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Meat consumption and risk of lung cancer: evidence from observational studies

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Background: A number of epidemiological studies have reported inconsistent findings on the association between meat consumption and lung cancer.
Design: We therefore conducted a systematic review and meta-analysis to investigate the relationship between meat consumption and lung cancer risk in epidemiological studies.
Results: Twenty-three case–control and 11 cohort studies were included. All studies adjusted for smoking or conducted in never smokers. The summary relative risks (RRs) of lung cancer for the highest versus lowest intake categories were 1.35 (95% confidence interval (CI) 1.08–1.69) for total meat, 1.34 (95% CI 1.18–1.52) for red meat, and 1.06 (95% CI 0.90–1.25) for processed meat. An inverse association was found between poultry intake and lung cancer (RR = 0.91, 95% CI 0.85–0.97), but not for total white meat (RR = 1.06, 95% CI 0.82–1.37) or fish (RR = 1.01, 95% CI 0.96–1.07).
Conclusions: The relationship between meat intake and lung cancer risk appears to depend on the types of meat consumed. A high intake of red meat may increase the risk of lung cancer by about 35%, while a high intake of poultry decreases the risk by about 10%. More well-designed cohort studies on meat mutagens or heme iron, meat cooking preferences, and doneness level are needed to fully characterize this meat–lung cancer association.
Key words: case–control study, cohort study, lung cancer, meat consumption, meta-analysis

introduction

Lung cancer is the leading cause of cancer-related mortality worldwide [1]. Cigaret smoking is the principal and an indisputable risk factor for lung cancer; however, numerous studies have shown that diet may also be of etiologic importance. Red meat (beef, pork, lamb, and goat from domesticated animals) and processed meat (meats preserved by smoking, curing, or salting, or by addition of chemical preservatives) have been hypothesized to play a role in carcinogenesis, owing to their high levels of saturated fat and heme iron content, and potent mutagens produced during high temperature cooking and meat processing or preservation, including heterocyclic amines (HCAs) [2, 3], polycyclic aromatic hydrocarbons (PAHs) [4, 5], and *N*-nitroso compounds (NOCs) [6]. On the contrary, white meat (poultry and fish), particularly fish intake, has been proposed to lower cancer risk [7–9], due to the relatively lower heme iron content in white meat and long-chain omega-3 polyunsaturated fatty acids present in fish.

Nevertheless, a previous report published in 2007 by World Cancer Research Fund and the American Institute for Cancer Research [10] concluded that the epidemiological evidence for a positive association of total fat, red meat, and processed meat intake with lung cancer risk is suggestive but not sufficient, while the evidence for poultry or fish intake in relation to the lung cancer risk is too limited to draw any conclusions. Recently, >30 epidemiological studies on the consumption of meat and the risk of lung cancer have been accumulated [11–44], and to our knowledge, there has not been any quantitative attempt to summarize the results of this possible meat–lung cancer association. We therefore conducted a systematic review and meta-analysis to investigate this relationship in observational studies.

methods

data sources and searches

We comprehensively identified studies through searching Medline (PubMed), EMBASE and Web of Science through November 2011 for both

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case–control and cohort studies that evaluated the effect of meat consumption on the risk of lung cancer. Our overall search strategy included terms for outcome (pulmonary neoplasm and lung cancer), exposure (meat, red meat, processed meat, white meat, beef, pork, lamb, goat, poultry, and fish), and study design (case–control study, cohort study, follow-up, prospective study, and longitudinal study). In addition, we carried out a broader search on diet and lung cancer so as to identify studies in which the aforementioned terms did not appear in abstracts. The searches were limited to studies of humans and published in English. The reference lists of retrieved articles were also reviewed in order to locate additional relevant studies.

study selection criteria

Published studies were included in the analysis if these (i) had a case–control or cohort design; (ii) evaluated the association between meats (total meat, white meat, red meat, processed meat, poultry, or fish) intake and lung cancer risk, and (iii) presented odds ratio (OR), relative risk (RR), or hazard ratio (HR) estimates with its 95% confidence interval (CI) or standard error (SE). If an article was duplicated or derived from the same study population as previously published, the most recent publication was included.

