the IOM 2006 data tables using a Poisson regression model (i.e., excluding Tola 1988 from the esophageal cancer analysis and including Tola 1988 in the stomach cancer analysis), the corrected meta estimates should be approximately 1.11 for esophageal cancer and 1.13 for stomach cancer. NB: Using a weighted random-effects meta-analysis model, the estimates are all above 1.00 regardless of whether Tola 1988 is included or excluded.

Highest Asbestos Exposure Evidence

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Esophageal cancer	Cohort studies: Range 1.35 (0.81 to 2.27) to 1.43 (0.79 to 2.58)	Evidence was discussed narratively	-	-	-	-	1.84 (1.27 to 2.68)	1.63 (1.29 to 2.06)
Stomach cancer	Cohort studies: Range 1.31 (0.97 to 1.76) to 1.33 (0.98 to 1.79)	Evidence was discussed narratively	Risk estimates tend to be higher in cohorts where heavy exposure to asbestos occurs and with long follow-up periods. There is also evidence that increasing exposure increases this risk. From the standpoint of magnitude of risk, the excess risk associated with ever exposure to asbestos varies between the studies but is of the order of 15–20%. Positive dose–response relationships have been observed between cumulative asbestos exposure and stomach cancer mortality in several cohort studies.	Not reported	Not reported	-	-	1.28 (1.09 to 1.52)
Colorectal cancer	Cohort studies: Range 1.24 (0.91 to 1.69) to 1.38 (1.14 to 1.67)	Evidence was discussed narratively	A few studies showed a significant positive association with cumulative exposure to asbestos, and one study showed a significant association with average exposure. A meta-analysis taking SMR of lung cancer as an indirect indicator of asbestos exposure showed a fairly consistent pattern with increasing SMR for colorectal cancer when the SMR for lung cancer increased. SMR for colorectal cancer increased with severity of asbestosis in one study. Taken together, these dose-response data are supportive of a causal relationship between asbestos exposure and colorectal cancer.	-	-	Not reported	-	1.29 (1.09 to 1.53)

Note: When multiple exposure-response metrics were reported within a given paper, the analyses selected the lowest and highest values of the estimates to produce a lower and upper bound.

Asbestos-related Lung Risk Estimates >2.00

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Esophageal cancer	Not reported	Evidence was discussed narratively	-	-	-	-	Not reported	Cohort studies: 1.40 (1.14 to 1.71)
Stomach cancer	Not reported	Scatterplot of stomach cancer SMR and lung cancer SMR (see Figure 2.1 on Page 249). A positive trend was observed with a correlation coefficient (r2) = 0.66.	… under conditions of asbestos exposure associated with a RR for lung cancer of 2, the estimated RR for stomach cancer is 1.2, with an estimated AF for asbestos- causation of about 17%. When the RR for stomach cancer is 2, the estimated RR for lung cancer is 3.96.	Cohort studies: - Men: 1.46 (1.22 to 1.77); - Women: 1.02 (0.69 to 1.52)	Not reported	-	-	Cohort studies: 1.33 (1.14 to 1.56)
Colorectal cancer	Not reported	Scatterplot of stomach cancer SMR and lung cancer SMR (see Figure 2.2 on Page 253). The trend was positive with a correlation coefficient (r2) of 0.59.	SMRs for colorectal cancer tended to be lower than SMRs for lung cancer, indicating … under conditions of asbestos exposure associated with a RR for lung cancer of 2, the estimated RR for colorectal cancer is 1.1, with an estimated AF for asbestos- causation of about 9%. When the RR for colorectal cancer is 2, the estimated RR for lung cancer is 5.2.	-	-	Cohort studies: 1.44 (1.29 to 1.60)	-	Cohort studies: 1.47 (1.34 to 1.61)

Note: Based on Kwak 2019 re-analysis of the data with excluded cohorts.

