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ORIGINAL COMMUNICATION

Meat, cancer and dietary advice to the public

M Hill[1]*

[1]*Nutrition Research Centre, South Bank University London, UK*

Background: It has been claimed for many decades that meat is a risk factor for colorectal cancer, and that it has no compensating benefits in terms of cancer risk. The evidence for this has been critically reassessed.
Methods: The epidemiological evidence, particularly that produced in recent years, has been re-examined to determine whether it is sufficiently consistent to warrant giving firm advice to the general public.
Results: Far from being supportive, the epidemiological data does not justify this claim. A large mass of evidence is presented from case–control studies and prospective studies, in which the data from Europe are not consistent with those from the United States. This is because of the different contexts (in terms of meal composition) within which meat is consumed in different countries. In fact the epidemiological data are much more consistent with there being a protective role for fruit, vegetables and whole grain cereals and no role for meat in colorectal cancer, and a protective role in gastric cancer.
Conclusions: Meat is a good source of protein, readily available iron, calcium, magnesium, selenium, zinc and a range of B vitamins. Since the evidence for any role in colon carcinogenesis is so weak, and since such a high proportion of women of child-bearing age are iron deficient, the consumption of meat, as part of a balanced and varied diet, should be actively encouraged.
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Background

Cancer has not always been a leading cause of death. In 1900 the main causes of death in all European countries were infectious diseases such as tuberculosis, typhoid, cholera, smallpox etc. All of them have been conquered, but none of them was conquered through treatment. In all cases apart from smallpox, these are still major causes of morbidity and mortality in the third world; respiratory infections and diarrhoeal diseases are ranked third and fourth, respectively, as leading causes of death worldwide (Murray & Lopez 1997). We do not get them in the West because the causes have been identified and, through improved water supply, food handling and sewage disposal, removed from our environment. As a result of this success we now live long enough to have cancer as a major cause of death. Although we have to die of something, and cancer will always be one of the major causes of premature death, we can hope to decrease the mortality rate by identifying the causes and removing them from our environment.

***Correspondence: M Hill, Nutrition Research Centre, South Bank University, 103 Borough Rd, London SEI OAA, UK.**

Numerous studies have shown that the major causes of cancer are tobacco and poor diet (eg Doll & Peto 1981); the other causes (such as sunlight, exogenous hormones, viruses etc) are minor in comparison. Many Western countries have had considerable success in decreasing the proportion of men who smoke, and this has led to a massive decrease in smoking-related death (Peto *et al*, 1994). In the UK, smoking-related deaths decreased from 80 000 per year in 1975 to 40 000 per year in 1995 (Peto *et al*, 2000). The 40 000 lives saved each year by the anti-smoking campaign is more than all the colorectal and breast cancers combined! And those are the two most common cancer sites after lung cancer. The number of male lung cancer deaths in the UK has been decreasing steadily for more than 10 y. So cancer prevention works!

Since diet is as important a cause of cancer as is tobacco (Doll & Peto, 1981), we should be able to achieve a similar level of success by improving our diet. However, smoking and diet are very different problems. We can give up smoking with only benefits to our health and no risks, but we have to eat. Furthermore, there are some cancers associated with 'poor diet' (eg stomach, oesophagus, liver) and some with 'good diet' (eg colorectum, breast, prostate). Although in Europe the latter tend to be more important than the former, globally (Table 1) the cancers associated with poor diet have

Table 1 The major causes of cancer mortality globally in 1990 (Murray & Lopez 1997), and their ranking in comparison with other diseases

Cancer site	*Number of deaths (thousands)*	*Rank amongst all causes*
Trachea, bronchus and lung	945	10
Stomach	752	14
Liver	501	22
Colon and rectum	472	24
Oesophagus	358	26
Breast	322	29

much higher mortality rates (Murray & Lopez, 1997). In Europe, countries with a high risk of one group tend to have a low risk of the other group of cancers and vice versa. In consequence, many diet factors that appear to be risk factors for colorectal cancer appear to be protective for gastric cancer. There is no value in promoting a dietary change that purports to protect against cancer at one site if it promotes cancer at another. We need to steer a middle course, and to commend only those diet changes that reduce the risk of cancers at a wide range of sites, and that do not increase the risk at any.

