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REVIEW

Red meat in the diet: an update

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Summary

This paper provides an update of a previous review '*Red Meat in the Diet*' published in the *Nutrition Bulletin* in 2005. An update on red meat consumption levels in the UK and other countries is provided, and a summary of the nutritional content of red meat in the diet is given. Current evidence on dietary and lifestyle factors associated with red meat consumption and the effects of red meat intake on health and chronic disease outcomes are discussed. As there is now continued debate about the environmental impact of different aspects of our diet, sustainability issues regarding red meat were also discussed.

Keywords: chronic disease, health, nutrient composition, processed meat, red meat

Definitions

This paper will adopt the same definitions used by Williamson *et al*. (2005) for red and processed meat, which are based on definitions most commonly used in epidemiological studies looking at the health effects of meat consumption, such as the European Prospective Investigation into Cancer and Nutrition (EPIC). Where studies have used different definitions, this is highlighted (Box 1).

Box 1 Definitions of red and processed meat
Red meat includes beef, veal, pork and lamb (fresh, minced and frozen).
Processed meat includes meat that has been preserved by methods other than freezing, such as salting, smoking, marinating, air-drying or heating, *e.g*. ham, bacon, sausages, hamburgers, salami, corned beef and tinned meat.
Source: (Linseisen *et al*. 2002).

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Evidence base

The majority of evidence in this review is from epidemiological studies investigating associations between meat intake and health/disease outcomes. Comparisons between studies are often not possible because of the lack of a standardised definition of what is meant by red meat. Some studies examine total meat consumption, which may include red and white meat and processed meat, while other studies analyse these separately. Williamson *et al*. (2005) discussed the definitions used in more detail.

Collecting accurate dietary data is a challenging aspect of nutritional epidemiology. It is particularly important to measure food and nutrient intake as accurately as possible in order to detect true associations. Most prospective cohort studies use food frequency questionnaires (FFQs) to assess usual food consumption. Although other methods would be more adequate to estimate portion sizes, such as food diary or 24-hour recall methods, there are major advantages of using FFQs in epidemiological studies. Food diaries and 24-hour recalls are generally used to assess the intake of foods and nutrients over a short period of time. Such information from a large enough population is useful to look at average intakes and compare them with dietary recommendations and are therefore useful in public health. However, in prospective studies investigating links between dietary habits and disease, habitual intakes over a longer period of time need to be assessed, and FFQs are useful for this purpose. They are relatively cheap and therefore the common choice for large cohorts. However, there are also limitations: it is difficult to assess portion sizes; respondents tend to overestimate consumption of what they consider ‘good’ foods and underestimate consumption of what they think are ‘bad’ foods, and recent changes in dietary habits may bias the reporting of food intakes prior to changes. Such limitations must be considered when interpreting the evidence. All FFQs should be validated, which means that their accuracy should have been assessed in a separate study comparing its outcomes with other more accurate methods, such as food diaries (Nelson *et al.* 2004). Ideally, the FFQ should be repeated throughout long prospective studies as dietary habits may change over time. However, many prospective studies only assess dietary habits at baseline, which may lead to bias in studies that last several years.

Although intervention studies generally deliver more accurate data, these tend not to be used when examining the influence of diet on chronic disease outcomes such as cardiovascular disease (CVD) and cancer risk for several reasons. It would be unethical to ask people to follow a diet that is thought to be associated with a higher risk of CVD or cancer. Also, people would have to be followed for long periods of time and therefore compliance with any dietary intervention is likely to be low. They are more commonly used to assess effects on disease risk factors such as hypertension or elevated blood cholesterol.

When exploring the relationship between red meat intake and health and chronic disease outcomes, this paper reviewed evidence from prospective cohort studies wherever possible as these are generally less prone to bias than other epidemiological study designs. However, cohort studies investigating chronic diseases such as CVD and cancer need to be very large to deliver statistically significant data and are very costly. Therefore, the number of available cohort studies is limited, and where data from cohort studies was not available, data from case control studies were considered.

Red meat consumption

Meat consumption trends vary greatly across the globe. Furthermore, some individuals choose to either avoid meat altogether or certain types of meat for a variety of reasons, such as ethical or religious reasons, or because of socio-economic factors.

Data sources and quality

There are different sources of data on food supply and consumption. Food balance sheets (FBS) provide data on the supply of foods of a whole country. FBS are agricultural statistics made up of food supply (including production and import) as well as food export information (changes in food stocks and utilisation for non-food purposes) such as feed, seed and manufacture. Data from FBS, which are collected annually, are useful to show trends in supply within a country, but no conclusions can be made on actual food consumption of individuals. Because a large quantity of material is discarded from meat (*e.g.* bones, cartilage, trimmed fat and wastage) prior to its presentation for consumption, FBS represent an overestimate of the available meat within a given population.

More accurate data on food trends at a household level come from the Expenditure and Food Survey, also known as the Living Costs and Food module of the Integrated Household Survey [Department for Environment Food and Rural Affairs (Defra) 2010]. Data include food purchased for consumption in the household as well as expenditure on items purchased out-of-home. Major advantages compared with FBS are that they give a more accurate estimation of what is consumed in a household. They also show trends in consumption rather than simply supply.

In contrast to FBS and household expenditure surveys, dietary intake surveys quantify food intake at an individual level. For this reason, they may more accurately reflect the amounts and type of foods consumed depending on the survey method. In the UK, intake at the individual level is assessed within the National Diet and Nutrition Surveys (NDNS). The method used for the current NDNS rolling programme is a 4-day food diary, whereas earlier surveys used 7-day weighed diaries. Intakes at the individual level can not easily be compared between different countries because of differences in the survey methodologies used in different areas (Elmadfa *et al.* 2009) or even different studies within a single country. However, data from the EPIC study provide comparable data of meat intakes in Europe. This is because this large prospective cohort study, which spans ten European countries ($n = 519\,978$), uses a standardised methodology to collect dietary data (Linseisen *et al.* 2002).

Accurately quantifying the amount of meat consumed in the diet is problematic, owing to the fact that meat is typically consumed as part of a composite meal, often containing non-meat components such as vegetables, pasta, legumes or potatoes (Cosgrove *et al.* 2004). For example, overestimation of meat consumption using aggregated data (where meat has not been separated from other ingredients in composite dishes such as pies or pasta dishes) has been reported to be as high as 32% to 40% (Prynne *et al*. 2009); (Cosgrove *et al*. 2004). For this reason, for a more accurate reflection of consumption levels in the UK, data from composite dishes have, for the first time, been reported in a disaggregated form in the new NDNS rolling programme (as well as the traditional 'aggregated' form). However, as historical data do not provide disaggregated figures, care must be taken when comparing meat intakes over time (Fitt *et al*. 2010).

Meat consumption: the UK perspective

Average intakes of meat in the UK have been estimated within the NDNS programme. Although the most recent NDNS (2008/2009) provide more up-to-date consumption data, the results of this survey may not be entirely representative of the UK population because of the small sample size from the new rolling programme. As survey data will be collected every year, only a small number of people will be surveyed each year (approximately 1000 people across all age groups) compared with the large sample sizes of previous surveys (*e.g.* a combined total of 3425 people in the 2000/2001 NDNS Adults and 1997 NDNS Young People surveys). Over the coming years, as more survey data are collected, the sample size for the rolling programme will increase and the data will become more representative of the UK population (Riley 2010). There are also differences in the recording of food intakes to limit the burden to the respondents and therefore ensure a reasonable response rate [*i.e.* from a 7-day weighed food diary in previous surveys to a 4-day estimated (unweighed) diary in the current survey]. Nevertheless, the methodology was carefully checked to ensure it remained reliable. For this reason, data from the 1997 and 2000/2001 studies were recalculated on a 4-day basis for comparative purposes.

The 2008/2009 NDNS data suggest an increase in total meat consumption (red and white meat) in males and females in all age groups when compared with data from the 1997 NDNS young people survey (Gregory *et al*. 2000) and the 2000/01 NDNS adult survey (Henderson *et al*. 2003a) (see Table 1, which provides data based on 4-day estimates as reported in the 2008/09 NDNS Rolling Programme report for comparison) (Bates *et al*. 2010). Red meat consumption was greater than white meat consumption. With regard to red meat consumption (beef, lamb, pork, veal, sausages, burgers and kebabs) in the UK, the average daily intake (grams) of red meat has increased since 1997 for all age groups. Males consume more red meat (grams/d) than females (see Table 1). Adult males reported consuming 123 g/d (2000/2001) on average vs. 149 g/d (2008/2009) of red meat, whereas females reported intakes of 73 g/d (2000/2001) vs. 102 g/d (2008/2009). However, it is important to note that these figures are likely to be an overestimate of the amount of meat actually consumed (see next section).

Table 2 provides estimates of current UK intakes of red meat from disaggregated intake data from the NDNS surveys (*i.e.* figures are for meat only, including meat components from meat dishes). Results also suggest that males (aged 11-to-18-years and 19-to-64-years) have higher average daily intakes of red meat than females, whereas estimated intakes for those aged 4-to-10-years are similar for both genders. These results also reveal an estimated average meat intake of 134 g/d for men and 91 g/d for women, with red meat intake being greater than white meat intake (chicken and turkey).