data extraction and quality assessment

Two investigators (WSY and RQT) independently searched the literature and determined study eligibility and conducted data extraction and quality assessment; discrepancies were settled by consensus or by involving a third reviewer (LX) for adjudication. Data extracted from the included studies are as follows: study name, authors, year of publication, study region, study design (case–control or cohort study), sample size (number of cases and controls or cohort size), length of follow-up for cohort studies, the exposure of meat intake, the study-specific adjusted ORs, RRs, or HRs with their 95% CIs or SEs for the highest category of meat consumption versus the lowest, and variables matched on or adjusted for in the design or data analysis. The total meat definition in our analysis included meat defined in the individual studies as 'all meat' without specifying the type, or 'total meat'. White meat definition in our analysis included meat defined in the individual studies as 'white meat', or poultry and fish.

To assess the study quality, an improved 10-point scoring system based on the Newcastle–Ottawa Scale was used, which has been described in detail elsewhere [45]. Briefly, each study was judged on four broad perspectives: selection of the study groups, comparability of the groups, ascertainment of exposure and outcome, and methods used in data analysis. The maximum score was 10 and a high-quality study was defined as one with a score of ≥7.

data synthesis and analyses

The study-specific most adjusted association estimates were used as the common measure of association across studies and the ORs were considered to be equivalent to RRs or HRs because lung cancer is a rare outcome in humans. The possible heterogeneity in results across studies was examined by using the Cochran Q and I^2 statistics [46]. The null hypothesis that the studies are homogeneous was rejected if the P value for heterogeneity was <0.10 or the I^2 was ≥50%. When substantial heterogeneity was detected, the summary estimate based on the random effects model (DerSimonian and Laird method) [47] was reported. Otherwise, the summary estimate based on the fixed effects model (the inverse variance method) [48] was reported.

Subgroup analyses were conducted on study quality, study design (case–control compared with cohort studies), sex (men compared with women), histologic subtypes (adenocarcinoma, squamous cell carcinoma, and small cell lung cancer), study adjustments (with compared with without smoking, total energy intake, fruit and vegetable intake, PA, and BMI adjustments), and smoking status (current, ever, and never smokers). Due to the limited number of studies that reported risk estimates according to the smoking status, we only conducted subgroup analysis on smoking status for red meat intake.

To assess the influence of selected studies on the pooled results, sensitivity analysis was conducted firstly by excluding studies that reported the lung cancer mortality rather than incidence as an outcome [17, 18, 20], and then by excluding each study one by one and recalculating the combined estimates on the remaining studies. To assess the potential for misclassification bias of exposure (e.g. the highest category of meat consumption for one study may lie in the lowest category in another study), we repeated analysis in studies with the similar exposure categories as well as the reference group.

Publication bias was evaluated using Egger's linear regression [49] and Begg's rank correlation [50] methods. A P value of <0.05 for the two aforementioned tests was considered representative of significant statistical publication bias. All data analyses were carried out using R 2.13.1 (meta 1.6-1) (R Development Core Team, R Foundation for Statistical Computing, Vienna, Austria).

results

literature search and study characteristics

Figure 1 illustrates the search process and the final selection of relevant studies. Our systematic literature search yielded a total of 34 articles on meat intake and lung cancer risk in the final analysis. Descriptive data for the included studies are presented in supplementary Table S1, available at *Annals of Oncology* online. All of the studies were published from 1989 to 2011, consisting of 11 cohort [11–21] and 23 case–control [22–44] studies. The studies were conducted in Asia {$n = 7$ [15–17, 22–24, 28]}, North America {$n = 10$ [11, 12, 14, 18, 20, 21, 34, 36, 39, 43]}, Europe {$n = 11$ [13, 19, 29–33, 37, 38, 40, 42]}, and others {$n = 6$ [25–27, 35, 41, 44]}. The total number of participants in this meta-analysis was 1 797 042 including 30 293 lung cancer cases. All studies used food frequency questionnaires for the assessment of meat consumption. Most studies matched or adjusted for a wide range of potential confounders, including age, physical activity (PA), total energy intake, body mass index (BMI), fruit and vegetable intake, and alcohol consumption. All studies adjusted for smoking or conducted in never smokers.