Epidemiological studies show that avoiding overweight, and eating a diet rich in fruit, vegetables and whole grain cereals might achieve that aim. The evidence for this is summarized elsewhere (eg Hill, 1999), and is accepted by almost all cancer authorities and societies. In 1997 the WCRF produced a report entitled 'Food, nutrition, and the prevention of cancer: a global report'. It produced a set of diet recommendations, the first six of which dealt with plant foods and were unexceptional; they would be supported in principle by most cancer scientists. The seventh, which was highlighted in all the publicity, concerned meat, and it stated that 'if eaten at all, consumption of red meat should be less than 80 g per day'. This recommendation was said to be justified because the majority of epidemiological studies supported it, and none showed protection by meat. It was stated that if red meat consumption was decreased then the mortality from colorectal cancer would decrease too.

This is an important recommendation. Approximately 97% of Europeans eat meat and enjoy it. If they have to decrease their intake of meat it will result in a decreased enjoyment of their food. That would be justified if the benefit were clear, but it would be unjustified if there was doubt about the value of the change. The rest of this paper is a critical examination of the evidence regarding meat and cancer.

The nature of the evidence

Evidence on the role of meat in human carcinogenesis comes from human epidemiology, animal models, *in vitro* studies, and dietary intervention studies. All of these have strengths and weaknesses, and these were discussed elsewhere (Hill, 1995). In summary, the animal models and the *in vitro* systems can be used to demonstrate that the diet is a risk factor in carcinogenesis. However, the different models give different answers concerning the nature of that role (eg Hill, 1989) and so information gained with one model may not apply at all to a different model of the same tumour site. In view of this, the models can only provide data on the mechanisms of known relationships, and cannot be used to prove the validity of those relationships.

Human epidemiology of meat and colorectal cancer

The WCRF committee concluded that the majority of epidemiological studies showed red meat to be a risk factor for colorectal cancer, and perhaps for breast and gastric cancer and that none showed it to be protective. There are three broad types of epidemiological study, namely population, case–control, and prospective studies.

Population studies. Population studies of red meat or animal protein intake and colorectal cancer do, indeed, usually show such a correlation for colorectal cancer (Gregor *et al*, 1969; Armstrong & Doll, 1975). Since all such studies are working with basically the same data set the agreement is unsurprising. Note, though, that such studies also show an *inverse* correlation between meat intake and gastric cancer risk (Gregor *et al*, 1969; Armstrong & Doll, 1975). This contradicts one of the claims of the WCRF Committee.

However, it is likely that they would have given little weight to the results of population studies, in which the populations, although having a wide range of intakes of red meat, would also differ in many other respects as well as diet. These 'other respects' can be controlled for in case–control studies, which are therefore given more weight.

Case–control studies. In 1973 Haenszel *et al* found that beef was a risk factor for colorectal cancer in Hawaiian Japanese. A follow-up study in Japanese living in Japan (Haenszel *et al*, 1980) failed to repeat the observation. A

Table 2 Recent European case-control studies of meat and colorectal cancer

Study	*Observation*
Franceschi *et al* (1997)	More than 2000 cases and 4000 controls; no relation between meat intake and colorectal cancer risk
Faivre *et al* (1997)	No increased risk with high consumption of red meat
Boutron-Rualt *et al* (1999)	No relation between red meat intake and colorectal cancer risk
Augustsson *et al* (1999)	No relation between red meat intake and colorectal cancer risk
Kampman *et al* (1999)	In a US study, no relation between red meat intake and risk of colon cancer
Slattery *et al* (2000)	In a US study, no relation between red meat intake and risk of colon cancer

review of the early case–control studies (Boutron *et al*, 1991) showed a relation between meat and colorectal cancer in nine of 15 studies. A feature of that review was that most of the studies from North America showed a relationship whilst the European studies tended not to (Hill, 1997). Since that review there have been many more studies. Truswell (2000) reviewed 30 case–control studies, of which 20 showed no relationship between red meat intake and colorectal cancer risk, four showed a clear correlation, and six showed a correlation in some but not all sub-groups. Table 2 shows the results of some recent European studies which were published since 1996 and so were not considered by the WCRF committee. In addition, two recent US studies are included. They all fail to show any relation between red meat and colorectal cancer.