Exposure Due to Nature of Work (Industry/Occupation)

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Esophageal cancer	No sub-group analyses by occupation/industry	Evidence was discussed narratively	-	-	-	-	Asbestos mining: 1.07 Asbestos textile: 1.45 Shipyard: 1.39 Others: 1.26	Asbestos cement: 1.12 (0.84 to 1.47) Asbestos mining: 1.13 (0.78 to 1.63) Asbestos insulation: 1.68 (1.19 to 2.36)
Stomach cancer	No sub-group analyses by occupation/industry	Evidence was discussed narratively	Evidence was discussed narratively	Asbestos cement: - Men 1.12; Women 1.27 Asbestos mining: - Men 1.18; Women 0.67 Asbestos insulation: - Men 1.27; Women 0.63 Asbestos textiles: - Men 1.15; Women 1.22 Generic asbestos workers: - Men 1.41; Women 0.87 Other occupations: - Men 0.87; Women 0.87	Asbestos cement: 1.15 Asbestos mining: 1.43 Asbestos textile: 0.94 Refitting shipyard: 1.14 Mix: 1.36	-	-	Asbestos cement: 1.14 (0.99 to 1.32) Asbestos mining: 1.30 (1.14 to 1.49) Asbestos insulation: 1.53 (0.93 to 2.51)
Colorectal cancer	No sub-group analyses by occupation/industry	Evidence was discussed narratively	Evidence was discussed narratively	-	-	Asbestos cement: 1.06 Asbestos mining: 1.09 Asbestos insulation: 1.49 Asbestos textile: 1.19 Miscellaneous: 0.87 Various: 1.35	-	Asbestos cement: 1.21 (1.06 to 1.38) Asbestos mining: 1.15 (0.82 to 1.63) Asbestos insulation: 1.59 (1.14 to 2.23)

Note: Based on Kwak 2019 re-analysis of the data with excluded cohorts

Asbestos Fibre Type

	IOM 2006	IARC 2009/2012	FIOH 2014	Fortunato 2015 stomach	Peng 2015 stomach	Kwak 2019 colorectal	Wu 2021 esophageal	Our analyses
Esophageal cancer	Studies were coded by fibre type, but no sub-group analyses was reported	Evidence was discussed narratively	-	-	-	-	Amosite: 1.14 Crocidolite: 1.20 Chrysotile: 1.27 Mixed asbestos: 1.28	Amphiboles (amosite, crocidolite, etc): 1.16 (1.02 to 1.31) Chrysotile: 1.17 (0.89 to 1.53) Mixed chrysotile/ amphibole: 1.44 (1.20 to 1.73) Unclear type: 1.05 (0.85 to 1.30)
Stomach cancer	Studies were coded by fibre type, but no sub-group analyses was reported	Evidence was discussed narratively	Evidence was discussed narratively	Amosite: - Men 1.25; Women N.R. Crocidolite: - Men 1.09; Women 0.84 Chrysotile: - Men 1.14; Women 1.14 Mixed: - Men 1.13; Women 1.05	Crocidolite: 1.55 Chrysotile: 0.97 Mix: 1.22	-	-	Amphiboles (amosite, crocidolite, etc): 1.35 (1.12 to 1.63) Chrysotile: 1.09 (0.88 to 1.35) Mixed chrysotile/ amphibole: 1.20 (1.04 to 1.41) Unclear type: 0.94 (0.84 to 1.06)
Colorectal cancer	Studies were coded by fibre type, but no sub-group analyses was reported	Evidence was discussed narratively	Evidence was discussed narratively	-	-	Studies were coded by fibre type, but no sub-group analyses was reported	-	Amphiboles (amosite, crocidolite, etc): 1.38 (1.27 to 1.49) Chrysotile: 1.05 (0.88 to 1.26) Mixed chrysotile/ amphibole: 1.23 (1.09 to 1.38) Unclear type: 0.99 (0.89 to 1.10)

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