According to the 10-point scoring system, the study-specific quality scores are summarized in supplementary Table S2, available at *Annals of Oncology* online. The quality scores ranged from 2 to 9. The average scores (standard deviation) of case–control and cohort studies were 6.2 (1.4) and 7.6 (1.3), respectively. The high-quality studies consisted of 11 case–control studies [24–29, 33, 35, 36, 40, 43] and 9 cohort studies [11–14, 16–20].

total meat intake and lung cancer

Among the 24 reports from the 22 studies on total meat intake, 18 reported a positive association, with 9 of them being statistically significant. We found that the high consumption of total meat was significantly associated with a 35% increased risk of lung cancer (RR = 1.35, 95% CI 1.08–1.69) (Table 1,

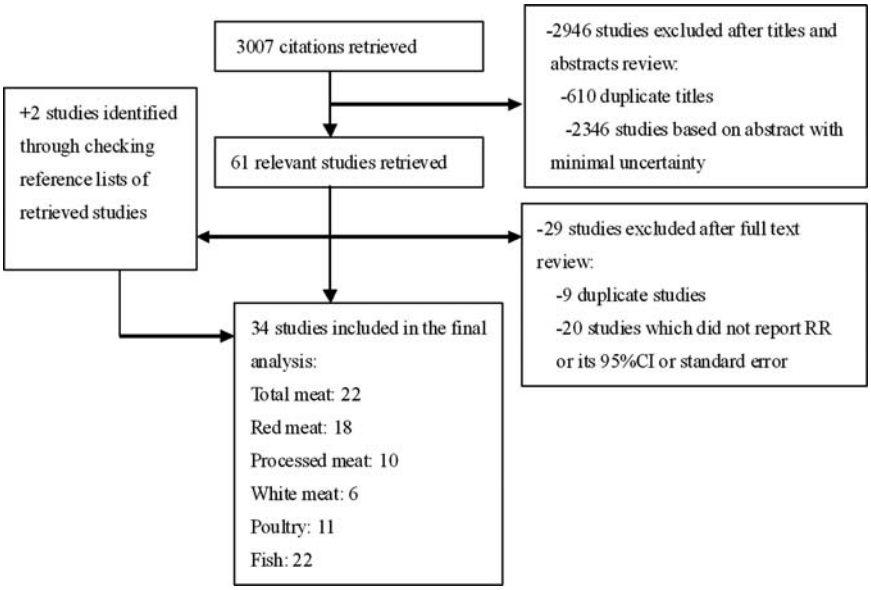

Figure 1 Search strategy and selection of studies for inclusion in the meta-analysis.

Figure 2). Statistically significant heterogeneity was detected ($I^2 = 75.7\%$, $Q = 94.78$, $P < 0.001$). There was no indication of a publication bias, either from Egger's test ($P = 0.857$) or from Begg's test ($P = 0.960$). In subgroup analyses (Table 1), the results were fairly consistent with the overall summary measure when the analyses were restricted to high-quality studies and stratified by study design and histologic type; however, the positive association was not statistically significant in studies that adjusted for fruit and vegetable intake or in those that adjusted for the BMI. Sensitivity analysis by removing each study separately showed that excluding the study by Dosil-Diaz *et al.* [29] resulted in the highest summary estimate (RR = 1.45, 95% CI 1.19–1.76), while excluding the study by Shen *et al.* [28] resulted in the lowest summary estimate (RR = 1.26, 95% CI 1.02–1.56); sensitivity analysis where we omitted two studies [18, 20] that reported the risk estimates for lung cancer mortality rather than incidence showed similar results (RR = 1.33, 95% CI 1.05–1.70); sensitivity analysis in studies with the similar categories of total meat [16, 19, 33, 34, 38, 42] revealed that persons consuming meat >3 times per week had a RR of 1.29 (95% CI 1.02–1.63) compared with those consuming <2 times per week.