Estimates of current UK intakes for year 1 (Table 2) may also overestimate the amount of meat actually eaten because the 4-day food diaries in the 2008/2009 NDNS were recorded to include both Saturday and Sunday and systematic differences have been observed in the reporting of intakes on the weekend as opposed to weekdays. This could have led to an overestimate in the amount of meat consumed because of oversampling of weekend days in the analysis (Sundays are often a day with high meat and vegetable intake because of the popularity of the traditional Sunday roast). This will be addressed by the sampling protocol in the next collection period, which has been modified to include fewer weekend days to even out the data (Riley 2010). In addition, the disaggregation of composite dishes into their separate food components in the recent survey provides a more accurate estimate of actual consumption of food types, but as this was not performed in previous surveys, direct comparisons could not be made. Hence, for comparative purposes, intake data for year 1 was compared to previous surveys using data that represented traditional methods (*i.e.* aggregated data/included non-meat components of meat dishes). Because former results were derived using 'traditional' NDNS survey methods, the stand-alone estimates of current intakes (taken from the 2008/2009 NDNS) may represent an overestimate of the amounts actually eaten, hence any observed increase may correspond to a higher content of non-meat components in meat dishes (*e.g.* vegetables) rather than meat *per se*. It should be noted that consumption of meat provides an opportunity to also consume vegetables.

Table 1 Average daily intake (grams) of meat for people aged 4-to-64-years (including non-consumers), comparing reanalysed data from the 1997 NDNS Young People Survey and 2000/01 NDNS Adults Survey with data from the 2008/09 NDNS Rolling Programme

	4–10 years					
	Boys		Girls		Total	
Intake by meat type, in grams	1997	2008/09	1997	2008/09	1997	2008/09
Total meat	86	110	82	113	84	111
red meat	60	74	54	75	57	74
white meat	26	36	28	38	27	37
	11–18 years					
	Boys		**Girls**		**Total**	
Intake by meat type, in grams	**1997**	**2008/09**	**1997**	**2008/09**	**1997**	**2008/09**
Total meat	150	199	106	146	126	172
red meat	105	122	68	94	85	108
white meat	45	77	38	52	41	64
	19–64 years					
	Men		**Women**		**Total**	
Intake by meat type, in grams	**2000/01**	**2008/09**	**2000/01**	**2008/09**	**2000/01**	**2008/09**
Total meat	184	217	118	161	146	189
red meat	123	149	73	102	95	125
white meat	61	68	45	59	51	64

Notes: (1) Red meat includes beef, lamb, pork, veal, sausages, burgers and kebabs. White meat includes chicken and turkey. (2) The figures presented in Table 1 include non-meat components of meat dishes, so are an overestimate of intakes. (3) The 1997 and 2000/01 figures for average daily consumption (Table 1) are reanalysed figures, based on 4-day estimates, as reported in the 2008/09 NDNS Rolling Programme report.

Table 2 Average daily intake of meat (grams) for people aged 4-to-64-years from disaggregated data reported in the 2008/2009 NDNS rolling programme

	4–10 y			11–18 y			19–64 y		
Intake by meat type (g)	Boys	Girls	Total	Boys	Girls	Total	Men	Women	Total
Total meat	71	70	71	119	84	102	134	91	113
Red meat	47	47	47	75	54	65	96	57	76
White meat	24	23	24	44	30	37	38	34	36

Notes: Red meat includes beef, lamb, pork, veal, sausages, burgers and kebabs. White meat includes chicken and turkey. The figures presented in Table 2 are for meat only, including meat components from meat dishes. The figures presented in Table 2 are from the report of the 2008/2009 NDNS Rolling Programme. In some instances, the average total daily meat intake does not equal the sum of the intake of red meat and white meat because of rounding.

The UK Women's Cohort study is one of the largest cohorts investigating the relationship between diet and cancer ($n = 33\,725$). Baseline data were gathered between 1995 and 1998 using a FFQ, developed from the EPIC study and validated against a semi-weighed 4-day food diary. This study categorised meat consumption levels as low [<62 g/d; mean age 53 (SD 9)], medium [62–103 g/d; mean age 54 (SD 9)] or high [>103 g/d; mean age 53 (SD 9)], with red meat consumption at <32 g/d for those reported as exhibiting low intake, 32–57 g/d for medium intake and >57 g/d for high meat intake (Taylor *et al.* 2007). An earlier Irish study reporting on the North/South Ireland food consumption survey (1997–1999) estimated meat intakes,

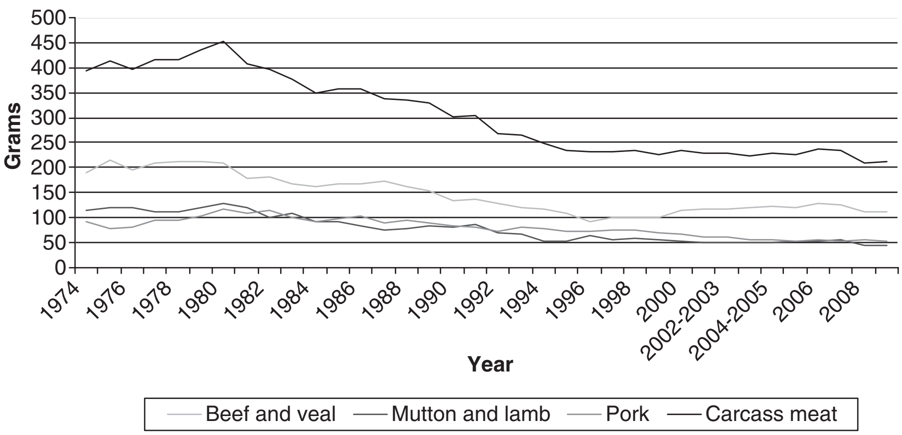

Figure 1 UK household purchased quantities of food and drink: averages per person per week. Source: Defra 2010: http://www.defra.gov.uk/evidence/statistics/foodfarm/food/familyfood/index.htm

calculated using disaggregated data on composite foods to exclude the contributions from non-meat components as 51 g/d, with males consuming significantly more red meat than females (Cosgrove *et al.* 2005). Overall, red meat was consumed by 88% of the Irish population (92% of men and 84% of women), with men and women consuming an average of 64 g/d and 38 g/d of red meat, respectively.

In contrast to the NDNS findings, household level data from the annual reports ('Family Food') published by the Department for Environment, Food and Rural Affairs (Defra) suggest a decrease in red meat consumption over the last three decades. These data on food and drink purchases by household in the UK provides information on purchased quantities, expenditure and nutrient intakes derived from both household and food and drink consumed outside the home. However, as these data do not reflect actual food consumption of individuals, direct comparisons with studies assessing individual intake, such as the NDNS, can not be made. Household purchases of raw carcass meat have fluctuated over the last decade, and since 2006, they have decreased overall by 10.7% (Defra 2010) (see Fig. 1). The most recent data from the Family Food Survey show that the amount of 'beef and veal' and 'lamb and mutton' purchased rose between 2008 and 2009, while the amount of pork fell. 'Meat based ready meals[1] and convenience meat products'[2] purchases have risen 4% in 2009 and 'all other meat products' dropped 4.4%.

[1]Includes hotpot, beef stew, casseroles, moussaka, beef risotto, meat-based pasta dishes, curry and Chinese meals.

[2]Includes breaded, battered meat, meat with substantial sauce, stuffed joints, breaded turkey escalopes, chicken kiev, savoury duck, black pudding, burgers in buns and pre-packed meat salads.

Meat consumption: the European perspective

Within Europe, the EPIC study (which used a consistent methodology across the participating countries) reported high variation in meat intake amongst the European countries/centres participating in this study, with the lowest meat intakes reported in Greece and the highest meat intakes in Spain (see Table 3). However, the authors stressed that the cohorts described in this study are not entirely representative of the population, although the sample size is sufficiently large enough to be used as reference data. Red meat intake ranged from 24–57 g/d in women and 40–121 g/d in men (Linseisen *et al.* 2002) within the ten countries participating. Germany showed the highest consumption of pork, whereas mutton/lamb was most frequently consumed in France and Spain. Processed meat (such as hamburger, schnitzel and cold roasted meat) was most popular in Sweden, Norway and Germany. Unfortunately, more up-to-date figures from EPIC are not available at present. However, as this is a prospective study, more data should be available in due course.

Meat consumption: the North American and Australian perspective

In the USA, Wang *et al.* (2010) reported a U-shaped trend in general meat consumption since 1988 using national food survey data from the two most recent National Health and Nutrition Examination Surveys (NHANES) 1988–1994 and 1999–2004 and the Continuing Survey of Food Intakes by Individuals (CSFII) (1994–1996) in which individual food intakes were estimated with 24-hour dietary recalls. In terms of red meat consumption, the average intake in g/d/person was 45.4 g (NHANES 1988–1994), 32.3 g (CSFII

Table 3 Mean daily intake (g/d) of total meat, red meat, processed meat and red + processed meat in selected countries

	Total meat‡		Red meat¶		Processed§ meat		Red meat + processed meat	
	Men	Women	Men	Women	Men	Women	Men	Women
UK*	108.1	72.3	40.0	24.6	38.4	22.3	78.4	46.9
Ireland†	167.9	106.6	63.9	37.5	30.9	19.9	94.8	57.4
Greece*	78.8	47.1	45.3	25.5	10.0	5.8	55.3	31.3
Spain*	170.4	99.2	74.0	37.8	52.8	29.6	126.8	67.4
Italy*	140.1	86.1	57.8	40.8	33.5	19.6	91.3	60.4
France	NA	106.0	NA	44.4	NA	30.0	NA	74.4
Germany*	154.6	84.3	52.2	28.6	83.2	40.9	135.4	69.5
The Netherlands*	155.6	92.7	63.8	41.0	72.4	37.9	136.2	78.9
Denmark*	141.1	88.3	69.6	44.1	51.9	25.3	121.5	69.4
Sweden*	138.8	91.9	56.8	35.3	65.8	43.3	122.6	78.6
Norway*	NA	88.6	NA	28.5	NA	46.4	NA	74.9
Australia	199.9**	116.1**	88.0††	45.0††	22.1¶¶	9.6¶¶	110.1	54.6
Canada§§	NA	NA	73.0	40.0	28.0	15	101.0	55.0
USA***	260***	168.5***	52.9***	28.0***	NA	NA	NA	NA

Source: *Linseisen *et al.* (2002) †Cosgrove *et al.* (2005).

