POSITION STATEMENT
Meat and cancer prevention

Key messages

- The term 'meat' encompasses a variety of foods, including unprocessed red meat (beef, veal, pork and lamb), processed meat, poultry and fish. Processed meat differs from unprocessed red meat in that it may be cured with the addition of preservatives and/or other additives.
- The relationship between meat consumption and the risk of cancer, especially colorectal cancer, has been controversial. The World Cancer Research Fund has found that the consumption of red meat and processed meat is *convincingly* associated with a modest increased risk of colorectal cancer. The risk is most associated with processed meats.
- There is *limited suggestive* evidence that red meat may be associated with an increased risk of oesophageal, lung, pancreatic and endometrial cancer, and processed meat with oesophageal, lung, stomach and prostate cancer.
- There does not appear to be a strong association between red meat or processed meat and the risk of other cancers.
- There is insufficient evidence to draw any conclusions on poultry intake and cancer risk.
- For fish consumption, there is limited but suggestive evidence that it may be linked to a reduced risk of breast, colorectal and prostate cancer.
- Despite the concerns about meat and cancer, Cancer Council recognises that lean red meat is an important contributor to dietary iron, zinc, vitamin B12 and protein in the Australian diet.
- Cancer Council recommends people consume moderate amounts of unprocessed lean red meat. A moderate amount of meat is 65-100g of cooked red meat, 3-4 times a week.
- Cancer Council also advises people to limit or avoid processed meats such as sausages, frankfurts, salami, bacon and ham, which are high in fat and salt. People should also try to limit their consumption of burnt or charred meat. It is best to choose lean cuts of meat and chicken, eat more fish and plenty of plant based foods such as fruit, vegetables and wholegrain cereals.

Background

The term 'meat' encompasses a variety of foods, including unprocessed red meat, processed meat, poultry (with and without skin) and fish. Unprocessed red meat generally includes beef, veal, pork, mutton and lamb and processed meat refers to sausages, smoked, cured and salted meats (e.g. frankfurts, salami, bacon and ham), and canned meats. Processed meat is sometimes referred to as preserved meat.

Australian food regulations define processed meat as a meat product containing not less than 30% meat, where meat either on its own or in combination with other ingredients or additives, has undergone a method of processing other than boning, slicing, dicing, mincing, or freezing. This includes manufactured meat and cured and/or dried meat.[1] Processed meat differs from unprocessed red meat (such as pork, lamb or beef steaks, chops or roasts), in that it may be cured with the addition of preservatives (salt, nitrite

or smoke) and/or other additives (phosphate, glutamate or ascorbic acid). Therefore it is possible that the role of unprocessed and processed red meat in carcinogenesis may differ.

Rationale

This position statement summarises epidemiological evidence from major cancer prevention reports, meta-analyses and systematic reviews that examined the relationship between meat consumption and cancer risk. More recent studies have separated the effect of unprocessed red meat from processed meat.

In addition to the meta-analyses and systematic reviews identified, the published results of the European Prospective Investigation into Cancer and Nutrition (EPIC) study were identified. EPIC is one of the largest cohort studies of men and women developed to specially examine the relationship between diet and cancer.

Views on meat in cancer prevention reports

The relationship between meat consumption and the risk of cancer, especially colorectal cancer, has been controversial. Several major public health bodies have reviewed the evidence on meat intake and the risk of cancer (table 1).

Table 1. Conclusions from the major reviews of the epidemiological literature regarding red and processed meat intake and increased cancer risk.

Organisation Review	Meat	Highest Evidence Convincing	Moderate Evidence Probable	Lower Evidence Possible / Limited	Insufficient Evidence	Inconsistent Evidence
WCRF/AICR (2007)[2]	Red*	Colorectal		Oesophageal Lung Pancreatic Endometrial		
	Processed^	Colorectal		Oesophageal Lung Stomach Prostate		
WHO/FAO (2003)[3]	Preserved^		Colorectal			
COMA (1998)[4]	Total (meat & meat products)			Breast Lung Prostate Pancreatic	Bladder Cervical Ovarian Testicular Oral Pharyngeal Laryngeal	Oesophageal
	Red*		Colorectal	Pancreatic		
	Processed#		Colorectal			
	Preserved^				Stomach	

*Refers to beef, lamb and pork, as well as goat for WCRF; #Refers to sausages, hamburgers, canned meat, smoked, cured and salted meat; ^Refers to cured and salted meat, as well as smoked meat or meat with chemical preservatives added for WCRF.

In 2007, an extensive review on diet and cancer conducted by the World Cancer Research Fund and the American Institute of Cancer Research (WCRF/AICR) found that red meat and processed meat *convincingly* increased the risk of colorectal cancer.[2] Red meat was associated with a *limited suggestive* increased risk of oesophageal, lung, pancreatic and endometrial cancer, and processed meat with a *limited suggestive* increased risk of oesophageal, lung, stomach and prostate cancer.[2]

WCRF also found that grilled or barbecued animal foods were associated with a *limited suggestive* increased risk of stomach cancer, and that foods containing iron had a *limited suggestive* increased association with colorectal cancer risk.[2]

The evidence on poultry was too limited in amount, consistency or quality to draw any conclusions.[2] WCRF recommended that people limit their intake of red meat and avoid processed meat.[2] Individuals who eat red meat should consume less than 500g cooked red meat per week.[2]