red and processed meat intakes and lung cancer risk

Our analysis of 18 studies on red meat consumption and lung cancer yielded a summary RR of 1.34 (95% CI 1.18–1.52) (Figure 3). This positive association was observed across all subgroup analyses, regardless of study quality, study design, smoking status, sex, histologic subtype (Table 1), and adjustments for total energy intake, fruit and vegetable intake, PA, and BMI (data not shown). We found no evidence of publication bias (Egger's test: $P = 0.799$; Begg's test: $P = 0.952$); however, substantial heterogeneity was observed in these studies ($I^2 = 63.9\%$, $Q = 55.35$, $P < 0.001$). There was no indication of increased risk for lung cancer when we combined the 10 studies of processed meat intake (RR = 1.06, 95% CI 0.90–1.25; $I^2 = 79.5\%$, $Q = 58.42$, $P < 0.001$) (Table 1, supplementary Figure S1, available at *Annals of Oncology* online). Like total meat intake, the sensitivity analyses conducted for processed meat did not alter the main results (data not shown).

white meat, poultry, fish intakes, and lung cancer risk

A high poultry intake can weakly decrease the risk of lung cancer (RR = 0.91, 95% CI 0.85–0.97) with an I^2 of 34.7% and Q statistic of 16.85 ($P = 0.112$) (Figure 4). The combined results were consistent among high-quality studies with a RR of 0.89 (95% CI 0.84–0.95), but not statistically significant within either cohort or case–control studies separately, with only few studies of each design ($n = 3$ and 8, respectively). Intake of fish (supplementary Figure S2, available at *Annals of Oncology* online) and total white meat (supplementary Figure S3, available at *Annals of Oncology* online) was not associated with a lower risk of lung cancer, with summary RRs of 1.01 (95% CI 0.96–1.07) and 1.06 (95% CI 0.82–1.37), respectively. These null associations were unchanged in subgroup or sensitivity analyses that accounted for study quality, study design, statistical adjustments, type of outcome reported, and levels of meat consumed (data not shown). No significant publication bias was observed for total white meat (Egger's test: $P = 0.310$; Begg's test: $P = 0.189$), poultry (Egger's test: $P = 0.913$; Begg's test: $P = 0.493$), or fish (Egger's test: $P = 0.594$; Begg's test: $P = 0.172$).

discussion

This, to our knowledge, is the first meta-analysis to explore the relationship between meat consumption and lung cancer risk. In the present study, we found that the total meat intake is positively associated with the risk of lung cancer. Nonetheless,

Table 1. Summary relative risks (RRs) of the association between meat consumption and lung cancer risk

