Red and processed meat consumption and risk of pancreatic cancer: meta-analysis of prospective studies

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BACKGROUND: Whether red and processed meat consumption is a risk factor for pancreatic cancer remains unclear. We conducted a meta-analysis to summarise the evidence from prospective studies of red and processed meat consumption and pancreatic cancer risk.

METHODS: Relevant studies were identified by searching PubMed and EMBASE databases through November 2011. Study-specific results were pooled using a random-effects model.

RESULTS: Eleven prospective studies, with 6643 pancreatic cancer cases, were included in the meta-analysis. An increase in red meat consumption of 120 g per day was associated with an overall relative risk (RR) of 1.13 (95% confidence interval (CI) = 0.93–1.39; P_heterogeneity = 0.001). Red meat consumption was positively associated with pancreatic cancer risk in men (RR = 1.29; 95% CI = 1.08–1.53; P_heterogeneity = 0.28; five studies), but not in women (RR = 0.93; 95% CI = 0.74–1.16; P_heterogeneity = 0.21; six studies). The RR of pancreatic cancer for a 50 g per day increase in processed meat consumption was 1.19 (95% CI = 1.04–1.36; P_heterogeneity = 0.46).

CONCLUSION: Findings from this meta-analysis indicate that processed meat consumption is positively associated with pancreatic cancer risk. Red meat consumption was associated with an increased risk of pancreatic cancer in men. Further prospective studies are needed to confirm these findings.

Keywords: diet; meat; meta-analysis; pancreatic cancer; prospective studies; review

Pancreatic cancer is one of the most fatal types of cancer, with a 5-year relative survival of about 5.5% (Howlader et al., 2010). Thus, identification of risk factors for this cancer is of great public health importance. Dietary factors could conceivably influence the risk of developing pancreatic cancer, although no dietary factor has been convincingly associated with pancreatic cancer risk (2007). High consumption of red meat and/or processed meat has been associated with increased risk of some gastrointestinal cancers, such as colorectal (Larsson and Wolk, 2006; Chan et al., 2011) and stomach cancer (Larsson et al., 2006b). Whether red and processed meat consumption is a risk factor also for pancreatic cancer remains unclear. We therefore conducted a dose–response meta-analysis of prospective studies to examine the associations of red and processed meat consumption with pancreatic cancer risk.

MATERIALS AND METHODS

Search strategy and study selection

To identify prospective studies of red and processed meat consumption and pancreatic cancer risk, we conducted a literature search in PubMed and EMBASE databases for articles published in any language from January 1966 through November 2011. The following search terms were used: ‘meat’ or ‘foods’ and ‘pancreatic cancer’ or ‘pancreatic neoplasm’, and ‘cohort’ or ‘prospective’, or ‘nested case–control’. In addition, we searched the reference lists of retrieved articles to identify further studies.

To be included in our meta-analysis, studies had to (1) have a prospective design and with pancreatic cancer incidence or mortality as the outcome; and (2) provide relative risks (RRs) with 95% confidence intervals (CI) of pancreatic cancer for at least three categories (or as a continuous variable) of red meat and/or processed meat consumption.

Data extraction

The following data were extracted from each publication: the first author’s last name, year of publication, country in which the study was performed, sex, age, sample size, duration of follow-up, variables adjusted for in the multivariable model, and the RRs with CIs for each category of meat consumption. From each study, we extracted the RRs that reflected the greatest degree of control for potential confounders.

Statistical analysis

Relative risks from individual studies and corresponding s.e. (derived from the CIs) were transformed to their natural logarithms to stabilise the variance and normalise the distributions. We used the method proposed by Greenland and Longnecker.
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(1992) and Ortsini et al (2006) to compute the trend from the correlated log RRs across categories of meat consumption. This method requires that the distribution of cases and person-time (or number of participants), and the RR with its variance estimate for at least three quantitative exposure categories be known. When meat consumption was expressed in 'servings' or 'times', we rescaled the consumption to grams per day using 120 g per day as the standard portion size for total and fresh red meat and 50 g as the standard portion size for processed meat (Norat et al, 2002). For each study, the median or mean level of consumption for each consumption category was assigned to each corresponding RR. When the median or mean consumption per category was not reported in the article, we assigned the midpoint of the upper or lower boundary of the highest or lowest category as the average consumption. If not provided, we assumed that it had the same amplitude as the closest category. If the amount of red meat per category was not specified in the article (Zheng et al, 1993; Coughlin et al, 2000; Isaksson et al, 2002), we estimated the amount using information from another article on meat consumption and disease in the same study population (Hsing et al, 1998; Chao et al, 2005; Rodriguez et al, 2006) or in a similar population with the same exposure (Stolzenberg-Solomon et al, 2002). We used an increase in red and processed meat consumption of 120 and 50 g per day, respectively, which corresponds to a standard serving. We combined the RRs from each study by the method of DerSimonian and Laird (1986), using the assumptions of a random effects model, which takes into account both within- and between-study variability. We checked for nonlinearity of the dose–response relationship between meat consumption and pancreatic cancer by estimating polynomial models.