The main problem with case–control studies is the difficulty in determining the diet from before the onset of symptoms (since the symptoms affect the diet consumed). Consequently, most epidemiologists would consider that cohort studies (in which the main determination of diet is unaffected by symptoms) give much more reliable evidence. The WCRF Committee gave much credence to two cohort studies from Boston, on nurses (Willett *et al*, 1990) and health professionals (Giovannucci *et al*, 1994). In both of these studies there was an excess risk of colorectal cancer only in the highest intake group (Table 3), suggesting a possible excess risk for those eating more than 140 g red meat per day. This result was recently supported by Hsing *et al* (1997). However the huge study by Hirayama (1990) showed an *inverse* correlation between meat intake and colorectal cancer risk, and in 10 other studies reviewed by Truswell (2000), including all four European studies, there was no relationship seen. The European studies are summarized in Table 4. An important contribution was the pooled analysis of cohort studies of vegetarians compared with omnivores (Key *et al*, 1999), which showed no difference at all between meat eaters and vegetarians with respect to risk of colorectal or breast cancers, or total cancers.

In conclusion, there is no evidence that, in the European context, there is any relation between red meat intake and colorectal cancer risk. Some support for such a relation is available from American studies, but only at the high intake levels of more than 140 g per day.

Red meat and cancer at other sites

In the WCRF report the point was made that meat does not protect at any site. The large Italian case-control study (Buiatti *et al*, 1991) is one of the few studies to find this. In contrast the population studies and the majority of case-control studies of gastric cancer show meat intake to be *inversely* related to cancer risk (Table 5). The problem with studies of meat and gastric cancer is that in may parts of the world most of the meat consumed is preserved, and the preservation methods used (eg salt and nitrite) have been associated independently with gastric cancer risk. The huge prospective study of 265 000 Japanese followed for 25 y

Table 3 Dose–response relationship between meat intake and risk of colorectal cancer in cohort studies

	Risk of colorectal cancer by quintile of meat intake relative to that in the first (lowest) quintile			
Study	*2*	*3*	*4*	*5*
Willett *et al* (1990)	1.2	1.3	1.1	1.8
Giovannucci *et al* (1994)	1.0	1.0	1.2	1.7
Thun *et al* (1992)	1.0	1.1	1.0	1.1
Bostick *et al* (1994)	1.1	1.2	0.9	1.0
Phillips & Snowden (1985)		1.4		0.9

Table 5 Meat and gastric cancer risk

Study	*Type*	*Observation*
Armstrong & Doll (1975)	Population	Inverse relationship
Gregor *et al* (1969)	Population	Inverse relationship
Correa *et al* (1983)	Population	Inverse relationship
Graham *et al* (1990)	case–control	No relationship
Boeing *et al* (1991)	case–control	No relationship
Tuyns *et al* (1992)	case–control	Inverse relationship
Buiatti *et al* (1991)	case–control	Meat is a risk factor
Kaaks *et al* (1998)	case–control	Meat non-significantly protective
Azevedo *et al* (1999)	Regional	Inverse relationship
Hirayama (1990)	Prospective	Inverse relationship

Table 4 European cohort studies of meat and colorectal cancer

	Relative risk of colorectal cancer by quintile of meat intake				
Study	*1*	*2*	*3*	*4*	*5*
Goldbohm *et al* (1994)	(1.0)	0.9	1.2	1.0	0.8
Knekt *et al* (1994)	(1.0)		0.7		1.0
Gaard *et al* (1996)	(1.0)	0.9	0.9	0.6	
Bjelke (1980)	No relationship				
Kinlen *et al* (1983)	No relationship				
Key *et al* (1999)	No difference in risk between vegetarians and matched omnivores				
Cox and Whichelow (1997)	No relation between meat intake and colorectal cancer risk				