	No. of studies	RR (95% CI)	Q statistic	P value for heterogeneity	I^2 value (%)
Overall studies					
Total meat	22	1.35 (1.08–1.69)	94.78	<0.001	75.7
Red meat	18	1.34 (1.18–1.52)	55.35	<0.001	63.9
Processed meat	10	1.06 (0.90–1.25)	58.42	<0.001	79.5
White meat	6	1.06 (0.82–1.37)	14.20	0.014	64.8
Poultry	11	0.91 (0.85–0.97)	16.85	0.112	34.7
Fish	22	1.01 (0.96–1.07)	40.76	0.013	43.6
Subgroup analyses for total meat					
High-quality studies (scores ≥7)	14	1.41 (1.06–1.87)	67.94	<0.001	77.9
Study design					
Case–control studies	16	1.38 (1.04–1.85)	90.51	<0.001	81.2
Cohort studies	6	1.30 (1.05–1.60)	4.12	0.532	0
Adjustments in models					
Smoking					
Yes	22	1.35 (1.08–1.69)	94.78	<0.001	75.7
No	–	–	–	–	–
Total energy intake					
Yes	5	1.52 (1.27–1.83)	6.49	0.165	38.4
No	17	1.27 (1.13–1.44)	85.74	<0.001	79.0
Fruit and vegetable intake					
Yes	7	1.12 (0.74–1.71)	53.17	<0.001	88.7
No	15	1.49 (1.15–1.94)	41.02	<0.001	61.0
Body mass index (BMI)					
Yes	4	1.22 (0.71–2.11)	21.45	<0.001	86.0
No	18	1.29 (1.08–1.80)	73.01	<0.001	74.0
Histologic subtypes					
Adenocarcinoma	8	1.23 (1.04–1.46)	44.42	<0.001	66.2
Squamous cell carcinoma	6	1.47 (1.31–1.66)	14.71	0.065	45.6
Small cell lung cancer	4	1.30 (1.14–1.49)	5.00	0.660	0
Subgroup analyses for red meat					
High-quality studies (scores ≥7)	9	1.32 (1.11–1.57)	49.84	<0.001	79.9
Study design					
Case–control studies	13	1.42 (1.16–1.74)	36.41	<0.001	67.0
Cohort studies	5	1.20 (1.10–1.30)	2.50	0.927	0
Smoking status					
Never smoking	5	1.66 (1.31–2.11)	8.00	0.156	37.5
Ever smoking	3	1.52 (1.07–2.16)	17.43	<0.001	82.8
Current smoking	5	1.41 (1.10–1.80)	21.82	<0.001	77.1
Gender					
Men	6	1.30 (1.02–1.66)	13.47	0.019	62.9
Women	9	1.23 (1.00–1.50)	16.29	0.038	50.9
Subgroup analyses for processed meat					
High-quality studies (scores ≥7)	8	1.07 (0.90–1.27)	55.90	<0.001	82.1
Study design					
Case–control studies	6	1.05 (0.75–1.49)	42.88	<0.001	86.0
Cohort studies	4	1.05 (0.92–1.19)	9.84	0.080	49.2
Gender					
Men	5	1.13 (0.85–1.49)	15.52	0.003	74.2
Women	4	0.95 (0.84–1.07)	5.42	0.147	44.7
Subgroup analyses for white meat					
High-quality studies (scores ≥7)	3	1.27 (0.79–2.03)	10.02	0.007	80.0
Study design					
Case–control studies	5	1.13 (0.78–1.62)	13.71	0.008	70.8
Cohort studies	1	0.95 (0.79–1.14)	N/A	N/A	N/A
Gender					
Men	2	1.09 (0.83–1.42)	0.39	0.531	0
Women	2	1.74 (0.54–5.58)	7.76	0.005	87.1

Continued

Table 1.. *Continued*

	No. of studies	RR (95% CI)	*Q* statistic	*P* value for heterogeneity	I^2 value (%)
Subgroup analyses for poultry					
High-quality studies (scores ≥7)	5	0.89 (0.84–0.95)	7.76	0.170	35.5
Study design					
Case–control studies	8	0.89 (0.75–1.06)	12.72	0.122	37.1
Cohort studies	3	0.95 (0.64–1.39)	4.10	0.129	51.2
Gender					
Men	5	0.83 (0.62–1.11)	8.08	0.089	50.5
Women	4	0.99 (0.67–1.48)	4.32	0.229	30.5
Subgroup analyses for fish					
High-quality studies (scores ≥7)	13	1.02 (0.96–1.08)	18.80	0.173	25.5
Study design					
Case–control studies	15	0.97 (0.83–1.15)	31.52	0.008	52.4
Cohort studies	7	1.02 (0.96–1.08)	9.17	0.241	23.7
Gender					
Men	6	1.03 (0.87–1.22)	4.98	0.419	0
Women	7	0.92 (0.66–1.29)	20.38	0.002	70.6

[a]CI, confidence interval; N/A, not available; RR, relative risk; –, no available studies.

this link was attenuated by other factors (adjustment for fruit and vegetable intake and BMI), and the association varied by the type of meat consumed. Specifically, a high intake of red meat, but not processed meat, was observed to increase the risk of lung carcinoma; while a higher consumption of poultry, but not total white meat or fish intake, was observed to decrease the risk of lung carcinoma, and these observed results were robust across the subgroup and sensitivity analyses that accounted for study quality, study design, smoking status (for red meat), sex, histologic subtype, statistical adjustments, type of outcome reported (lung cancer incidence and mortality), the influence of each individual study, and the potential for misclassification of meat consumption.