Statistical heterogeneity among study results was investigated using the I²-statistics (Higgins and Thompson, 2002). We conducted analyses stratified by geographical area (United States and Europe) and sex. Publication bias was examined with Egger’s regression test (Egger et al, 1997). All statistical analyses were conducted with Stata (StataCorp, College Station, TX, USA). P-values were two-sided and P < 0.05 was considered statistically significant.

RESULTS

Study characteristics

We identified 13 prospective studies (Mills et al, 1988; Hirayama, 1989; Zheng et al, 1993; Coughlin et al, 2000; Isaksson et al, 2002; Stolzenberg-Solomon et al, 2002, 2007; Michaud et al, 2003; Nöthlings et al, 2005; Lin et al, 2006; Larsson et al, 2006a; Heinen et al, 2009; Inoue-Choi et al, 2011) that were potentially eligible for inclusion in the meta-analysis. Two studies were excluded, because the exposure was total meat, including white meat (poultry and fish; Mills et al, 1988), or the article was a review about the epidemiology of pancreatic cancer in Japan (Hirayama, 1989). The remaining 11 studies (Zheng et al, 1993; Coughlin et al, 2000; Isaksson et al, 2002; Stolzenberg-Solomon et al, 2002, 2007; Michaud et al, 2003; Nöthlings et al, 2005; Lin et al, 2006; Larsson et al, 2006a; Heinen et al, 2009; Inoue-Choi et al, 2011) were eligible for inclusion in the meta-analysis. Among these studies, six were carried out in the United States, four in Europe, and one in Japan (Table 1). The study population consisted of men and women in six studies: of only women in three studies, and of only men in two studies. Sample sizes ranged from 17 633 – 110 308, and the number of pancreatic cancer cases varied from 57 to 3751. Combined, these studies involved 6643 pancreatic cancer cases and a total of 2 307 787 participants. All studies adjusted for age and smoking, and most studies also adjusted for energy intake (n = 7). Fewer studies controlled for body mass index (n = 2) and/or history of diabetes (n = 5).

Red meat

Eleven studies examined the association between consumption of fresh red meat (Michaud et al, 2003; Nöthlings et al, 2005; Lin et al, 2006; Larsson et al, 2006a; Heinen et al, 2009), pork (Isaksson et al, 2002), or total red meat (including processed meat; Zheng et al, 1993; Coughlin et al, 2000; Stolzenberg-Solomon et al, 2002, 2007; Inoue-Choi et al, 2011) and risk of pancreatic cancer. The RRs of pancreatic cancer associated with an increase of 50 g per day of red meat consumption are shown in Figure 1. We found no evidence of a non-linear association (P for nonlinearity = 0.13). The overall RR indicated no statistically significant association between red meat consumption and pancreatic cancer (RR = 1.13; 95% CI = 0.93 – 1.39). There was statistically significant heterogeneity among studies (P < 0.001; I² = 69.8%). In a sensitivity analysis in which we removed one study at a time and analysed the rest, the RRs ranged from 1.08 (95% CI = 0.89 – 1.31) after excluding the study by Nöthlings et al (2005) to 1.17 (95% CI = 0.95 – 1.45) after excluding the study by Heinen et al (2009).

In stratified analysis, a statistically significant positive association between red meat consumption and risk of pancreatic cancer was observed in men (RR = 1.29; 95% CI = 1.08 – 1.53; P heterogeneity = 0.28; five studies), but no association in women (RR = 0.93; 95% CI = 0.74 – 1.16; P heterogeneity = 0.21; six studies). No association was observed in studies conducted in the United States (RR = 1.13; 95% CI = 0.90 – 1.42; P heterogeneity < 0.001) or in Europe (RR = 0.87; 95% CI = 0.43 – 1.76; P heterogeneity = 0.01). We found no evidence of publication bias (P = 0.98).