The data for Gaard *et al* (1996) are for fried or roasted meat; those for Knekt (1994) are for fried meat; Goldbohm *et al* (1994) are for fresh meat.

found meat to be a major protection against gastric cancer. Note that the WCRF report is 'A Global Report'. Globally, gastric cancer is much more important than colorectal cancer and so these protective effects would have been expected to take priority.

Other data

The WCRF Report states that if we decrease our meat intake then our risk of colorectal cancer will decrease too. In the UK during the period 1963 to 1998 the intake of red meat decreased by 25% (Table 6). Far from falling substantially, the incidence of colorectal cancer (Hill, 1999) *increased* substantially! Similarly, in Norway the intake of red meat has remained steady or has even decreased at a time when their risk of colorectal cancer increased by 50% (Trettli S, personal communication).

The lack of linkage between meat intake and colorectal cancer risk can be illustrated further by comparing the countries in the European Union (Table 7). Meat consumption in the UK is less than that in any of the EU Mediterranean countries (Hill, 1999) and yet the colorectal cancer risk is much higher. Spain has a very much higher intake of red meat and the third lowest colorectal cancer risk.

A possible explanation

The study by Hirayama (1990) provides a possible explanation. In this huge study of 265 000 Japanese persons followed for 25 y, a detailed diet history was taken at recruitment and regularly updated. He could therefore follow changes in the Japanese diet, and so the study is a goldmine of information. When he analysed the data, he first stratified into four broad intake groups. These were (a) daily, (b) often, (c) sometimes, and (d) never consumed. He stratified them thus for vegetable intake, then analysed the relation between meat consumption and cancer risk in each group. Of course, group (a) had very much lower cancer rates than group (d) with the other two groups intermediate, as would be expected. Interestingly, in the group who ate green-yellow vegetables daily, there was an inverse relation between meat intake and

Table 6 Intake of red meat (bovine, sheep + goat, and pig-meat) in the UK between 1963 and 1998, in relation to colorectal cancer risk. Meat intake data were from the FAO website (http://apps.fao.org/lim500/nph-wra)

	Intake of meat (kg/y/person)				*Cancer incidence*		
Year	*Bovine*	*Sheep*	*Pig*	*Total*	*Colon*	*Stomach*	*Breast*
1963	26.4	11.0	26.4	63.8	18.6	31.2	71.3
1968	23.3	10.7	27.4	61.4	*	*	*
1973	22.0	8.4	27.7	58.1	24.5	32.1	73.0
1978	24.1	6.9	26.3	57.3	25.5	28.9	74.4
1983	21.0	7.2	26.1	54.3	27.5	28.9	78.0
1988	21.6	6.8	26.1	54.5	29.8	24.7	95.2
1993	16.8	6.8	24.3	47.9	31.6	21.3	101.4
1998	16.3	6.2	25.9	48.4	30.5	20.1	107.0

Table 7 Red meat intake (cow, sheep + goat and pig meats) in EU countries in 1993 (kg/person/annum), and mortality from cancer of the large bowel (males), prostate (females) and breast (females) (age-adjusted deaths/100 000/annum)