In their 2009 Policy and Action for Cancer Prevention report, WCRF estimated that population attributable fraction for red meat and bowel cancer was 5%, and 10% for processed meat and bowel cancer. (ref to be inserted here - World Cancer Research Fund and American Institute for Cancer Research. Policy and action for cancer prevention. Food, nutrition, and physical activity: a global perspective. Washington DC, AICR. 2009)

An expert report by the World Health Organization (WHO) and Food and Agriculture Organisation in 2003 advised those who are not vegetarian to consume moderate amounts of preserved meat (e.g. sausages, salami, bacon and ham).[3] WHO found that the evidence for an association between fish and decreased cancer risk was *possible/insufficient*.[3]

In 1998 the United Kingdom Department of Health's Committee on the Medical Aspects of Food & Nutrition Policy (COMA) recommended that, "*For adults, individuals' consumption of red and processed meat should not rise (around 90g/day); higher consumers (greater than 140g/day) should consider a reduction; and as a consequence of this the population average will fall*".[4] COMA found that there was moderately consistent evidence that poultry and fish intake were not associated with colorectal cancer, however the evidence for breast and prostate cancer was insufficient to draw conclusions.[4]

Epidemiological evidence

Colorectal cancer

A large amount of interest in the relationship between meat intake and colorectal cancer has led to a number of reviews being published. The results of these reviews have been summarised for high versus low intake (table 2) and as dose-response per serve (table 3).

Table 2. Summary relative risk (RR) and corresponding 95% confidence interval (CI) associated with highest versus lowest meat intake and colorectal cancer from meta-analyses, where n = number of studies included in analysis.

Type of meat	Author & date	All studies		Cohort Studies		Case-control studies		Fresh meat only		Fresh & processed meat	
		n	RR (95% CI)	n	RR (95% CI)	n	RR (95% CI)	n	RR (95% CI)	n	RR (95% CI)
Total	Norat et al. (2002)[5]	24	1.14 (0.99-1.31)	6	1.03 (0.81-1.32)	18	1.18 (0.99-1.40)	6	1.01 (0.64-1.60)	18	1.16 (1.01-1.34)
Red	Norat et al. (2002)[5]	23	1.35 (1.21-1.51)	9	1.27 (1.11-1.45)	14	1.36 (1.17-1.59)	13	1.28 (1.11-1.47)	11	1.49 (1.26-1.77)
	Larsson & Wolk (2006)[6]	-	-	15	1.28 (1.15-1.42)	-	-	9	1.22 (1.08-1.37)	8	1.24 (1.09-1.42)
Processed	Norat et al. (2002)[5]	23	1.31 (1.13-1.51)	7	1.39 (1.09-1.76)	16	1.29 (1.09-1.52)	-	-	-	-
	Larsson & Wolk (2006)[6]	-	-	14	1.20 (1.11-1.31)	-	-	-	-	-	-

Table 3. Summary relative risk (RR) or odds ratio (OR) and corresponding 95% confidence interval (CI) associated with a dose-response analysis of meat intake and colorectal cancer from meta-analyses, where n = number of studies included in analysis.

Type of meat	Author & date	Dose or amount of meat	All studies		Cohort Studies		Case-control studies	
			n	RR (95% CI)	n	RR (95% CI)	n	RR (95% CI)
Total	Sandhu et al. (2001)[7]	Increase of one portion (100g/day)	-	-	17	1.14 (1.04-1.25)*	-	-
	Norat et al. (2002)[5]	Increase in consumption of 120g/day	18	1.12 (0.98-1.30)	5	0.99 (0.71-1.39)	13	1.10 (0.94-1.29)
Red	Sandhu et al. (2001)[7]	Increase of one portion (100g/day)	-	-	8	1.17 (1.05-1.31)*	-	-
	Norat et al. (2002)[5]	Increase in consumption of 120g/day	17	1.24 (1.08-1.41)	9	1.22 (1.05-1.41)	8	1.26 (1.02-1.55)
	Larsson & Wolk (2006)[6]	Increase in consumption of 120g/day	-	-	14	1.28 (1.18-1.39)	-	-
Processed	Sandhu et al. (2001)[7]	Increase of one portion (25g/day)	-	-	10	1.49 (1.22-1.81)*	-	-
	Norat et al. (2002)[5]	Increase in consumption of 30g/day	16	1.36 (1.15-1.61)	7	1.54 (1.10-2.17)	9	1.37 (1.13-1.66)
	Larsson & Wolk (2006)[6]	Increase in consumption of 30g/day	-	-	11	1.09 (1.05-1.13)	-	-

* Results reported for the random effects model.

Overall, it appears as though the consumption of meat, particularly processed meat, may be associated with a modest increased risk of colorectal cancer. Some non-systematic reviews however, have questioned the link between meat consumption and colorectal cancer risk,[8] and it has been suggested that perhaps meat intake is only a risk factor for colorectal cancer in those who do not eat enough foods that appear to be protective e.g. fruit, vegetables and wholegrain cereals.[9]

Total meat

In 2001, Sandhu et al. reported a dose-response meta-analysis of prospective cohort studies in which they estimated the relative risk as a function of the amount consumed daily.[7] This study found that an increase of one portion of total meat (100g/day) modestly increased the risk of colorectal cancer (Table 3).[7] When this RR is converted to a value for 120g/day like the other study comparing total meat,[5] the new RR increases slightly to 1.17.