Processed meat

Seven studies provided results for processed meat consumption (Stolzenberg-Solomon et al, 2002, 2007; Michaud et al, 2003; Nöthlings et al, 2005; Lin et al, 2006; Larsson et al, 2006a; Heinen et al, 2009). There was no evidence of a non-linear association between processed meat consumption and pancreatic cancer (P for nonlinearity = 0.75). When results from all studies were combined, an increase of 50 g per day of processed meat consumption was associated with a statistically significant 19% increased risk of pancreatic cancer (RR = 1.19; 95% CI = 1.04 – 1.36), without heterogeneity among studies (P = 0.46; I² = 0%) (Figure 2). In a sensitivity analysis excluding one study at a time and analysing the rest, the RRs ranged from 1.11 (95% CI = 0.95 – 1.30) to 1.24 (95% CI = 1.05 – 1.46) after excluding the study by Nöthlings et al (2005) and Stolzenberg-Solomon et al (2002), respectively.

In analysis stratified by sex, the overall RRs were 1.11 (95% CI = 0.92 – 1.34; P heterogeneity = 0.68; three studies) in men and 1.12 (95% CI = 0.75 – 1.67; P heterogeneity = 0.29; four studies) in women. There was no statistically significant association between processed meat consumption and pancreatic cancer in studies conducted in the United States (RR = 1.25; 95% CI = 0.96 – 1.62; P heterogeneity = 0.17; three studies) or Europe (RR = 1.06; 95% CI = 0.86 – 1.30; P heterogeneity = 0.85; three studies), possibly because of limited statistical power. No publication bias was detected (P = 0.53).

DISCUSSION

This meta-analysis showed a statistically significant positive association between processed meat consumption and risk of pancreatic cancer. An increase in processed meat consumption of 50 g per day, about one serving, was associated with a 19% increased risk of pancreatic cancer. The positive association between processed meat consumption and pancreatic cancer risk was attenuated and not statistically significant in a sensitivity analysis excluding one of the studies (Nöthlings et al, 2005). There was no overall association between red meat consumption and risk of pancreatic cancer. However, red meat consumption was
Table 1   Characteristics of prospective studies of red and processed meat consumption and pancreatic cancer risk

| Study, country          | Sample size, sex and age | No. of cases | Years of follow-up | Type of meat | RR (95% CI), highest vs lowest category | Adjustments                                      |
|-------------------------|--------------------------|--------------|-------------------|--------------|----------------------------------------|------------------------------------------------|
| Zheng et al (1993), United States | 17,633 men, ≥ 35 years | 57 | 20 | Red meata | 2.4 (1.0–6.1) | Age, smoking index, intakes of energy and alcohol |
| Coughlin et al (2000), United States | 110,308 men and women, ≥ 30 years | 3751 (1967 men, 1784 women) | 14 | Red meata | 1.1 (0.9–1.2) men | Age, race, smoking history, education, family history of pancreatic cancer, history of gallstones, history of diabetes, BMI, intakes of alcohol, citrus fruits and vegetables |
| Isaksson et al (2002), Sweden | 21,884 men and women, NA | 176 | 16 | Pork | 0.25 (0.08–0.81) | Age, sex, smoking, BMI |
| Stolzenberg-Solomon et al (2002), Finland | 26,948 men, 50–69 years | 163 | 13 | Red meata | 0.95 (0.58–1.56) | Age, years of smoking and energy intake |
| Michaud et al (2003), United States | 88,802 women, 30–55 years | 178 | 18 | Beef, pork or lamb | 1.04 (0.66–1.65) | Age, pack years of smoking, BMI, height, history of diabetes, energy intake |
| Nöthlings et al (2005), United States | 190,545 men and women, 45–75 years | 482 | 7 | Beef, pork, or lamb | 1.45 (1.19–1.76) | Age, sex, ethnicity, smoking status, history of diabetes, family history of pancreatic cancer and energy intake |
| Larsson et al (2008a), Sweden | 61,433 women, 40–76 years | 172 | 15.3 | Beef, pork, or veal | 1.73 (0.99–2.98) | Age, education, smoking status and pack years of smoking, BMI, and intakes of total energy, alcohol and folate |
| Lin et al (2006), Japan | 105,438 men and women, 40–79 years | 222 (106 men, 116 women) | 9.9 | Beef and porkb | 1.92 (0.95–3.86) men | Age, area and pack years of smoking |
| Stolzenberg-Solomon et al (2007), United States | 537,302 men and women, 50–71 years | 836 (555 men, 281 women) | 5 | Red meata | 1.42 (1.05–1.91) men | Age, education, race, smoking, BMI, history of diabetes and intakes of energy and saturated fat |
| Heinen et al (2009), The Netherlands | 120,852 men and women, 55–69 years | 350 | 13.3 | Fresh red meat | 0.75 (0.52–1.09) | Age, sex, smoking status and number of cigarettes smoked per day and number of years, BMI, history of diabetes, history of hypertension, intakes of energy, alcohol, vegetables and fruits |
| Inoue-Choi et al (2011), United States | 34,642 women, 55–69 years | 256 | 16.3 | Red meatb | 0.97 (0.65–1.44) | Age, race, education, smoking, physical activity and alcohol intake |

Abbreviations: BMI = body mass index; CI = confidence interval; NA = not available; RR = relative risk (rate ratio or hazard ratio). aIncluding processed meat. bResults for beef and pork were combined using a random effects model.