‡Total meat includes pork, beef, veal, lamb/mutton, poultry, game, rabbit, horse, goat and offal.

¶Red meat includes beef, veal, pork and lamb/mutton.

§Processed meat includes ham, bacon, processed meat cuts, minced meat and sausages.

**Total meat includes muscle meat (beef, corned beef, veal, lamb, pork, bacon, ham), game (kangaroo, rabbit, venison), poultry (chicken, turkey, duck, quail, emu), sausages, frankfurters, saveloys, processed meat (delicatessen meat, ham paste, canned corned beef), mixed dishes where beef or veal is the major component, mixed meat dishes which include lamb or pork, bacon, ham as the major component, mixed dishes where poultry or game is the major ingredient. Excludes meat in cereal based products (meat pies, pizza, hamburgers). Mean intake data for persons aged 19 years and over. Australia Bureau of Statistics, Commonwealth Department of Health and family services. National Nutrition Survey Australia, 1995. Canberra ABS; 1999.

††Baghurst K, Record S and Leppard P. 'Red meat consumption in Australia: intakes, nutrient contribution and changes over time'. *Australian Journal of Nutrition and Dietetics* 57 (2000): 1032–22. Red meat includes beef, veal, lamb from cuts, mixed dishes and products (stews, curries, rice and pasta dishes, meat pies where only the meat component has been included).

¶¶Processed meat has been derived from mean daily intake data of processed meat (delicatessen meat, ham paste, canned corned beef) and sausages, frankfurters, saveloys. Mean intake data for persons aged 19 years and over. Australia Bureau of Statistics, Commonwealth Department of Health and family services. National Nutrition Survey Australia, 1995. Canberra ABS; 1999.

§§Males and females ≥ 19 years of age, Source: Statistics Canada, Canadian Community Health Survey 2.2.

***Wang *et al.* (2010). Total meat including all animal source food and red meat consisted of beef, pork, lamb, veal and game.

1994–1996) and 39.9 g (NHANES 1999–2004) over time, with males consuming 62 g, 32.7 g and 52.9 g during these respective periods compared with 30.4 g, 31.9 g and 28 g for women (see Table 3). Overall, the results suggest a reduction in meat intake among US females but an increased intake among US males. See Table 3 for consumption data for Canada and Australia.

Nutrient composition of red meat

Red meat contains high biological value protein and important micronutrients, all of which are essential for good health throughout life. Meat is a source of fat and contributes to intake of saturated fatty acids (SFAs). However, meat contains a range of fatty acids, including the essential omega-6 (n-6) and omega-3 (n-3) polyunsaturated fatty acids (PUFAs) [linoleic and α-linolenic acids (ALNAs)]. As depicted by the *Eatwell Plate* model, most healthy balanced diets will include lean meat in moderate amounts, together with starchy carbohydrates (including wholegrain foods), plenty of fruit and vegetables and moderate amounts of milk and dairy foods.

Nutrient composition data

Data on the nutrient composition of red meat are available in numerous food composition tables and databases around the world, although slightly different nutrient values are likely to be found in different versions.

Table 4 shows the nutrient composition of 100 g of lean raw beef, lamb and pork from the food composition tables of a selection of countries. These data were

Table 4 Comparison of selected nutrients in beef, lamb and pork (per 100 g) according to food composition databases from selected countries

	UK	Denmark	Finland	France	Italy	Australia	Canada	USA
Beef, lean, raw								
Energy (kJ)	542	647	639	716	455	528	607	526
Protein (g)	22.5	21.1	19.3	19.5	21.6	23.0	22.0	21.0
Fat (g)	4.3	7.8	8.4	10.4	2.4	3.6	5.6	4.0
SFA (g)	1.7	3.3	4.0	4.4	0.8	1.5	2.3	1.4
MUFA (g)	1.9	3.7	1.9	4.9	0.8	1.6	2.8	1.6
PUFA (g)	0.2	0.3	0.4	0.4	0.5	0.35	0.24	0.2
Niacin (mg)	9.7	10.0	10.4	3.9	5.1	4.1	6.6	6.2
Thiamin (mg)	0.1	0.05	0.09	0.08	0.11	0.04	0.12	0.08
Vitamin B_{12} (μg)	2.0	1.4	1.4	2.0	2.0	1.1	2.6	1.5
Iron (mg)	2.7	2.1	2.5	2.2	1.6	2.0	2.2	1.8
Zinc (mg)	4.1	4.8	4.0	4.8	3.8	4.2	5.8	3.9
Selenium (μg)	7.0	6.8	15.1	No data	5.6	12.0	19.0	26.0
Sodium (mg)	63.0	65.0	51.5	64.0	43.0	49.0	64.0	54.0
Potassium (mg)	350.0	325.0	317.0	342.0	334.0	360.0	327.0	323.0
Lamb (leg), lean, raw								
Energy (kJ)	639	704	767	No data	510	628	473	536
Protein (g)	20.2	20.1	19.0	No data	20.0	22.0	21.8	20.6
Fat (g)	*8.0*	9.8	12.0	No data	4.6	7.0	2.7	4.5
SFA (g)	*3.5*	4.5	4.2	No data	2.2	2.0	1.1	1.6
MUFA (g)	*3.1*	3.6	3.4	No data	1.7	2.7	1.2	1.8
PUFA (g)	*0.5*	0.8	0.4	No data	0.2	1.0	0.0	0.4
Niacin (mg)	*5.4*	7.5	8.4	No data	4.9	5.6	7.8	6.2
Thiamin (mg)	*0.1*	0.2	0.2	No data	0.1	0.1	0.1	0.1
Vitamin B_{12} (μg)	*2.0*	1.2	1.2	No data	2.0	1.0	2.2	2.7
Iron (mg)	*1.4*	1.5	1.9	No data	2.0	2.0	1.3	1.8
Zinc (mg)	*3.3*	3.4	2.3	No data	3.1	4.0	3.8	3.8
Selenium (μg)	*4.0*	1.4	13.5	No data	18.0	13.8	23.5	23.5
Sodium (mg)	*70.0*	66.0	58.0	No data	100.0	63.0	68.0	62.0
Potassium (mg)	*330.0*	218.0	240.0	No data	350.0	325.0	358.0	289.0
Pork, lean, raw								
Energy (kJ)	519	784	669	612	658	461	561	602.0
Protein (g)	21.8	20.0	20.6	21.0	21.3	23.5	21.2	21.2
Fat (g)	4.0	12.0	8.6	6.9	8.0	1.7	4.8	5.9
SFA (g)	1.4	4.8	3.2	2.6	3.7	0.6	1.0	2.0
MUFA (g)	1.5	5.4	3.4	2.9	2.5	0.7	1.3	2.7
PUFA (g)	0.7	0.9	1.0	0.7	1.5	0.3	0.3	0.6
Niacin (mg)	6.9	7.3	8.9	5.3	3.0	9.5	5.6	4.8
Thiamin (mg)	1.0	0.8	1.0	0.8	0.8	1.0	0.7	1.0
Vitamin B_{12} (μg)	1.0	0.7	0.6	0.3	1.0	0.3	0.6	0.7
Iron (mg)	0.7	0.9	0.9	1.7	0.8	0.7	0.8	0.9
Zinc (mg)	2.1	3.6	2.2	1.9	1.6	1.8	2.2	2.0
Selenium (μg)	13.0	6.9	18.3	40.0	14.0	20.0	33.5	32.4
Sodium (mg)	63.0	84.0	58.3	47.0	56.0	49.0	59.0	54.0
Potassium (mg)	380.0	366.0	300.0	370.0	290.0	388.0	364.0	384.0

Notes:
UK – Figures in italics for lamb are from Chan *et al.* (1995), all other figures are from FSA (2002).
Denmark – Danish Food Composition databank ed. 7.01. http://www.foodcomp.dk/v7/fcdb_default.asp.
Finland – Fineli Food Composition Database release 10, 30 June 2009. http://www.fineli.fi/index.php?lang=en.
France – French Food Composition database, version 2008. http://www.afssa.fr/TableCIQUAL/.
Italy – Food Composition Database for Epidemiological Studies in Italy (Banca Dati di Composizione degli Alimenti per Studi Epidemiologici in Italia – BDA) http://www.ieo.it/bda2008/uk/ricercadati.aspx.
Australia – Food Standards Australia New Zealand. NUTTAB 2006 http://www.foodstandards.gov.au/consumerinformation/nuttab2006/onlineversionintroduction/; (Greenfield *et al.* 2009; Müller *et al.* 2009; Sinclair *et al.* 2010).
Canada – Health Canada, Canadian Nutrient File, released 2007. http://webprod.hc-sc.gc.ca/cnf-fce/index-eng.jsp.
USA – US Department of Agriculture, Agricultural Research Service. 2010. USDA National Nutrient Database for Standard Reference, Release 23. http://www.ars.usda.gov/ba/bhnrc/ndl. Beef (NDB# 23651), Lamb (NDB # 17013), Pork (NDB # 10228).