The following year, Norat et al. reported another meta-analysis that included case-control as well as cohort studies.[5] This study calculated relative risks for the highest versus the lowest categories of consumption reported in the individual studies and also performed a dose-response meta-analysis. The results showed that the highest category of total meat consumption appeared to be associated with a slight increased risk of colorectal cancer, particularly in case-control studies (table 2).[5] However the relative risk was lower when total meat intake was defined as total fresh meat than when it was defined as total fresh and processed meat (table 2).[5] A small non-significant increase in colorectal cancer risk was associated with an increment in consumption of 120g total meat per day (table 3).[5]

Interestingly, an analysis of five prospective studies showed that there is no significant difference in mortality from colorectal cancer between vegetarians and non-vegetarians.[10]

Red meat

In 2001, Sandhu et al. found that an increase of one portion of red meat (100g/day) modestly increased the risk of colorectal cancer (table 3).[7] When this RR is converted to a value for 120g/day like the other studies comparing red meat,[5, 6] the new RR decreases slightly to 1.09.

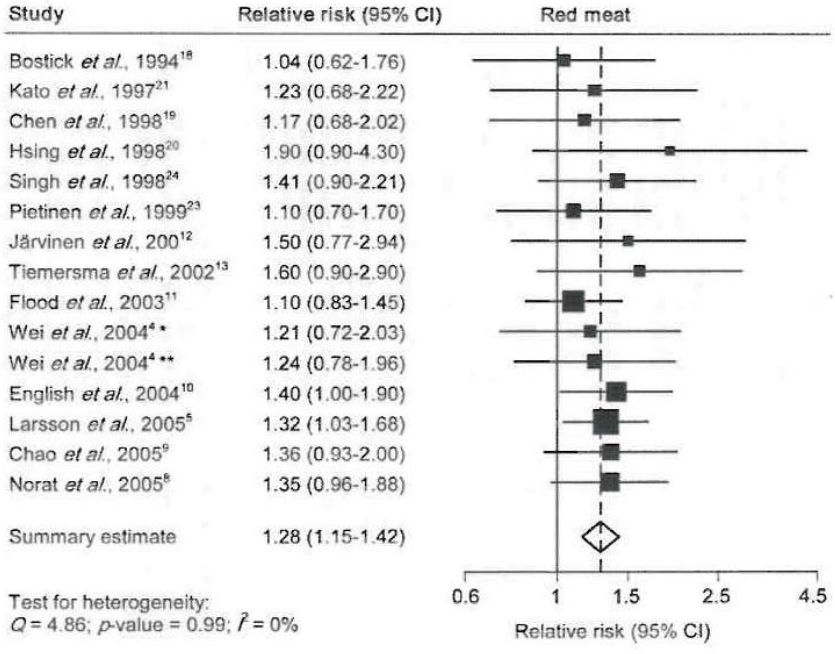
Norat et al. found the relative risk of colorectal cancer for those in the highest categories of intake was larger for red meat intake than for total meat intake in both cohort and case-control studies (table 2).[5] Results were similar for colon (RR= 1.32, 95% CI= 1.18-1.48; n= 19) and rectal cancer (RR= 1.36, 95% CI= 1.17-1.57; n= 7). As with total meat, the relative risk was lower when red meat intake was defined as fresh red meat than when it was defined as fresh and processed red meat (table 2).[5] The dose-response analysis also found an increase in colorectal cancer risk associated with increasing intake (table 3).[5]

In 2006, Larsson and Wolk reported a second meta-analysis of prospective cohort studies,[6] which included 10 studies published after the review by Sandhu et al. This study conducted analyses both for the highest versus lowest categories of intake and for a dose-response. The relative risks of colorectal cancer for the highest versus lowest category of red meat intake in individual studies can be seen in figure 1.

A positive association between high red meat intake and the risk of colorectal cancer was seen (table 2 and figure 1).[6] In the analyses of the highest versus lowest categories of intake, the association with red meat consumption was stronger for rectal cancer (RR= 1.56, 95% CI= 1.25-1.95; n= 7) than for colon cancer (RR= 1.21, 95% CI= 1.05-1.40; n= 9).[6] Relative risks for fresh plus processed red meat were similar to results for fresh red meat only (table 2).[6] In the dose-response analysis, an increased risk of colorectal cancer was also seen for red meat (table 3).[6]

In 2005, results from the EPIC study were published, showing that red meat (fresh, minced and frozen beef, veal, lamb and pork) increased the risk of colorectal cancer, although the association was not significant (Hazard ratio (HR)= 1.17, 95% CI= 0.92-1.49).[11] Red meat was also associated with a non-significant increased risk of colon cancer (HR= 1.20, 95% CI= 0.88-1.61), however red meat did not increase the risk of rectal cancer (HR= 1.13, 95% CI= 0.74-1.71).[11]

Figure 1. Relative risks of colorectal cancer for the highest versus lowest category of red meat intake in prospective studies taken from Larsson and Wolk.[6] Squares represent study-specific relative risks and the sizes of the squares reflect the statistical weight that each study contributed to the summary estimate; horizontal lines represent 95% confidence intervals (CI); diamond represents the summary estimate and its 95% CI. *Nurses Health Study; **Health Professionals Follow-Up Study.