It has been hypothesized that mutagenic byproducts, including HCAs [2, 3] and PAHs [4, 5], from cooking meat could contribute to lung carcinogenesis. However, evidence from the epidemiological studies of HCAs and PAHs and lung cancer has been inconclusive and limited to a few investigations [12, 14, 22, 25, 27]. Two studies [14, 39] have shown that the intake of well-done meat was significantly associated with the elevated risk of lung cancer, whereas a recent study by Tasevska *et al.* [12] found no such effect. A second possible mechanism for the adverse effect of red meat, specifically, on lung cancer is via its high content of heme iron which may act as a pro-oxidant and catalyze lipid peroxidation causing DNA damage in tissues [51]. Heme iron has also been shown to induce endogenous formation of NOCs [52]. In addition, high levels of saturated fat present in red meat may be associated with the increased risk of lung cancer, but a study that pooled the raw data from 12 prospective cohorts failed to show such a relation [53]. A possible explanation for the differences in associations between red and processed meats is that cooking red meat is more likely to be over an open grill and produce PAHs than cooking processed meat. However, the mechanism by which poultry intake alone may be associated with a lower lung cancer risk is not well understood, but may be possibly due to its lower content of heme iron compared with red meat. Another explanation is that high poultry eaters often have a healthier overall eating pattern and lifestyle [54].

Strengths of our study include a large sample size (30 293 cases among 1 797 042 participants) and no significant evidence of publication bias. However, several limitations to this meta-analysis should be noted. First, as a meta-analysis of observational data, the possibility of recall and selection biases cannot be ruled out. However, cohort studies, which are less susceptible to bias, showed similar results to case–control studies, indicating that the findings were unlikely to be attributed to recall and selection biases. Second, because of the inability to fully adjust for various confounders, particularly for total energy intake, fruit and vegetable intake, PA, and BMI, which tend to be highly correlated to consumption of most foods and nutrients including meats, and could also be an independent risk factor for lung cancer, may have confounded the reported links making the independent effect of meat intake difficult to determine. For example, the pronounced association between total meat intake and the increased risk of lung carcinoma was no longer observed among studies that adjusted for fruit and vegetable intake [12, 22, 25, 26, 29, 39, 43] or for the BMI [22, 39, 41]. Nonetheless, when we explored the associations for other types of meat (i.e. red meat, processed meat, total white meat, poultry, and fish), the aforementioned variables did not appear to attenuate the findings, given the consistent results observed in each stratum of subgroup analysis. Moreover, since smoking is the most important risk factor for lung cancer, all of included studies adjusted for smoking in statistical models or were conducted among nonsmokers [22, 30, 33, 34, 38, 40]. When we estimated the effect of red meat by smoking status, the results were similar in each stratum. Third, there was a statistically significant heterogeneity across studies with the exception of the studies on poultry intake. For studies on total meat and