Table 5 Meat consumption data for UK adults (aged 19-to-64-years)

	Men (%)		Women (%)	
Meat type, in grams (% total energy)	2000/2001	2008/2009	2000/2001	2008/2009
Total meat	184 (17)	217 (18)	118 (15)	161 (17)
Red meat	123 (13)	149 (12)	73 (10)	102 (11)
White meat	61 (4)	68 (5)	45 (4)	59 (6)

Notes: Red meat includes beef, lamb, pork, veal, sausages, burgers and kebabs. White meat includes chicken and turkey. The values may include non-meat components of meat dishes. The 2000/2001 values for average daily consumption are the reanalysed values (based on 4 days) as presented in the 2008/2009 NDNS rolling programme report. The 2000/2001 values for percentage contribution to total energy intake for adults is taken directly from the 2000/2001 NDNS Adults Survey.

Table 6 Meat consumption data for UK children and adolescents (aged 4-to-18-years)

	4–10 y (%)				11–18 y (%)			
	Boys		Girls		Boys		Girls	
Meat type, in grams (% total energy)	1997	2008/2009	1997	2008/2009	1997	2008/2009	1997	2008/2009
Total meat	86 (14)	110 (13)	82 (13)	113 (14)	150 (14)	199 (18)	106 (13)	146 (17)
Red meat	60 (10)	74 (10)	54 (9)	75 (11)	105 (10)	122 (12)	68 (9)	94 (11)
White meat	26 (4)	36 (4)	28 (4)	38 (4%)	45 (4)	77 (7)	38 (4)	52 (6)

Notes: Red meat includes beef, lamb, pork, veal, sausages, burgers and kebabs. White meat includes chicken and turkey. The values may include non-meat components of meat dishes. The 1997 and 2000/2001 values for average daily consumption are the reanalysed values (based on 4 days) as presented in the 2008/2009 NDNS rolling programme report. The 1997 values for percentage contribution to total energy intake for boys and girls are a combined average for all children aged 4-to-18-years rather than separate values for 4-to-10-year-olds and 11-to-18-year-olds. Also, this information is taken directly from the 1997 NDNS Young People Survey report.

obtained from the EuroFIR website (http://www.eurofir.net/eurofir_knowledge/european_databases), and illustrate how these figures can vary because of differences in the nutrient composition of the meats selected for sampling, which may be a result of differences in the animal's feeding regimen, differences in sampling techniques, for example values for beef may be based on one particular cut of meat from one breed of cattle, while others may be based on a variety of cuts and breeds, variable time periods of analyses, with some analyses being conducted more recently than others and hence using newer methods and different classifications used for the various cuts of meat. Differences in the age of the animal at slaughter and season of year may also result in variations in the nutrient composition of similar foods.

Energy

The amount of energy provided by meat is variable and will be influenced in particular by the fat content. In the UK, the contribution of meat (all sources) to total energy intake is 18% for men (19-to-64-years) and 17% in women (19-to-64 years) (2008/2009 NDNS data) (see Table 5). The contribution of meat to total energy intake in adolescents (11-to-18-years) is similar to that of adults (18% for boys and 17% for girls), whereas for younger children (4-to-10-years), the contribution is 13% for boys and 14% for girls (see Table 6).

Meat provides virtually no carbohydrate and is principally composed of protein (which provides 17 kJ/4 kcal of energy per gram). Meat also contains fat in varying amounts (providing 37 kJ/9 kcal of energy per gram). The more fat meat contains, the higher the energy content will be as shown in Table 7.

Protein

Dietary protein is essential for growth, maintenance and the repair of the body and can also provide energy. Protein from foods is composed of chains of hundreds to thousands of amino acids. Some amino acids can be synthesised in the body, while others – essential amino acids – can not. Therefore, essential amino acids need to be consumed in the diet to maintain good health. Red meat (and in some cases, meat products), as well as other animal foods, is an important source of the eight essential amino acids for adults and histidine, which is considered to be an additional essential amino acid for children. This is because the pattern of amino acids in animal cells is comparable with the pattern in human cells.

Red meat contains, on average, 20–24 g of protein per 100 g (when raw). Cooked red meat contains 27–35 g of protein per 100 g (cooked weight). As meat is cooked, the water content decreases and the nutrients become more concentrated, therefore the protein content increases. Lean meat contains a higher proportion of protein than fattier cuts (see Table 7).

In most developed countries, average protein intakes for all age groups are in excess of the minimum protein requirements needed for good health, provided energy intakes are sufficient. Any excess protein in the diet is used to provide energy. Recent data from the UK suggest that meat and meat products (including poultry) contribute 40% and 37% of the average daily protein intakes in men and women, respectively, aged 19-to-64-years (Bates *et al.* 2010).

Table 7 Energy, fat and protein content of lean and untrimmed cuts of red meat (per 100 g; UK figures)

Meat (barbequed or grilled)	Energy kJ (kcal)	Fat (g)	Protein (g)
Rump steak – lean	741 (176)	5.7	31.2
Rump steak – lean and fat	849 (203)	9.4	29.5
Leg joint of lamb – lean	879 (210)	9.6	30.8
Leg joint of lamb – lean and fat	986 (236)	13.0	29.7
Loin chops of pork – lean	780 (186)	6.8	31.1
Loin chops of pork – lean and fat	1066 (255)	15.8	28.3

Source: Chan *et al.* (1995).

Fat

Fat provides the richest dietary source of energy and also supplies essential nutrients such as fat-soluble vitamins and essential fatty acids but should be consumed in moderation to prevent excessive weight gain. Fat also provides palatability and flavour to foods. It is now recognised that it is the type of fat rather than the total amount of fat that is particularly important for cardiovascular health (Stanner 2005); different fatty acids have different effects on blood cholesterol levels and risk of heart disease, some beneficial and some adverse.

The three main types of fat found in meat are intermuscular fat (between the muscles), intramuscular fat or 'marbling' within the muscles and subcutaneous or visible fat (below the skin). The fat content of red meat varies depending on the type of red meat, the cut and the degree of trimming as well as the breed, sex, age at slaughter and the feeding regimen that the animal experienced throughout life and just before slaughter (Talbot 2006). In some countries, meat with a low fat content is classified as 'lean meat'. Although there is no international definition, lean meat generally has between 5% and 10% fat (Williamson *et al.* 2005).

Recommendations for the percentage of food energy provided by the various types of fatty acids and the current average intakes in adults in the UK are provided in Table 8. In the UK diet, meat, meat dishes and meat products (combined) are the highest contributor to fat intake, supplying 23% of total fat and 22% of saturated fat intake for adults aged 19-to-64-years (Hoare *et al.* 2004).

Fatty acid composition of meat

The fatty acid composition of meat is dependent on whether or not the species is a ruminant. In ruminant

Table 8 Recommendations and intakes of different fatty acids in the UK diet

Fat	Recommendations (% food energy)	Men		Women	
		2000/2001	2008/2009	2000/2001	2008/2009
Total fat	35	35.5	35.5	34.7	34.7
Saturates	11	13.3	13.0	13.2	12.6
Monounsaturates	13	12.0	12.8	11.4	12.3
n-6 polyunsaturates	Minimum 1%	5.3	5.2	5.3	5.3
n-3 polyunsaturates	Minimum 0.2%	1.0	1.1	1.0	1.1
Trans fat	<2	1.2	0.8	1.1	0.8

Source: Henderson *et al.* (2003a); SACN 2007a; Bates *et al.* (2010).

Table 9 Fatty acid composition of different types of meat

Fat and total fatty acids, g per 100 g food	Beef, average, trimmed lean, raw	Lamb, average, trimmed lean, raw	Pork, average, trimmed lean, raw	Chicken, light meat, raw
Total Fat	4.3	8	4.0	1.1
Saturated Fat	1.74	3.46	1.36	0.31
Cis-MUFA	1.76	2.58	1.5	0.48
Total *Cis*-PUFA	0.2	0.36	0.69	0.22
n-6 PUFA	0.17	0.28	0.61	0.18
n-3 PUFA	0.07	0.16	0.09	0.04
Total Trans	0.14	0.6	0.02	0.01
Total Branched	0.08	0.15	0.01	Trace
C10:0	0	0.02	0	0
C12:0	0	0.04	0	0
C14:0	0.1	0.38	0.04	0.01
C15:0	0.02	0.05	Trace	Trace
C16:0	0.97	1.59	0.83	0.22
C17:0	0.04	0.08	0.01	Trace
C18:0	0.59	1.29	0.45	0.07
C20:0	0	0.01	0.01	0
Cis				
14:1	0.02	0.01	0	0
15:1	0	0.01	0	0
16:1	0.15	0.13	0.09	0.04
17:1	0.04	0.05	0.01	Trace
18:1	1.54	2.37	1.36	0.42
Cis/trans				
18:1 *n*-9	1.47	2.31	1.26	0.4
18:1 *n*-7	0.13	0.43	0.12	0.03
Cis				
20:1	0.01	0.02	0.03	0.01
22:1	0	0	0	Trace
Trans MUFA	0.11	0.51	0.02	0.1
Cis n-6				
18:2	0.11	0.13	0.54	0.16
18:3	0	0.04	0	0
20:3	0.01	0	0.01	Trace
20:4	0.02	0.02	0.04	0.01
Cis n-3				
18:3	0.03	0.09	0.05	0.02
18:4	0	0.01	0	0
20:5	0.01	0.02	0.01	Trace
22:5	0.02	0.03	0.02	0.01
22:6	0	0.01	0.01	0.01
Trans PUFA	0.03	0.09	Trace	Trace

Source: Ministry of Agriculture, Fisheries and Food (MAFF) (1998).
Note: This table includes only the fatty acids present in these types of meat with values above 0.0.

animals (*e.g.* cows, sheep), the majority (>90%) of the dietary unsaturated fatty acids are hydrogenated to SFAs in the rumen during digestion (Lunn & Theobald 2006). Non-ruminant meat, *e.g.* pork, contains proportionally more unsaturated fatty acids, but its composition is still dependent on the fatty acid profile of the animal feed. Overall, lean red meat contains similar proportions of monounsaturated fatty acids (MUFAs) to SFAs, although as illustrated in Table 9, the exact proportions vary depending on the type of meat. The fatty acid profile of meat will also vary depending on the proportions of lean and fat present. For example, lean meat is relatively higher in PUFAs and lower in SFA (*e.g.* less than 2 g of SFAs per 100 g of meat) compared with untrimmed meat. Trimming the fat off meat will affect the proportions of fatty acids as visible fat is higher in SFA, containing around 37 g of SFA per 100 g of meat (Li *et al.* 2005). One study conducted in Switzerland reported that trimming off visible fat from six different meat cuts reduced the fat content by approximately 23.8–59.1% after cooking (Gerber *et al.* 2009).