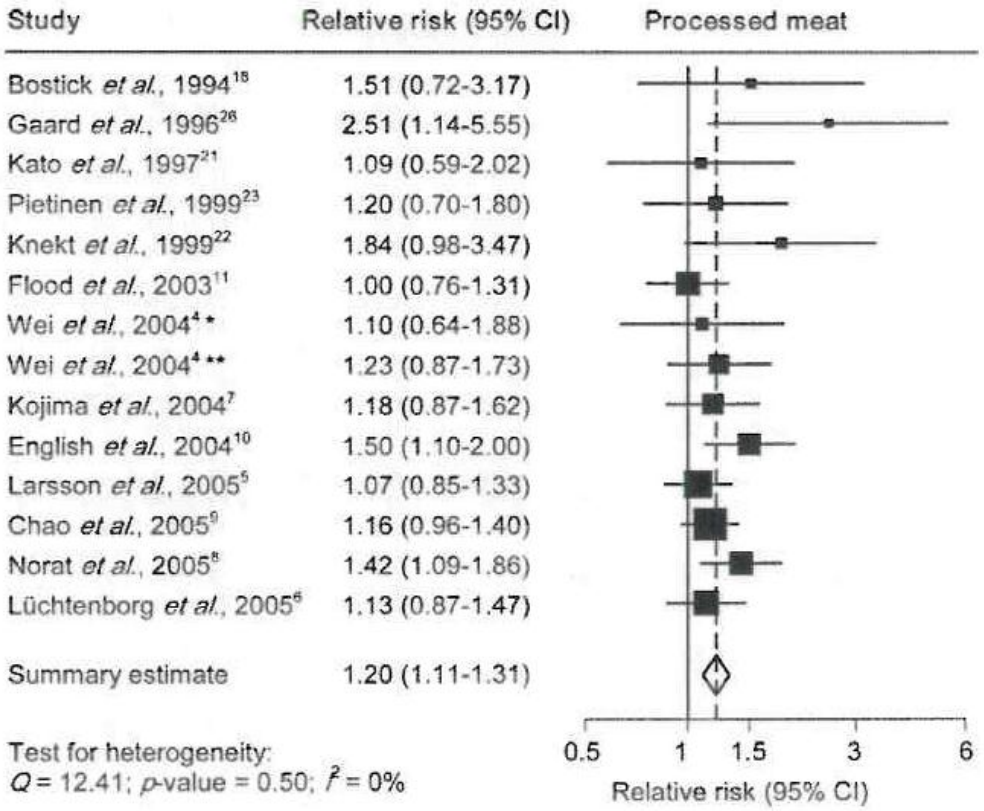
Processed meat

In 2001, Sandhu et al. found that an increase of one portion of processed meat (25g/day) modestly increased the risk of colorectal cancer (table 3).[7] When this RR is converted to a value for 30g/day like the other studies comparing processed meat,[5, 6] the new RR decreases to 1.23.

Norat et al. found the highest category of processed meat consumption was associated with a modest increase in the risk of colorectal cancer (table 2).[5] The relative risks were similar for colon cancer (RR= 1.22, 95% CI= 1.06-1.39; n= 15) and rectal cancer (RR= 1.21, 95% CI= 0.98-1.50; n= 5), although the estimate for rectal cancer was not significant.[5] The relative risk of colorectal cancer was higher for males (RR= 1.57, 95% CI= 1.27-1.93; n= 7) compared to females (RR= 1.17, 95% CI= 0.95-1.44; n= 7), but no p-value for the difference in these relative risks was presented.[5] From the dose-response analysis, there was also an increase in colorectal cancer risk associated with an increment of 30g of processed meat per day (table 3).[5] Norat et al. found that the relative risks for processed meat and colorectal cancer in the dose-response analysis were stronger than those seen for both total and red meat (table 3).[5]

In 2006, Larsson and Wolk found that a higher consumption of processed meat was also associated with an increased risk of colorectal cancer (table 2).[6] The relative risks of colorectal cancer for the highest versus lowest category of processed meat intake in individual studies can be seen in figure 2.

Figure 2. Relative risks of colorectal cancer for the highest versus lowest category of processed meat intake in prospective studies taken from Larsson and Wolk.[6] Squares represent study-specific relative risks and the sizes of the squares reflect the statistical weight that each study contributed to the summary estimate; horizontal lines represent 95% confidence intervals (CI); diamond represents the summary estimate and its 95% CI. *Nurses Health Study; **Health Professionals Follow-Up Study.



The relative risk of colon (RR= 1.21, 95% CI= 1.09-1.34; n= 10) and rectal (RR= 1.20, 95% CI= 0.98-1.46; n= 8) cancer associated with processed meat intake were similar, although the relative risk was only statistically significant for colon cancer.[6] When processed meat consumption was assessed in a dose-response analysis, an increase of 30g per day was associated with a smaller increase in colorectal cancer risk than seen in the other two meta-analyses (table 3).[6]

In 2005, the EPIC study showed that processed meat (mostly beef and pork preserved by methods other than freezing such as salting, smoking, marinating, air drying or heating) increased the risk of colorectal cancer (HR= 1.42, 95% CI= 1.09-1.86).[11] Processed meat was associated with an increased risk of colon cancer (HR= 1.30, 95% CI= 0.92-1.84) and rectal cancer (HR= 1.62, 95% CI= 1.04-2.50), although the risk for colon cancer was non-significant.[11] When analysed together, red plus processed meat non-significantly increased the risk of colorectal cancer (HR= 1.35, 95% CI= 0.96-1.88), colon cancer (HR= 1.17, 95% CI= 0.78-1.77) and rectal cancer (HR= 1.75, 95% CI= 0.98-3.10).[11]

Chicken

In 2005, the EPIC study showed that poultry consumption was associated with a non-statistically significant reduced risk of colorectal cancer (HR= 0.92, 95% CI= 0.76-1.12), colon cancer (HR= 0.89, 95% CI= 0.70-1.13) and rectal cancer (HR= 0.99, 95% CI= 0.71-1.37).[11]