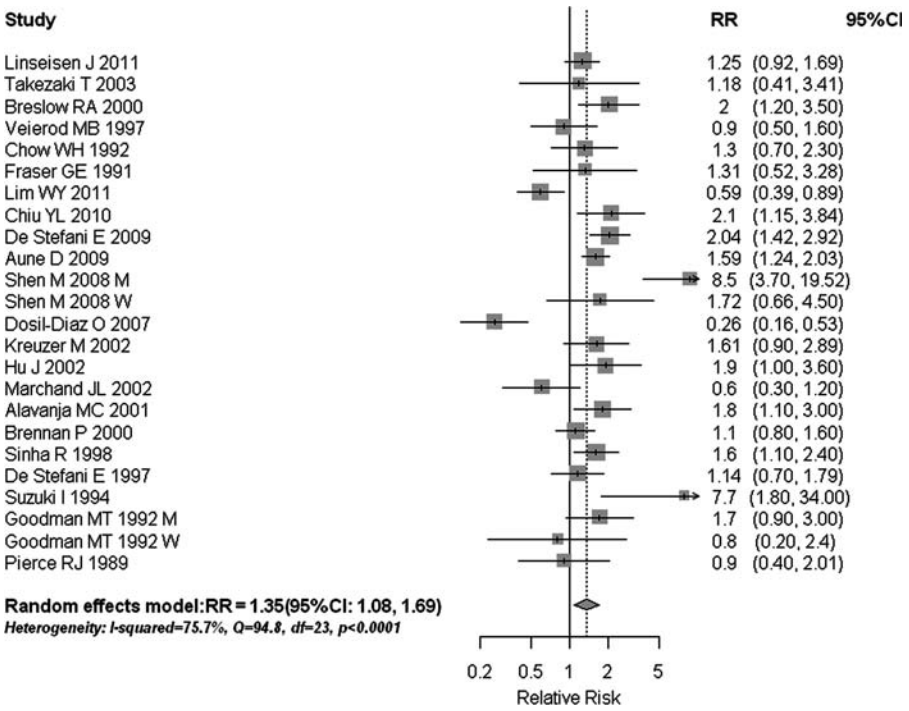

Figure 2 Estimates (95% CIs) of total meat consumption and lung cancer risk. Squares represent study-specific estimates [size of the square reflects the study-specific statistical weight (i.e. inverse of the variance)]; horizontal lines represent 95% CIs; and diamonds represent summary estimates with corresponding 95% CIs. M, men; W, women.

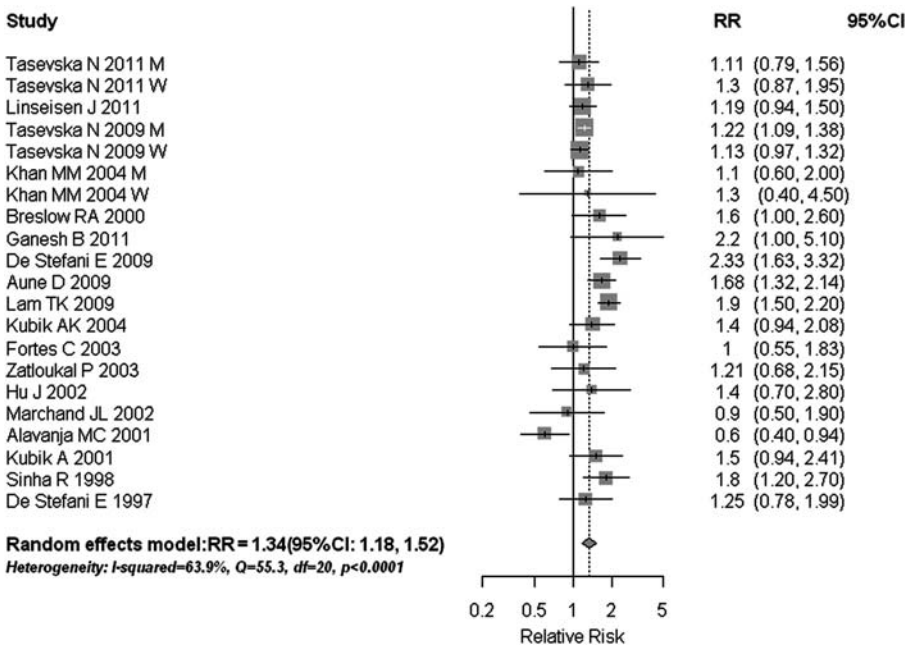

Figure 3 Estimates (95% CIs) of red meat consumption and lung cancer risk. Squares represent study-specific estimates [size of the square reflects the study-specific statistical weight (i.e. inverse of the variance)]; horizontal lines represent 95% CIs; and diamonds represent summary estimates with corresponding 95% CIs. M, men; W, women.