Saturated fatty acids (SFAs)

The main SFAs present in red meat are palmitic acid (C16:0) and stearic acid (C18:0) [Ministry of Agriculture Fisheries and Food (MAFF) 1998]. There are also minor amounts of myristic acid (C14:0) and lauric acid (C12:0), which are thought to increase blood cholesterol levels more potently than palmitic acid. However, the levels of these cholesterol raising fatty acids are relatively low (see Table 9). Stearic acid has no effect on blood cholesterol levels (Daley *et al.* 2010) and other CVD risk factors such as blood lipids, haemostatic factors and plasma glucose and insulin concentrations (Hunter *et al.* 2010). Although lauric acid (C12:0) increases total serum cholesterol, it decreases the ratio of total cholesterol : high density lipoprotein (HDL) cholesterol because of a preferential increase in HDL cholesterol (Daley *et al.* 2010).

Monounsaturated fatty acids (MUFAs)

Lean beef has similar proportions of SFA and MUFA (Miles & Caswell 2008) (see Table 9). The principal MUFA in meat is oleic acid (C18:1), and typically, between 30–40% of the fat in meat is composed of MUFAs (MAFF 1998).

Polyunsaturated fatty acids (PUFAs)

The predominant PUFAs in meat are the essential fatty acids, linoleic (*n*-6) (LA) and ALNA (*n*-3). When consumed, the body converts ALNA into the long chain *n*-3 PUFAs eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), although the rate of synthesis may not be sufficient to meet requirements. Evidence shows that EPA and DHA may help prevent heart attack, stroke, atherosclerosis and cancer and reduce the risk of inflammatory conditions (Stanner 2005; Stanner *et al.* 2009). These long-chain *n*-3 fatty acids have also been linked to improvement in depression, schizophrenia and dementia (Lunn & Theobald 2006). This is an area of current research.

Although only a small amount of the long-chain *n*-3 PUFAs (EPA and DHA) and *n*-6 PUFAs (arachidonic acid) are found in meat, there are very few rich sources apart from oily fish, and therefore, meat can usefully contribute to intakes of these important fatty acids for those who consume little or no oily fish (Higgs 2000); (Red Meat and Health Expenditure Advisory Committee 2001). In the UK, meat and meat products (including poultry) contribute substantially to intakes, providing 18% of *n*-6 PUFAs and 17% of total *n*-3 PUFAs (Henderson *et al.* 2003a). In recent years, animal husbandry techniques have been modified to produce meat with a more favourable fatty acid profile (see section on Changes in the fat content and fatty acid composition of meat). Scientists are currently developing feeds that will increase the concentrations of the long-chain *n*-3 PUFAs in meat and ultimately enhance human health (Moloney *et al.* 2008).

Factors affecting PUFA content

The proportion of PUFAs in meat is strongly influenced by the feeding regime of the animal (Wood *et al.* 1999). For example, meat from ruminant animals fed on grass throughout the year (as in Northern Europe, Australia and New Zealand) has higher levels of PUFAs. This is because a small proportion of the major fatty acid in grass, ALNA, can escape hydrogenation in the rumen and is absorbed into the tissue lipids (Givens 2005).

Meat also provides long-chain *n*-3 PUFAs as a result of the transformation of dietary ALNA to EPA and DHA as referred to earlier. Oil seeds, such as linseed and rapeseed, are also high in ALNA and therefore the meat of animals reared on feeds containing these seeds will correspondingly contain higher levels of *n*-3 PUFAs.

Trans fatty acids

Trans fatty acids may be produced by the industrial hydrogenation of vegetable oils to produce the semi-solid and solid fats that can be used in food manufacture (*e.g.* traditional margarines and bakery and confectionery products that contain these partially hydrogenated oils) [Scientific Advisory Committee on Nutrition (SACN) 2007a]. In the UK, in recent years, voluntary action by the food industry has considerably reduced *trans* fatty acid levels in foods and thus significantly reduced UK dietary intakes. In particular, *trans* fatty acid levels in vegetable oils used as ingredients in the UK are at a minimum and spreads are now processed differently to be virtually *trans* fatty acid free.

Trans fatty acids also naturally occur at low levels in dairy products and meats from ruminant animals. The proportion of *trans* fatty acids in the UK diet derived from these food products is approximately 40–50%, although this proportion is increasing as the *trans* fatty acids levels in manufactured foods fall (SACN 2007a).

High intakes of *trans* fatty acids have been shown to adversely influence the ratio of low density lipoprotein (LDL)-cholesterol to HDL-cholesterol, a recognised risk factor for CVD. Therefore, dietary guidelines in the UK recommend an upper limit for the consumption of *trans* fatty acids of 2% of dietary energy intake (Department of Health 1994). Data from the most recent NDNS survey show *trans* fatty acids intakes were lower than in previous NDNS and represented 0.8% of food energy (Bates *et al.* 2010). This level of intake for adults was also lower than the re-estimated value calculated in 2007 at 1.0% food energy based on consumption data from the 2000/2001 NDNS and information from the food industry on the current levels of *trans* fats in processed foods (see http://www.food.gov.uk/multimedia/pdfs/publication/ndnsreport0809.pdf) (Bates *et al.* 2010). Although ruminant meat and milk also contribute to *trans* fatty acid intake, these foods are valuable sources of other nutrients, particularly protein, calcium and iron (SACN 2007a). It has therefore been the industrially produced sources such as fat spreads, biscuits, cakes and pastries, crisps and confectionary that have been targeted for reduction.

Meat from ruminant animals also contains another naturally occurring fatty acid – conjugated linoleic acid (CLA). CLA is a collective term used to describe a mixture of positional and geometric isomers of linoleic acid. CLA isomers are intermediates in the biohydrogenation of linoleic acid and the majority of CLA is produced within the peripheral tissues from the rumen-derived fatty acid vaccenic acid. CLA is naturally found in small amounts in products from ruminant animals, *e.g.* lamb, beef, cheese and milk. Evidence from *in vitro* and animal studies indicates that CLA has many potential health benefits on cancer, coronary heart disease (CHD) and diabetes. However, human studies are needed before the effects on human health can be determined (Mulvihill 2001). A focus of current research is to improve the CLA content in beef. One method is to increase the production of vaccenic acid (a *trans* fat found in the fat of ruminents), which is required for the conversion of CLA in the tissue (Moloney *et al.* 2008). Another method is to add oil seeds, vegetable oils and fish oil to the diet to increase CLA content in muscle lipids (Moloney *et al.* 2008).

Feeding practices influence the CLA content of meat. Grass-fed beef has been shown to have higher CLA levels than grain-fed beef (Daley *et al.* 2010). Storage of meat and different methods of cooking meat (*e.g.* frying, grilling, microwaving and baking) have not been found to reduce the CLA content (Mulvihill 2001).

Changes in the fat content and fatty acid composition of meat

Advances in food processing technologies and breeding programmes, as well as modification of animal feeds and modern butchery techniques, have all led to a reduction in the fat content of carcass meat over the past few decades. For example, the fat content of carcass meat in the UK has been reduced by over 30% for pork, 15% for beef and 10% for lamb (Higgs 2000).

Successful modification of the fatty acid profile of some carcass meat has also taken place by alterations in feeding practices. The fatty acid profile of non-ruminant meat is essentially a reflection of that in the diet (Givens & Shingfield 2004). For example, in mono-gastric animals such as pigs, inclusion of vegetable and fish oils in feeds has resulted in significant increases in *n*-3 PUFA, particularly when compared with a traditional cereal-based diet that results in meat fat principally composed of SFAs and MUFAs. In ruminants, the composition of fat is less variable, and it has been more difficult to modify the fatty acid profile of the meat, owing to the influence of gut bacteria (Higgs 2000) that hydrogenate some of the double bonds in MUFA and PUFA in a characteristic manner.

A reduction in fat content has not been seen for all cuts of meat, but this may be because some fat is added back to the meat at retail level (*e.g.* some cuts of topside of beef for roasting) in some countries, such as the UK. There remains further potential to make greater fat savings in the meat sector, for example in meat products, by using fat replacers such as starch (*e.g.* tapioca, potato and maize) or fibre (*e.g.* inulin, wheat fibre (bran) or vegetable, pulse or fruit fibres (*e.g.* pea fibre) (Hughes *et al.* 2010).