Fish

In the EPIC study, fish consumption was associated with a statistically significant reduced risk of colorectal cancer (HR= 0.69, 95% CI= 0.54-0.88) and rectal cancer (HR= 0.49, 95% CI= 0.32-0.76), and a non-significant reduced risk of colon cancer (HR= 0.82, 95% CI= 0.60-1.11).[11] The results for fish intake were surprising considering the exposure included both salted and smoked fish, which have been shown to independently increase the risk of some cancers.[12]

A systematic literature review in 2006 found that fish intake was associated with a decreased risk of rectal cancer in case-control studies (pooled odds ratio (OR)= 0.68, 95% CI= 0.48-0.96; n= 5).[13] A non-significant negative association was found between fish intake and colon cancer in cohort (pooled OR= 0.95, 95% CI= 0.66-1.39; n= 4) and case-control studies (pooled OR= 0.86, 95% CI= 0.63-1.17; n=8), while a small negative association was seen between fish intake and colorectal cancer in cohort (pooled OR= 0.95, 95% CI= 0.71-1.28; n= 5) and case-control studies (pooled OR= 0.99, 95% CI= 0.49-2.03; n=2).[13]

Oesophageal cancer

In 2006, a systematic review by Jakszyn and González found that one out of two cohort studies showed a positive relationship between red meat intake and oesophageal cancer risk.[14] Eighteen case-control studies have investigated meat intake and oesophageal cancer risk, with 11 studies finding a positive association.[14] For processed meat, no cohort studies have reported results for oesophageal cancer. Eight out of nine case-control studies found a positive association between processed meat intake and oesophageal cancer risk.[14]

Stomach cancer

A systematic review in 2006 by Jakszyn and González found that two (out of three) cohort studies showed a positive relationship between red meat intake and stomach cancer risk.[14] In addition, 11 (out of 16) case-control studies found a positive association between meat intake and stomach cancer risk.[14] For processed meat, four out of six cohort studies have found no association with stomach cancer risk.[14] Ten out of 14 case-control studies found a positive association between processed meat intake and stomach cancer risk.[14]

A meta-analysis by Larsson et al. published the same year found that when comparing high versus low intake categories for processed meat, cohort studies showed a non-significant increased relative risk (summary relative risk (RR)= 1.24, 95% confidence interval (CI)= 0.98-1.56; number of studies (n)= 7) and case-control studies showed a greater relative risk (summary RR= 1.63, 95% CI= 1.31-2.01; n= 12) of stomach cancer.[15] There was statistically significant heterogeneity among the cohort (p= 0.04) and case-control (p= 0.06) studies.[15] For individual processed meats (bacon, sausage and ham) the results were consistent, with studies showing a positive association between high versus low intake of all individual processed meats and the risk of stomach cancer.[15] A dose-response analysis found that the risk of stomach cancer increased when processed meat consumption increased by 30g per day for both cohort (summary RR= 1.15, 95% CI= 1.04-1.27; n= 6) and case-control (summary RR= 1.38, 95% CI= 1.19-1.60; n= 9) studies.[15]

Prostate cancer

A review of prospective studies found that meat consumption was not consistently associated with prostate cancer risk.[16] Three studies showed an increased risk, one a decreased risk and three a small decreased risk with meat consumption and prostate cancer.[16] Results for fish and poultry were also mixed, however most reported associations for processed meat were positive.[16]

In 2006, a systematic literature review found that the evidence was suggestive that fish intake may be associated with a decreased risk of prostate cancer.[13] For fish intake and prostate cancer risk, the pooled OR for cohort studies was 0.95 (95% CI= 0.84-1.09; n= 2), and for case-control studies the pooled OR was 0.65 (95% CI 0.47-0.90; n= 4).[13]

Breast cancer

A pooled analysis of eight prospective studies in 2002 found that total (pooled RR for highest quartile= 1.08, 95% CI= 0.98-1.19) and white (pooled RR for highest quartile= 1.02, 95% CI= 0.91-1.13) meat intake appeared to have small non-significant positive associations with breast cancer, where total meat included eggs.[17] However, red meat appeared to have a small non-significant negative association with breast cancer (pooled RR for highest quartile= 0.94, 95% CI= 0.87-1.02).[17] There was no significant heterogeneity among studies for total (p= 0.29), white (p= 0.11) and red (p= 0.58) meats.[17]

The following year, Boyd et al. published a meta-analysis (including nine cohort and 22 case-control studies) of the relationship between total meat (red meat, poultry and pork) intake and breast cancer risk.[18] The analysis showed that high versus low meat intake was linked to an increased risk of breast cancer in cohort studies (summary RR= 1.32, 95% CI= 1.12-1.56) and case-control studies (summary RR= 1.13, 95% CI= 1.01-1.25).[18]

In 2006, a systematic literature review found that the evidence was suggestive that fish intake may be associated with a decreased risk of breast cancer.[13] For fish intake and breast cancer risk, the pooled OR for cohort studies was 1.01 (95% CI= 0.83-1.24; n= 4). For case-control studies however, the pooled OR was 0.82 (95% CI= 0.71-0.96; n= 7).[13] In addition, many of the studies showed a significant trend (p<0.05) of decreasing risk with higher levels of fish intake.[13]

Endometrial cancer

A 2007 meta-analysis on meat intake and endometrial cancer risk suggested that meat, particularly red, may be associated with an increased risk of endometrial cancer, however the definition of meat varied substantially among both cohort and case-control studies and several case-control studies did not adjust for energy intake.[19]