Figure 1   Relative risks of pancreatic cancer for a 120 g per-day increase of red meat consumption. Squares indicate study-specific relative risks (size of the square reflects the study-specific statistical weight, i.e., the inverse of the variance); horizontal lines indicate 95% CIs; diamond indicates the summary relative risk estimate with its 95% CI. Test for heterogeneity: Q = 43.05, P < 0.001, I² = 69.8%. All statistical tests were two-sided.
Epidemiology

Meats are usually preserved with nitrite and may also contain nitroso compounds. Nitroso compounds reach the pancreas via the bloodstream and are potent carcinogens that have been shown to induce pancreatic cancer in animal models (Risch, 2003). A population-based case-control study observed that intake of dietary nitrite from animal sources was statistically significantly positively associated with risk of pancreatic cancer in both men and women (highest vs lowest quartile odds ratio = 2.3; 95% CI = 1.1–5.1, for men and odds ratio = 3.2; 95% CI = 1.6–6.4, for women; Coss et al, 2004). A prospective study found that men in the highest quintile of summed nitrate/nitrite intake from processed meat had a nonsignificantly elevated risk of pancreatic cancer (hazard ratio = 1.18, 95% CI = 0.95–1.47; Aschebrook-Kilfoy et al, 2011).

Besides processed meat consumption, humans are exposed to N-nitroso compounds via cigarette smoking, which is an established risk factor for pancreatic cancer (Risch, 2003). Given that the main route of human exposure to N-nitroso compounds is cigarette smoke, the relation between processed meat consumption and pancreatic cancer risk may be modified by smoking status. Lin et al (2006) examined the association between ham and sausage consumption and risk of pancreatic cancer by smoking status, but observed no statistically significant association in neither smokers (highest vs lowest category RR = 1.44; 95% CI = 0.45–4.63) nor in nonsmokers (corresponding RR = 1.16; 95% CI = 0.43–3.19). However, the number of cases in the highest categories was very limited (≤4 cases).

In conclusion, results from this meta-analysis indicated a statistically significant positive association between processed meat consumption and risk of pancreatic cancer. Red meat consumption was not associated with risk of pancreatic cancer overall, but was positively associated with risk in men. Large prospective studies with better adjustment for potential confounders are warranted to establish potential associations of red and processed meat consumption with pancreatic cancer risk. Whether the association between processed meat consumption and pancreatic cancer is modified by smoking needs further study.

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**Figure 2** Relative risks of pancreatic cancer for a 50 g per day increase of processed meat consumption. Squares indicate study-specific relative risks (size of the square reflects the study-specific statistical weight, i.e., the inverse of the variance); horizontal lines indicate 95% CIs; diamond indicates the summary relative risk estimate with its 95% CI. Test for heterogeneity: *Q* = 7.77, *P* = 0.46, *I*² = 0%. All statistical tests were two-sided.

| First author          | Year | Sex | Relative risk (95% CI) |
|-----------------------|------|-----|------------------------|
| Stolzenberg-solomon   | 2002 | M   | 1.09 (0.86, 1.38)      |
| Michaud               | 2003 | W   | 1.55 (0.96, 2.50)      |
| Nöthlings             | 2005 | MW  | 1.44 (1.11, 1.86)      |
| Larsson               | 2006 | W   | 0.93 (0.43, 2.01)      |
| Lin                   | 2006 | M   | 1.62 (0.69, 3.80)      |
| Lin                   | 2006 | W   | 1.19 (0.49, 2.93)      |
| Stolzenberg-solomon   | 2007 | M   | 1.09 (0.78, 1.52)      |
| Stolzenberg-solomon   | 2007 | W   | 0.62 (0.26, 1.46)      |
| Heinig                | 2009 | MW  | 0.96 (0.56, 1.63)      |
| Overall               |      |     | 1.19 (1.04, 1.36)      |

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First author: SC Larsson and A Wolk

Year: 2002–2009

Sex: M/W

Relative risk: 95% CI
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