any other types of meat, the heterogeneity was none or smaller when restricted analysis in cohort studies. The little heterogeneity was also shown in never smokers for red meat, in female sex for processed meat, and in male sex for fish. In addition, studies with data analysis that adjusted for PA or did not adjust for the BMI, or fruit and vegetable intake revealed the homogenous results for any types of meat intake (data not shown). These suggested that the heterogeneity may be partly

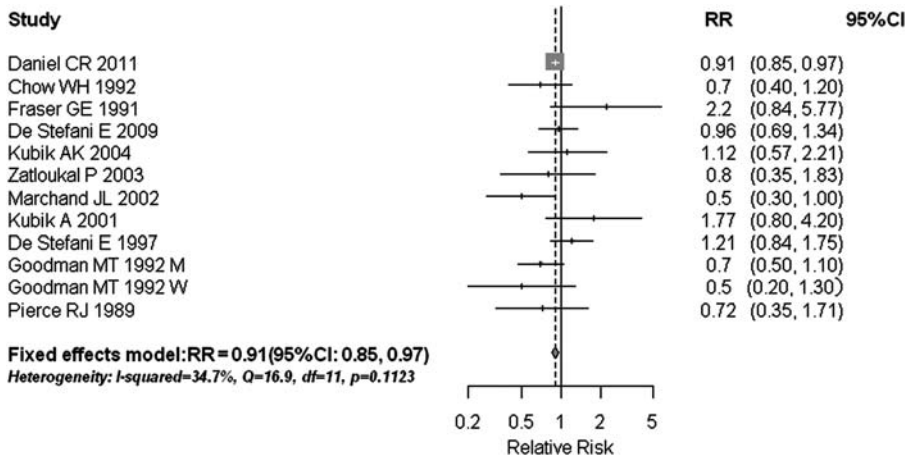

Figure 4 Estimates (95% CIs) of poultry consumption and lung cancer risk. Squares represent study-specific estimates [size of the square reflects the study-specific statistical weight (i.e. inverse of the variance)]; horizontal lines represent 95% CIs; and diamonds represent summary estimates with corresponding 95% CIs. M, men; W, women.

due to the difference in study design, study populations, and analytic strategies. Although heterogeneity still remained in some subgroups (Table 1), indicating that other unknown factors may also contribute to the aforementioned heterogeneity, results from subgroup analysis cannot alter the main findings in our study. Fourth, because the majority of studies used food frequency questionnaires to collect data regarding the meat consumption, our findings are likely to be influenced by the misclassification of exposure. In cohort studies, this misclassification would likely be non-differential if the exposure variable was dichotomous, and thereby result in an underestimate of the true association, whereas the influence of a misclassification on the results in case–control studies is less predictable. Lastly, due to the different methods used to assess and categorize meat intake among studies (supplementary Table S1, available at *Annals of Oncology* online), we were unable to evaluate potential dose–response trends between meat intake and lung cancer risk.

In conclusion, our meta-analysis of 34 epidemiological studies suggests that a high intake of red meat may increase the risk of lung cancer by ∼35%, while the high intake of poultry may decrease the risk by about 10%. There was no evidence that the consumption of processed meat, total white meat, or fish was related to lung cancer risk. Cohort studies with long-term follow-up and large sample sizes that fully adjust for potential confounders, such as total energy intake, BMI, PA, and other dietary factors that are highly correlated to meat consumption, are warranted to reach more definitive conclusions. In addition, additional research on meat type, heme iron, cooking method, doneness level, and consumption of meat mutagens should be conducted in order to test the different possible mechanisms for the effect of meat on lung carcinoma.

funding

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disclosure

The authors have declared no conflicts of interest.

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