For highest versus lowest consumption categories, case-control studies showed there was a modest association between meat intake (type unspecified) and endometrial cancer (OR= 1.39; 95% CI= 1.13-1.71; n= 9).[19] The same finding was noted from a dose-response analysis, in which the OR for an increase in meat intake of 100g/day was 1.26 (95% CI= 1.03-1.54; n= 8).[19] For red meat intake, case-control studies showed that a high intake (OR= 1.48; 95% CI= 1.22-1.80; n= 7) and an increase in intake of 100g/day (OR= 1.51; 95% CI= 1.19-1.93; n= 7) was associated with an increased risk of endometrial cancer.[19] Data for processed meat was not pooled, however three case-control studies found inconsistent results.[19]

In addition, the meta-analysis showed that a high versus low intake of poultry (OR= 0.96; 95% CI= 0.72-1.29; n= 7) and an increase in poultry intake of 100g/day (OR= 1.03; 95% CI= 0.32-3.28; n= 5) was not strongly associated with endometrial cancer risk.[19] Likewise an increase in fish intake of 100g/day was not strongly associated with endometrial cancer risk (OR= 1.04; 95% CI= 0.55-1.98; n= 7).[19] However individual study results for both poultry and fish intake were heterogeneous (p<0.01).[19]

Potential mechanisms of action

The following hypotheses have been proposed to explain the association between meat consumption and cancer risk, particularly colorectal cancer. However, none of these hypotheses have been confirmed in human experiments:

- A high iron intake may cause oxidative damage and hence induce tumours by catalysing the production of hydroxyl radicals.[20]
- Nitrogenous residues from meat are available for N-nitrosation by colonic bacteria, thereby increasing the formation of ammonia and N-nitrosocompounds (NOC).[21] Ammonia is a promoter of carcinogenesis and NOC have been shown to induce the formation of DNA adducts in human colonocytes. Meat or fish processed by the addition of nitrites and by smoking or direct-fire drying may contribute even further to the production of NOC. Endogenous production of NOC is increased with the consumption of red meat, but not white meat or fish. The difference may be related to the higher haem content of red meat which can act as a nitrosating agent under certain conditions.[22]
- Heterocyclic amines (HCAs) formed when meat, fish or poultry are cooked are known to be absorbed from the human gastrointestinal tract and have been shown to be large-bowel carcinogens in animal models and to produce adducts in mammary tissue of rodents.[20, 23] Different amounts of HCAs are produced according to duration and temperature of the cooking method, with higher levels of HCAs found in meat cooked using high-temperature methods such as grilling, pan-frying and barbequing.[24] Therefore, cooking method rather than the type of meat may be a more important determinant of cancer risk. In addition, differences in genotype may also play a role in the association between HCA exposure and colorectal cancer.[25] For HCAs to bind to DNA and hence initiate carcinogenesis, they must be activated by N-acetyltransferase (NAT) enzymes. Differences in an individual's capacity to metabolise HCA may explain inconsistency in the relationship between meat consumption and risk of colorectal cancer, where rapid NAT acetylators may have a higher risk of colon cancer than slow NAT acetylators.[25]
- Polycyclic aromatic hydrocarbons (PAHs) formed from the incomplete combustion of organic material, may induce the formation of DNA adducts and interfere with apoptosis. PAHs are in a wide variety of foods, however higher levels are found in foods that have been exposed to combustion products and foods that have been charred or burned when cooked at high-temperatures.[26]
- The total fat content of meat may contribute to an increased production of bile acids in the colonic lumen. Bile acids metabolised into the secondary bile acid, deoxycholic acid, may act as tumour promoters by increasing cell proliferation in the colonic mucosa.[20] However epidemiological studies have not consistently shown an association with dietary fat intake and cancer risk.[27, 28]
- The low ratio of polyunsaturated to saturated fatty acids in meat fat may contribute to hyperinsulinaemia. Exposure to elevated blood-insulin levels may promote the growth of colon tumours which has been demonstrated in a rat model.[29, 30]

- Fish, particularly those high in omega-3 polyunsaturated fatty acids, may be protective against colorectal cancer as these fatty acids have been shown to reduce cell proliferation and aberrant crypt formation, probably by modifying the inflammatory response.[20]

Factors to consider when evaluating the literature on meat and cancer risk

Cut and quality of meat

Studies do not always collect information on the type of meat cut typically used (or species of fish), and if the meat consumed was lean or fatty.

Cooking technique

Studies do not usually take into account the cooking method of meat and the doneness level e.g. raw versus well done. Differences in cooking method possibly may explain inconsistencies in the relationship between meat consumption and colorectal cancer. Given the effect of cooking method on HCAs and PAHs it may be important to include information on cooking method in dietary data.

Differences in meat definition

Different studies may define "total meat", "red meat", "white meat" and "processed meat" to include slightly different meat categories, which makes it difficult to compare results across studies.

Other lifestyle and dietary factors

It is difficult to separate the independent role of individual foods when studying cancer incidence and other health outcomes. The relative risk estimates for meat and cancer could also be affected by other eating and lifestyle patterns, such as:

- Fruit and vegetable intake
- Dietary fibre intake
- Dietary fat intake (particularly total and saturated fat intake)
- Body mass index
- Physical activity levels

For example, a higher intake of meat may be linked with an increased cancer risk because the diet of those eating large quantities of meat may contain inadequate fruit, vegetables and fibre. Many epidemiological studies on meat and cancer risk do not comprehensively control for all potential confounding factors.