	Red meat intake				*Cancer mortality*		
Country	*Cow*	*Sheep/goat*	*Pig*	*Total*	*Large bowel*	*Prostate*	*Breast*
Austria	23.1	1.1	66.4	90.6	23.5	17.3	21.8
Belgium	21.3	2.0	53.2	76.5	19.2	18.3	25.8
Denmark	2.4	1.0	64.8	86.2	23.3	19.5	27.2
Finland	19.1	0.3	29.4	48.8	13.9	18.1	16.5
France	26.4	4.4	35.8	66.6	20.8	16.6	19.7
Germany	17.8	0.9	54.4	73.1	22.6	16.6	22.1
Greece	20.3	14.4	21.2	55.9	9.5	8.8	15.5
Ireland	17.5	9.8	32.7	60.0	24.8	18.4	26.5
Italy	26.5	1.7	34.2	62.4	19.4	11.4	20.4
Netherlands	18.6	1.3	58.1	78.0	20.1	18.7	26.8
Portugal	17.4	3.5	34.6	55.5	18.8	15.2	18.1
Spain	13.2	6.3	53.2	72.7	16.7	13.5	17.4
Sweden	17.3	0.6	33.4	51.3	15.2	21.1	17.4
UK	16.8	6.8	24.3	47.9	21.1	17.2	27.1

Cancer mortality data from Levi *et al* (1999) for the period 1990–1994. Meat intake data from the FAO website (http://apps.fao.org/lim500/nph-wra).

Table 8 Data from the prospective study of 265 000 Japanese followed for 25 y by Hirayama (1990)

Group	*Observation*
Total population	Little relation between meat consumption and risk of colon and rectal cancer. Meat inversely related to gastric cancer risk.
Those eating GYV daily	Meat consumption inversely related to colon and rectal cancer risk.
Those not eating GYV daily	Meat consumption correlated with colon cancer risk

Table 9 Dietary sources of some minerals and vitamins, illustrating the contribution of meat; Data from USDA (1980)

	Percentage contribution to intakes of			
Food source	*Ca*	*Fe*	*Mg*	*Vitamin B_6*
Meat, poultry, fish	8.4	33.8	18.0	39.3
Grain products	22.7	33.1	21.3	18.7
Dairy products	45.8	3.5	17.2	10.9
All other foods	23.1	29.6	43.5	31.1

colorectal cancer (Table 8). In the group who never ate vegetables there was a positive correlation between meat intake and cancer of the colon and rectum.

If meat is only a risk factor for those who do not eat sufficient amounts of the protective factors, this would provide an explanation of time course of meat and colorectal cancer in the UK seen in Table 6. Over that time period, vegetable and cereal intake fell progressively, and it is this fall that correlates with the increased colorectal cancer risk, not the (decreasing) meat intake.

Further, this would also explain the data in Table 7. The UK has a lower meat intake than any of the Mediterranean countries in the EU and yet it has a much higher colorectal cancer mortality. However the UK has a much lower intake of vegetables and cereals than do those populations, and it is this that determines the risk of colorectal cancer.

Finally, it would explain why meat intake is only a risk factor in the highest quintile of intake (more than 140 g per day); at that level of intake it would begin to displace the (protective) plant foods from the diet.

Conclusions

The speakers at this conference are not alone in rejecting the claimed correlation between red meat intake and colorectal cancer. Truswell (1999) reported the conclusions of an expert workshop on meat intake and colorectal cancer risk organized by the Australian CSIRO. Their conclusion was that there was no evidence of such a risk. Since meat is such an important source of iron and vitamin B6, as well as of calcium and magnesium (Table 9), the benefits of meat in the diet should not be ignored. The Australian Cancer Society, in launching its Lifetrack Cookbook, advocates a balanced diet containing red meat as part of a balanced varied diet rich in fruit and vegetables.

In New Zealand a similar expert committee reached a very similar conclusion to that reached by the Australians (Tasman-Jones, 1999). A report to the Swedish Food Administration by Pearson and Bruce (1998) similarly concluded that there was no evidence to justify a recommendation to decrease meat intake.

The UK Committee on the Medical Aspects of food policy (COMA) concluded that there was a risk only for those consuming more than 140 g red meat per day (Department of Health 1998), and then the risk was of substituting (neutral) meat for (beneficial) vegetables.

The conclusion should be that we should encourage increased intake of fruit, vegetables and whole grain cereals. If the intake of those is sufficient, then there is no need to worry about meat intake. In fact, because of its major contribution to the protein and micronutrient intake, meat should be encouraged as a valuable component of a rich and varied diet.

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