Consumers can modify the fat content of meat through preparation methods and cooking. For example, fat gains can occur as a result of the meat sitting in the fat used for frying, and deep frying with breadcrumb coating. Other methods of cooking, such as grilling or dry frying, can result in fat losses for meat products (Clausen & Ovesen 2005). One plausible mechanism to explain why red meat intake may be a

Table 10 Nutrients that could potentially be classified as a 'source' or 'rich source' in red meat according to EU health claims legislation (Regulation (EC) No 1924/2006)*

	Beef	Lamb	Pork	Calf liver
Vitamin A (retinol)	–	–	–	Rich source
Vitamin B_1 (thiamin)	–	–	Rich source	Rich source
Vitamin B_2 (riboflavin)	Source	–	Source	Rich source
Vitamin B_3 (niacin)	Rich source	Rich source	Rich source	Rich source
Vitamin B_6				Rich source
Vitamin B_{12}	Rich source	Rich source	Rich source	Rich source
Vitamin C	–	–	–	Source
Vitamin E	–	–	–	–
Iron	Source	–	–	–
Zinc	Rich source	Rich source	Source	Rich source
Selenium	–	–	Source	Rich source
Potassium	Source	Source	Source	Source
Phosphorus	Source	Source	Source	Rich source

≥15% of the RDA per 100 g = *Source*; ≥30% of the RDA per 100 g = *Rich source*. * Based on raw data.
Note: Recommended daily allowance (RDA) values are estimates of the amount of vitamins and minerals sufficient to meet or more than meet the needs of groups of adults rather than individuals. RDA values are part of EU food law and reflect the variation in opinion across Europe. There is only one figure for each nutrient, derived from figures for adults rather than a range of figures that vary with age, sex and physiological status as exists for UK Reference Nutrient Intakes.

risk factor for colorectal carcinogenesis involves the meat-related mutagens heterocyclic amines, which are formed from amino acids, creatine, creatinine and sugars when food is cooked at high temperatures, *e.g.* frying and barbequing. However, at normal levels of human intake, it is unlikely that they could be the sole causative factor (Williamson *et al.* 2005).

Micronutrient composition of red meat

Red meat contains an array of micronutrients, some in substantial amounts, all of which are required for general health and wellbeing. The nutrients discussed in this section are those for which red meat is a *source* or *rich source* according to European Union (EU) regulations, and the nutrients for which red meat makes an important contribution in the UK diet.

According to EU health claims legislation [Regulation (EC) No 1924/2006] (The European Parliament and the Council of the EU 2007), beef, lamb and pork can be classified as a *source* or *rich source* of several nutrients (see Table 10) Nutrition claims are statements that suggest that a food has particular beneficial nutritional properties because of the nutrients it does or does not contain. In order to make a nutrition claim, the claim in question has to be included in the annex to the Regulation on nutrition and health claims (Regulation (EC) No 1924/2006) and the food may in future have to comply with the nutrient profiles specified by the European Commission.

Vitamins

B vitamins

Red meat contains a number of B vitamins: thiamin (vitamin B_1), riboflavin (vitamin B_2), pantothenic acid, folate, niacin (vitamin B_3) and vitamins B_6 and B_{12} (Chan *et al.* 1995) (see Table 10). The B vitamins work as co-factors in different enzyme systems in the body. In developed countries, intakes of B vitamins meet dietary requirements for most groups. However, as meat- and animal-derived foods (such as milk) and fish are the only foods that naturally provide vitamin B_{12}, some individuals who exclude such foods from their diet are at risk of inadequate intakes. Dietary intakes of vitamin B_{12} are consistently reported as being lower from vegetarian diets and are particularly low in vegan diets (Phillips 2005), thus indicating the important contribution of meat- and animal-derived products to vitamin B_{12} intake. Meat and meat products provide 30% of vitamin B_{12}, 21% of vitamin B_6, 21% of thiamin, and 15% of riboflavin in the UK diet (Henderson, 2003b). Low blood levels of several B vitamins, including vitamin B_{12}, folate and vitamin B_6, have been associated with elevated blood levels of homocysteine, which is a risk factor for CVD and stroke (Stanner 2005).

Vitamin D

The role of vitamin D in the development and maintenance of bones is well established. In addition, by increasing muscle strength, adequate vitamin D status reduces the risk of falling in older adults. More recently, vitamin D deficiency has been associated with CVD, autoimmune disease and cancer (Souberbielle *et al.* 2010). Dietary sources of vitamin D are relatively insignificant compared with the synthesis in the skin from exposure to sunlight or ultraviolet rays because there are not many rich food sources of vitamin D. Nevertheless, housebound people and those who wear concealing clothing are particularly reliant on a dietary supply of vitamin D, and meat usefully contributes to this intake.

Oily fish is the richest source of the vitamin D. However, as only 27% of the UK population are consumers of oily fish and average intakes are relatively low (SACN 2004), other dietary sources of vitamin D such as meat, eggs and fortified products (*e.g.* margarine, reduced fat spreads and some breakfast cereals) are important dietary sources.

Low vitamin D status is prevalent in the UK, particularly among young people and older adults and in ethnic minorities. Rickets, once thought to be a disease of the past, is now re-emerging in some subgroups of the population in the UK (SACN 2007b), mainly those of African-Caribbean and South Asian origin.

The contribution meat and meat products make to dietary intakes of vitamin D among UK adults is 18% in women and 24% in men (Henderson *et al.* 2003b). It is thought that the vitamin in meat is derived from the action of sunlight on the skin of animals or from the animals' feed (MAFF 1995). In particular, the vitamin D metabolite 25-hydroxycholecalciferol [25(OH)D_3] is found in significant quantities in meat and liver and is assumed to have a high biological activity, resulting in better and faster absorption from the diet compared with its parent compound (Groff *et al.* 1995). Furthermore, it has been suggested that components of meat protein may enhance the utilisation of vitamin D in humans, particularly where exposure to sunshine is limited (Dunnigan & Henderson 1997).

Vitamin E

Vitamin E acts as an antioxidant and protects the body's cells against damage. Meat contains a small amount of vitamin E. Consumption of meat and of meat products (red and white meat) contributes to 11% of average daily vitamin E (Henderson *et al.* 2003b). As this is a fat-soluble vitamin, concentrations of vitamin E will be higher in fattier cuts of meat. Vegetable oils are particularly high in vitamin E, and therefore, the recent trend to include oil seeds in animals' diets will have contributed to an increase in the vitamin E content of meat. Vitamin E also delays lipid oxidation and browning of meat, which helps extend shelf life (Wood *et al.* 2003).

Vitamin A

Vitamin A is important for the normal structure and function of the skin and mucous membranes (*e.g.* in lungs), normal growth and development, normal vision and for the immune system. Meat and meat products contribute 28% of vitamin A intake in the UK (Henderson, 2003b). Offal, particularly liver, is a rich source of vitamin A, in the form of retinol (see Table 10). However, the amount present in liver can be variable and indeed very high, and will depend on the age of the animal and the composition of the feed consumed. As liver and liver products may contain a large amount of vitamin A, these should be avoided in pregnancy, owing to the increased risk of birth defects associated with very high intakes of this vitamin. Large amounts of retinol can also cause liver and bone damage. The UK Food Standards Agency (FSA) advises that as a precaution, regular consumers of liver (once a week or more) should not increase their intake of liver or take supplements containing retinol (for example cod liver oil) (FSA 2005).

Minerals

Meat is an important dietary source of bioavailable minerals and trace elements, in particular iron and zinc.

Iron

Iron is essential for the formation of haemoglobin in red blood cells and is also an essential component in many enzymic reactions, for example it is a component of the enzyme system that detoxifies and removes foreign compounds from the body. It also plays an important role in the immune system and is required for normal energy metabolism. Dietary iron exists in two forms, haem and non-haem iron, with haem iron being more readily absorbed and utilised by the body. Most of the iron present in meat is in the haem form. Approximately, 15–35% of haem iron is absorbed in the intestine compared with generally less than 10% of non-haem iron [British Nutrition Foundation (BNF) 1995]. Absorption of dietary iron is regulated tightly, the main regulator being hepcidin, which is made primarily in hepatocytes in response to liver iron levels, inflammation, hypoxia and anaemia (Muñoz 2009). In terms of the overall diet, meat and meat products provide 17% of total dietary iron intake in the UK (Henderson *et al.* 2003b).

Iron deficiency is a worldwide problem, even in developed countries. Data from the UK National Diet and

Nutrition Survey showed that population groups with substantial proportions with iron intakes below the Lower Reference Nutrient Intake (LRNI) were children aged 1.5-to-3.5-years (12–24%), girls aged 11-to-18-years (44–48%) and women aged 19-to-49-years (25–40%) (Henderson *et al.* 2003a). The groups that had the highest proportions with haemoglobin concentrations below World Health Organization (WHO) cut-offs for anaemia were adults aged 65 years and over in institutions (39% of women; 52% of men), free-living adults aged 75 years and over (13–38%) and girls aged 14-to-16-years (15% based on 115 g/l cut-off; 9% based on 110 g/l cut-off).

For adults aged 19-to-64-years, the prevalence of anaemia was higher in women (8%) compared with men (3%) (Henderson *et al.* 2003b). The bioavailability of iron from a food is also extremely important and can be influenced by other dietary factors, especially in the case of non-haem iron. The main enhancers of non-haem iron absorption are ascorbic acid found in fruit and vegetables and meat. Small amounts of meat are recognised to enhance the absorption of non-haem iron from plant foods, although the mechanism for the enhancing effect of meat on non-haem iron absorption is unknown (SACN 2009). The main inhibitors of non-haem iron absorption are calcium, phytates in cereals and legumes, and phenolic compounds found in tea, coffee and other beverages (SACN 2009). UK diets contain a broad range of foods containing iron and various enhancers and inhibitors of iron absorption. Consequently, the bioavailability of dietary iron may have little influence on the iron status of the UK population (SACN draft iron report 2009).