Current consumption levels in Australian adults

- In 1995, the National Nutrition Survey showed that most adults (85% of men and 77% of women) ate some meat, poultry or game on the day of the survey.[31]
- National Nutrition Survey data show that approximately 20% of adults ate a fish or seafood product on the day of the survey in both 1983 and 1995.[31]
- On the day of the 1995 National Nutrition Survey, adults' mean intake of red meat was consistent with the recommendations of the Australian Guide to Healthy Eating. Also, around two-thirds of red meat cuts eaten were reported to be either trimmed of fat or lean when eaten.[32]
- Between 1983 and 1995, mean daily intake of red meat and pork declined for both men and women; whereas mean daily intake of poultry and seafood increased.[32]
- Meat consumption patterns differ between countries. Australia has high consumption of beef, veal and mutton, whereas Europe has high consumption of pork.[33]

Contribution of meat to the Australian diet

- Meat is an important source of iron, particularly haem iron in the diets of females. It contributes 16% and 11% of total iron intake in adult males and females respectively.[34] Iron is an essential mineral required for formation of haemoglobin.
- Meat is a major source of zinc, contributing a third and a quarter of total zinc intake in adult males and females respectively.[34] Zinc is present in many tissues and plays a role in enzyme reactions and maintaining the immune system.
- Meat is an important source of protein, contributing a quarter and a fifth of total protein intake in adult males and females respectively.[34] Protein is involved in the growth and repair of cells.
- Meat also contributes to the intake of vitamin B12, thiamin, riboflavin, niacin, phosphorus and magnesium.[34] These nutrients are involved in essential chemical reactions including cell growth and repair, metabolism, and nerve and muscle function.
- Meat contributes 14% and 11% of total fat in adult males and females respectively.[34] Meat contributes 15% and 11% of saturated fat in adult males and females respectively.[34] Fat is energy dense and therefore over consumption leads to weight gain and saturated fat raises cholesterol levels.

Recommendations

Cancer Council recognises that red meat is an important contributor to dietary iron, zinc, vitamin B12 and protein in the Australian diet.

Cancer Council recommends people:

- Consume moderate amounts of unprocessed (or fresh) lean red meat. A moderate amount of meat is 65-100g of cooked red meat, 3-4 times a week (as specified in the *Australian Guide to Healthy Eating*).[35] This would be approximately ½ cup lean mince, 2 small chops or 2 slices of roast meat. Other substitutes for a serve of meat include 65-100g cooked chicken, 80-120g cooked fish fillet, 2 small eggs, 1/3 cup cooked legumes (lentils, chickpeas, split peas, dried or canned beans) or 1/3 cup nuts. Those eating meat should also try to eat plenty of plant-based foods like fruit, vegetables and wholegrain cereals.
- Limit or avoid processed meats, which are high in fat and salt. Processed meats include sausages, frankfurts, salami, bacon and ham.
- Choose lean cuts of meat and chicken, and remove any skin.
- Eat fish (preferably oily) at least two times per week (*see position statement on omega-3 fatty acids, fish and cancer prevention).*
- Limit consumption of burnt or charred meat, and choose cooking methods such as casseroling, boiling or microwave heating over high-temperature grilling, pan-frying or barbequing, as while the evidence for an association between heterocyclic amines and cancer risk is inconsistent, there is reasonable doubt to permit cautionary advice.

Future research

More studies are needed on the association between meat and cancer risk. In the future, there is a need for more studies that:

- Investigate the differences found between cohort and case-control studies.
- Use a standard set of definitions for different meat categories.
- Collect and report separate data for unprocessed and processed meat.
- Collect and report data on the cooking method used.
- Adequately control for potential confounding factors such as fruit, vegetable, fibre and fat intake, body weight, and physical activity levels.
- Systematic reviews that assess and report on the quality of individual studies.

Further information

Cancer Council New South Wales
PO Box 572 Kings Cross NSW 1340
www.cancercouncil.com.au
ABN 51 116 463 846

Contact

Kathy Chapman, Nutrition Program Manager: kathyc@nswcc.org.au

Acknowledgments

This position statement has been reviewed by:

- Allison Hodge
- Dallas English
- Hayley Griffin
- Jenny Atkins
- Udani Abeypala

References

1. Food Standards Australia New Zealand (FSANZ). Australia New Zealand Food Standards Code. Canberra, FSANZ. 2007.
2. World Cancer Research Fund and American Institute for Cancer Research. Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington DC, AICR. 2007.
3. World Health Organization. Diet, nutrition and the prevention of chronic diseases. Geneva, WHO. 2003.
4. United Kingdom Department of Health. Nutritional aspects of the development of cancer. Report of the working group on diet and cancer of the Committee on Medical Aspects of the Food and Nutrition Policy. Committee on Medical Aspects of the Food and Nutrition Policy. Norwich, UK, The Stationery Office. 1998.
5. Norat T, Lukanova A, Ferrari P, Riboli E. Meat consumption and colorectal cancer risk: dose-response meta-analysis of epidemiological studies. *International Journal of Cancer*. 2002; 98(2): 241-256.
6. Larsson SC, Wolk A. Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies. *International Journal of Cancer*. 2006; 119(11): 2657-2664.
7. Sandhu MS, White IR, McPherson K. Systematic review of the prospective cohort studies on meat consumption and colorectal cancer risk: a meta-analytical approach. *Cancer Epidemiology, Biomarkers & Prevention*. 2001; 10(5): 439-446.
8. Truswell AS. Meat consumption and cancer of the large bowel. *European Journal of Clinical Nutrition*. 2002; 56(Suppl 1): S19-24.
9. Hill M. Meat, cancer and dietary advice to the public. *European Journal of Clinical Nutrition*. 2002; 56(Suppl 1): S36-41.
10. Key TJ, Fraser GE, Thorogood M, Appleby PN, Beral V, Reeves G *et al.* Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. *Public Health Nutrition*. 1998; 1(1): 33-41.
11. Norat T, Bingham S, Ferrari P, Slimani N, Jenab M, Mazuir M *et al.* Meat, fish, and colorectal cancer risk: the European Prospective Investigation into cancer and nutrition. *Journal of the National Cancer Institute*. 2005; 97(12): 906-916.
12. The World Cancer Research Fund and American Institute for Cancer Research. *Food, nutrition and the prevention of cancer: a global perspective*. New York: American Institute for Cancer. 1997.
13. Ralph H, Chapman K, Yu XQ, O'Connell D. The association between omega-3 fatty acid and fish intake and the risk of breast, colon, rectum and prostate cancer: A systematic review. Sydney, The Cancer Council NSW. 2006.
14. Jakszyn P, González CA. Nitrosamine and related food intake and gastric and oesophageal cancer risk: a systematic review of the epidemiological evidence. *World Journal of Gastroenterology*. 2006; 12(27): 4296-4303.
15. Larsson SC, Orsini N, Wolk A. Processed meat consumption and stomach cancer risk: a meta-analysis. *Journal of the National Cancer Institute*. 2006; 98(15): 1078-1087.

16. Dagnelie PC, Schuurman AG, Goldbohm RA, Van den Brandt PA. Diet, anthropometric measures and prostate cancer risk: a review of prospective cohort and intervention studies. *BJU International*. 2004; 93(8): 1139-1150.
17. Missmer SA, Smith-Warner SA, Spiegelman D, Yaun SS, Adami HO, Beeson WL *et al.* Meat and dairy food consumption and breast cancer: a pooled analysis of cohort studies. *International Journal of Epidemiology*. 2002; 31(1): 78-85.
18. Boyd NF, Stone J, Vogt KN, Connelly BS, Martin LJ, Minkin S. Dietary fat and breast cancer risk revisited: a meta-analysis of the published literature. *British Journal of Cancer*. 2003; 89(9): 1672-1685.
19. Bandera EV, Kushi LH, Moore DF, Gifkins DM, McCullough ML. Consumption of animal foods and endometrial cancer risk: a systematic literature review and meta-analysis. *Cancer Causes & Control*. 2007; 18(9): 967-988.
20. Bingham S. Epidemiology and mechanisms relating diet to risk of colorectal cancer. *Nutrition Research Reviews*. 1996; 9(1): 197-239.
21. Hughes R, Cross AJ, Pollock JR, Bingham S. Dose-dependent effect of dietary meat on endogenous colonic N-nitrosation. *Carcinogenesis*. 2001; 22(1): 199-202.
22. Bingham SA, Luben R, Day NE, Riboli E. Plant polysaccharides, meat and colorectal cancer. *IARC Scientific Publications*. 2002; 156: 349-352.
23. Turesky RJ. Formation and biochemistry of carcinogenic heterocyclic aromatic amines in cooked meats. *Toxicology Letters*. 2007; 168(3): 219-227.
24. Sinha R, Kulldorff M, Chow WH, Denobile J, Rothman N. Dietary intake of heterocyclic amines, meat-derived mutagenic activity, and risk of colorectal adenomas. *Cancer Epidemiology, Biomarkers & Prevention*. 2001; 10(5): 559-562.
25. Roberts-Thomson IC, Ryan P, Khoo KK, Hart WJ, McMichael AJ, Butler RN. Diet, acetylator phenotype, and risk of colorectal neoplasia. *Lancet*. 1996; 347(9012): 1372-1374.
26. Jagerstad M, Skog K. Genotoxicity of heat-processed foods. *Mutation Research*. 2005; 574(1-2): 156-172.
27. Kushi L, Giovannucci E. Dietary fat and cancer. *American Journal of Medicine*. 2002; 113(Suppl 9B): 63S-70S.
28. Willett WC. Dietary fat intake and cancer risk: a controversial and instructive story. *Seminars in Cancer Biology*. 1998; 8(4): 245-253.
29. Giovannucci E. Insulin and colon cancer. *Cancer Causes & Control*. 1995; 6(2): 164-179.
30. Tran TT, Medline A, Bruce WR. Insulin promotion of colon tumors in rats. *Cancer Epidemiology, Biomarkers & Prevention*. 1996; 5(12): 1013-1015.
31. McLennan W & Podger A. National nutrition survey: foods eaten. Canberra, Australian Bureau of Statistics. 1999.
32. Baghurst K, Record S, Leppard P. Red meat consumption in Australia: intakes, nutrient contribution and changes over time. *Nutrition and Dietetics*. 2000; 57(4): S3-S36.
33. FAOSTAT. Food Consumption Data. Food and Agriculture Organisation of the United Nations. 2005.
34. Australian Bureau of Statistics and Commonwealth Deptartment of Health and Family Services. National Nutrition Survey: nutrient intakes and physical measurements. Canberra, Australian Bureau of Statistics. 1998.
35. Department of Health and Family Services. The Australian guide to healthy eating. Canberra, AGPS. 1998.

Cancer Council Australia, GPO Box 4708, Sydney NSW 2001
Ph: (02) 8063 4100 Fax: (02) 8063 4101 Website: www.cancer.org.au