Zinc

Zinc is essential for cell division and therefore for growth and tissue repair. It is also necessary for normal reproductive development, a healthy immune system and healing of wounds. Meat contains substantial amounts of zinc, and beef and lamb can be classified as a *rich source* and pork as a *source* of zinc (see Table 10). Red meat makes a greater contribution to total zinc intake from all foods (32% for men; 27% for women) than to total iron intake (12% for men; 9% for women) (SACN 2009). Modelling by SACN suggests that a reduction in meat intake may have an even greater impact on zinc intake than on iron intake (SACN 2009). Importantly, the zinc contained in meat is present in a highly bioavailable form. There are some concerns regarding low intakes of zinc among some subgroups of the UK population. Infants, children and adolescents, in particular young females, and older adults (aged 65+ years) have low intakes of zinc. Recent data from the UK NDNS showed that 37% of 11-to-14-year-old girls have zinc intakes below the LRNI (SACN 2008). As with dietary iron, a number of factors affect the bioavailability and absorption of dietary zinc, including the composition of the diet. For example, a small amount of lean beef (75 g/d) has been found to enhance iron and zinc utilisation in young women (Johnson & Walker 1992), whereas phytates in cereals and legumes are known to inhibit zinc absorption.

Other minerals

Meat and meat products contain useful amounts of magnesium, copper, cobalt, phosphorus, chromium and nickel. In particular, red meat contains useful amounts of selenium, although the concentration will depend on the diet of the livestock and the soil in which the animal feed was grown. Selenium acts as an antioxidant and is also necessary for the use of iodine in thyroid hormone production and for immune system function. The proportion of selenium that meat and meat products contributes to the diet was estimated to be 32% when last measured in 1997 (MAFF 1997). However, it is recognised that intakes of selenium in the UK and many other European countries have been decreasing over the past few decades as European wheat has replaced the selenium-rich wheat from North America (BNF 2001). Meat, therefore, may now contribute a larger proportion of selenium in the diet (BNF 2001).

Processed meats and meat products

Processed meats and meat products, which contain ingredients other than meat, are likely to have lower micronutrient contents per 100 g but may provide other nutrients not usually found in meat (*e.g.* carbohydrate and fibre). Also, the addition of some ingredients (*e.g.* soya, fibre) to add functional properties to meat products can also offer potential benefits for health (Fernández-Ginés *et al.* 2005). It should be noted that the composition of meat classed as processed meats vary widely and the term includes different products in different countries. Overall, meat products and processed meats are more likely to have a higher content of sodium than lean meat. Sodium is added to meat products to enhance and modify the flavour, the physical properties and sensory attributes of the food and to contribute to the preservation of the product (Matthews & Strong 2005). Owing to the adverse health effects associated with a high intake of sodium, there has been a considerable amount of work within the UK over the last few years to reduce the amount of salt in processed meat products, in particular focusing on products that contain the highest levels of sodium (FSA 2010). This work is ongoing.

Summary

Meat and meat products can make an important contribution to nutrient intakes. They provide a number of essential nutrients, including protein, long-chain *n*-3 fatty acids, iron, zinc, selenium, vitamin D and vitamins B_3 and B_{12}. In particular, some of these nutrients are more bioavailable in meat than alternative food sources, and their intake is also recognised to be below recommendations for some population subgroups.

Health aspects of red meat

Obesity

Overweight and obesity is an increasing public health problem worldwide, affecting people of all ages and socio-economic groups. Globally, the prevalence of overweight and obesity is increasing in both adults and children and it is no longer restricted to affluent countries (WHO 2000). Obesity is associated with an increased risk of chronic diseases including CVD, type 2 diabetes and some types of cancer (Buttriss 2005).

Obesity is a complex disorder with a diverse range of causal factors. Most cases of obesity arise as the result of an adverse environment working on a susceptible genotype. Susceptibility may be mediated through a wide range of metabolic (*e.g.* control of fuel selection) and behavioural (*e.g.* binge eating) traits. One incontrovertible fact, however, is that for an individual to become obese, energy intake must be higher than energy expenditure for an extended period of time. This means that either more energy than needed is consumed and/or that too little energy is used by the body because of a lack of physical activity. In general, weight gain seems to be a result of a combination of both increased energy intake and decreased energy expenditure. The wide range of aetiological factors makes obesity both a complex and challenging disorder (Butland *et al.* 2007).

There has been discussion about the role that meat and meat products play in the development of overweight and obesity. Studies have shown that people eating a vegetarian diet tend to have lower bodyweight compared with non-vegetarians. A review of the evidence from 31 observational studies reported bodyweight to be lower in vegetarians compared with non-vegetarians at a range of 4–20%, the findings being significant in 18 studies (Berkow & Barnard 2006). Also, a lower prevalence of obesity was reported, ranging from 0–6% in vegetarians and from 5–45% in non-vegetarians. Lower bodyweight in vegetarians has been reported for both men and women (Berkow & Barnard 2006). It is difficult to establish the factors contributing to these differences in bodyweight. People who eat a vegetarian diet often tend to be more health conscious than non-vegetarians, which means they may lead an overall healthier lifestyle, including more physical activity and more health-conscious dietary choices (Phillips 2005). Vegetarians have been found to have somewhat lower energy intakes compared with non-vegetarians. The reduced energy intake appears to be mainly caused by higher fibre intake bulking out the diet and to a lesser extent, a higher proportion of energy from carbohydrate and a lower fat intake (Berkow & Barnard 2006). Meat makes a relatively high contribution to dietary fat and saturated fat intakes (see section on nutrient composition of red meat); a recent UK dietary survey indicated that meat and meat products contribute around 26% of total fat intake, whereas the contribution to total energy intake is, on average, 18% (Bates *et al.* 2010). However, cutting out meat from the diet does not mean automatically reducing total fat intake as a vegetarian diet can also be high in energy and fat if large amounts of energy dense foods, such as cheese, are consumed and a diet containing lean meat can be low in energy. As discussed earlier, the fat content of lean meat can be less than 5% (*i.e.* '5 g/100 g' classified as 'low-fat'), whereas foods typically consumed in a vegetarian diet, such as cheese, can be relatively high in fat (*e.g.* approximately 47 g/100 g in cream cheese and 35 g/100 g in cheddar cheese). As dietary fat comes from a variety of sources, reducing the fat content of the diet *as a whole* (target recommendation 35% of food energy) is a key feature of health policy activities. In addition to choosing leaner cuts of meat, consumers may also limit their fat and saturated fat intake from meat products by choosing reduced fat meat products.

There is evidence that diets restricted in carbohydrates and with a stronger emphasis on protein intake can aid weight loss. A high intake of meat and other animal products is a feature of this type of diet. A meta-analysis of five trials including 447 individuals showed that after 6 months, individuals assigned to low-carbohydrate (LC) diets (not restricted in fat, protein or energy) had lost more weight than individuals randomised to low-fat diets that were restricted in energy, although this difference was no longer obvious after 12 months, which may be explained by a greater weight gain in the carbohydrate restricted groups compared with the fat-restricted groups (Nordmann *et al.* 2006). Suggested reasons for initially increased weight loss with higher protein diets include increased satiating properties of protein (which may explain decreased *ad libitum* food intake) (Benelam 2009) (see section on meat and satiety) and effects on thermogenesis, body composition and decreased energy efficiency (Westerterp-Plantenga *et al.* 2007). However, even though LC, high-protein (HP) diets may support weight loss in the short term, a diet that is very restricted in carbohydrate-containing foods is not desirable over a longer period of time. Cutting out a whole food group (*e.g.* starchy foods) can result in low intakes of some important nutrients. An American study in overweight or obese pre-menopausal women, looking at four popular diets, found that when participants followed a diet with very restricted carbohydrate intake (less than 20 g/d), intakes of six micronutrients (vitamin B_1, folate, vitamins C and E, iron and magnesium) were below the Estimated Average Requirements (EAR) in at least 25% of women. In comparison, a diet that was very low in fat (<10% of energy) resulted in intakes of five micronutrients (magnesium, zinc, vitamins A, B_{12} and E) below the EAR in at least 25% of women. An energy-restricted diet that met the healthy eating recommendations of the WHO resulted in intakes of four nutrients (vitamins A, C and E and magnesium) below the EAR in at least 25% of women. The most adequate vitamin and mineral intakes were found when women followed an energy-restricted diet that provided a moderately increased proportion of protein (30% of energy) and a moderately decreased proportion of carbohydrate (40% of energy), with fat intakes being limited to 30% of total energy intake. This resulted in the intake of only two vitamins to be below the EAR in at least 25% of the women (vitamin E and magnesium) (Gardner *et al.* 2010). As the people following the latter diet had decreased their total energy intake, it is likely that their absolute protein intake (g/d) had actually not changed but that the decrease in energy intake was caused by decreased intake of carbohydrate and fat. In addition to insufficient micronutrient intakes, results from three recently published long-term cohort studies found that LC diets increased all-cause mortality (Fung *et al.* 2010; Sjogren *et al.* 2010), although results from two cohorts only found an increased mortality for LC diets based on animal sources but not for vegetable-based LC diets in both men and women (Fung *et al.* 2010).

Summary

In summary, people following a vegetarian diet tend to weigh less than non-vegetarians. However, this may be caused by various dietary and lifestyle factors as vegetarians generally adopt healthier behaviours. While protein from various sources, including meat, may promote healthy weight maintenance and weight loss as part of a reduced energy diet because of various factors, including its satiating effects, diets that are very low in carbohydrate are not recommended. Therefore, people who choose to increase their intake of *lean* meat in order to lose weight are advised to also include starchy carbohydrates in their diet.

Meat and satiety

Satiety is defined as the sensation of fullness that persists after eating until hunger returns, while satiation is the process that leads us to stop eating, ending an eating occasion (Benelam 2009). In recent years, there has been great interest in the manipulation of satiation and satiety in order to control energy intake and thus bodyweight. Research has been conducted on various aspects of the diet and satiety, and the role of protein in modulating appetite and energy intake has been the subject of a number of studies. In addition, some studies have investigated the effect of different protein sources on satiety, including the role of red meat.

A review investigating the effect of protein on satiety, energy intake and bodyweight looked at both short- and long-term effects of HP foods and diets. In the short-term (1–24 hours), 11 of the 14 studies included found that satiety was significantly increased in the higher protein vs. the lower protein condition. The longer term studies (ranging from 2 weeks to 1 year) included in the review investigated the effect of HP diets on weight loss. Where the diets were isocaloric, few studies showed that HP diets increased weight loss. However, where diets were consumed *ad libitum*, all five studies included recorded significantly higher weight losses in the HP condition (Halton & Hu 2004). Satiety ratings were not taken in these studies, but it can be speculated that the spontaneous reduction in energy intake seen in the *ad libitum* HP diets could be caused by increased feelings of satiety. A subsequent review also found that overall, protein tends to be more satiating than the other macronutrients, both at the level of a single eating occasion and over days and weeks (Paddon-Jones *et al.* 2008).

The energy density of foods appears to have a significant effect on their satiating potential (Benelam

2009). It is important to note that in a study where macronutrient content of test meals was varied while energy density remained constant, no differences in satiety or subsequent energy intake were observed (Raben *et al.* 2003). Thus, it may be that energy density of foods has a greater effect on satiety than their content of macronutrients.

A feature of some popular HP weight loss diets is that they are also very low in carbohydrate, resulting in ketogenesis (associated with a smell of acetone on the breath). This is the state where ketone bodies are generated from fat stores in response to reduced glucose availability when carbohydrate intake is very low. An intervention study with obese male subjects by Johnstone *et al.* (2008) was designed to test whether HP, LC, ketogenic diets were more satiating and caused greater weight loss than HP, medium-carbohydrate (MC), non-ketogenic diets. The study compared a HP, LC, ketogenic diet (30% energy from protein, 4% energy from carbohydrate) with a HP, MC, non-ketogenic diet (30% energy from protein, 35% energy from carbohydrate). Hunger and energy intakes were found to be significantly lower on the LC ketogenic diet than on the MC non-ketogenic diet and subjects lost significantly more weight on the LC ketogenic diet. It is not possible to conclude whether it was the ketosis on the LC diet or the difference in fat content between the two diets that caused the reduction in hunger and energy intake, and the relevance of ketosis to satiety may warrant further investigation (Johnstone *et al.* 2008). It should be noted, however, that there are safety concerns about very HP diets (including those with very low carbohydrate content) and caution should be exercised in promoting them (Eisentstein *et al.* 2002).

Some studies have looked at the effect of meat protein, in particular, on satiety. Two studies have investigated the effect of protein from fish vs. that from beef on satiety, and in both cases, satiety ratings were higher after consuming fish than after consuming beef (Uhe *et al.* 1992); (Borzoei *et al.* 2006). However, it should be noted that both studies were relatively small (six and 23 subjects, respectively) and included only lean men. One study also investigated the satiating properties of different beef preparations: roast beef, boiled beef and canned beef in jelly, each of which differed in their protein content and energy density. The results of the study suggested that it was the energy density of the preparations rather than their protein content that had the greatest impact on subsequent satiety and energy intake (Berti *et al.* 2008).

Summary

Overall, there is evidence that higher protein meals and diets may be more satiating than those with lower protein content and may help to control bodyweight. It should be noted that this effect has not been observed when the energy density of test foods is controlled and the energy density of a food or diet may be more important in determining its satiating effect than its protein content. Current evidence regarding the effect of the source of protein does not suggest that this has a significant effect on satiety in the context of a normal diet.

Cardiovascular disease (CVD)

CVD includes all the diseases that affect the heart and circulation such as CHD, stroke and heart attack. CVD is the main cause of death worldwide. CHD death rates have been steadily decreasing since the 1980s. However; rates in the UK remain among the highest in Europe. Recent estimates showed that CHD was responsible for more than 190 000 deaths in the UK in 2007, which is almost a third of all deaths (British Heart Foundation 2010). The aetiology of CVD is complex, but diet, alongside other lifestyle factors (*e.g.* physical activity and smoking cessation), influences many important risk factors such as hypertension, obesity, diabetes, high blood cholesterol and high blood triglyceride concentrations (Stanner 2005).

Despite the presence of a number of potentially protective nutrients (*e.g.* selenium, *n*-3 fatty acids, B vitamins), meat and meat products have often being thought of as a contributor to increased risk of heart disease because of their relatively high contribution to fat intake and perceived high content of SFAs and sodium in the case of processed meats. Recent NDNS data show that meat and meat products contribute approximately one quarter (23%) of SFAs and 28% of sodium intakes for young adolescents (11-to-18-years) and adults (Bates *et al.* 2010).

Several prospective studies have demonstrated a positive association between meat intake and CVD (Kelemen *et al.* 2005; Steffen *et al.* 2005; Kontogianni *et al.* 2008; Ashaye *et al.* 2010). The type of study design used to investigate this association varies widely and includes case control, cross-sectional and cohort studies. The outcomes measured also vary from study to study, making it challenging to compare and interpret their results. Additionally, meat intakes of subjects are based typically on retrospective data collected by means of a semi-quantitative FFQ, and hence, under- or over-reporting may be likely and could impact on study results.

In the Nurses' Health study, a high red meat intake was associated with an elevated risk of CHD (Bernstein *et al.* 2010). Study investigators found that higher intakes of red meat, red meat excluding processed meats and high-fat dairy products, were significantly associated with increased risk of CHD in men and women at 26-year follow-up, independently of established dietary and non-dietary risk factors. Another randomised case control study of 846 patients with a first symptom of CHD and more than 1000 controls found that patients consumed higher quantities of meat compared with controls (6.5 ± 2.9 vs. 4.9 ± 2.1 portions per month, $P < 0.001$) (Kontogianni *et al.* 2008). Statistical analysis of their nutritional habits revealed that red meat consumption was strongly associated with a 52% increased odds of acute coronary syndrome [95% confidence interval (CI) 1.47–1.58]. Conversely, white meat consumption was associated with only 18% likelihood of having cardiac events (95% CI 1.11–1.26). The study investigators adjusted for confounders such as body mass index (BMI), smoking and physical activity level. When low meat intake (up to four portions a month) and low white meat intake (up to eight portions a month) were compared with no meat intake, low meat intake did not increase the likelihood of acute cardiac events ($P > 0.05$). The Physicians' Health Study observed a positive and graded association between red meat consumption and heart failure (Ashaye *et al.* 2010). The study investigators compared the lowest and highest quantities of red meat intake and found the hazard ratios [HRs, (95% CI)] for heart failure were 1.0, 1.02 (0.85–1.22), 1.08 (0.90–1.30), 1.17 (0.97–1.41) and 1.24 (1.03–1.48), respectively, after adjustment for confounding factors. Many studies have defined red and processed meats poorly owing to the retrospective methods used to assess meat intakes and there is little information about the effects of different types of meat, such as lean vs. untrimmed meat or processed vs. unprocessed meat (see section on Evidence base).

Dietary patterns and CVD

Many studies have investigated dietary patterns rather than meat consumption *per se* in relation to risk of CVD (Harriss *et al.* 2007; Teixeira Rde *et al.* 2007; Heidemann *et al.* 2008; Panagiotakos *et al.* 2009; Guallar-Castillon *et al.* 2010), owing to the difficulty in accurately assessing intakes of foods or nutrients and the fact that individuals choose foods and combinations of foods rather than isolated nutrients. For these reasons, scientists increasingly use dietary pattern analysis to assess risk of CVD.

Prospective studies have compared mortality of CVD between vegetarians and meat eaters. Many of these studies have demonstrated that individuals who consume a long-term vegetarian diet tend to have lower levels of cardiovascular risk factors (*e.g.* blood pressure, fasting plasma glucose and total cholesterol, etc.) (Key *et al.* 1998; Key *et al.* 1999; Szeto *et al.* 2004; Teixeira Rde *et al.* 2007). Subgroup analysis of subjects from the EPIC-Oxford study showed that vegetarians, especially vegans, have a lower prevalence of hypertension and lower systolic and diastolic blood pressures than meat eaters (Appleby *et al.* 2002). The age-adjusted prevalence of hypertension ranged from 15% in male meat eaters to 5.8% in male vegans, and from 12.1% in female meat eaters to 7.7% in female vegans. Teixeira Rde *et al.* found that blood pressure, fasting plasma glucose, total cholesterol, LDL cholesterol and triglycerides were lower among vegetarians ($P < 0.001$) compared with meat eaters (Teixeira Rde *et al.* 2007). Overall, vegetarians had lower cardiovascular risk ($P < 0.001$). Another study on a pooled analysis of vegetarians and meat eaters from five prospective studies found that vegetarians had a 24% reduction in mortality from ischaemic heart disease than meat eaters (death rate ratio: 0.76; 95% CI: 0.62, 0.94; $P < 0.001$), and this reduction was confined to those individuals who followed a vegetarian diet for more than 5 years (Key *et al.* 1999). While the findings from these studies are, at first sight, convincing, there are many aspects of a vegetarian dietary pattern apart from not eating meat that may contribute to a reduced risk. For example, meat can be replaced with other foods such as pulses, soy protein, nuts and vegetables that may have beneficial cardiovascular effects. Vegetarians are also generally more physically active, have a lower body mass index and are less likely to smoke (Phillips 2005). It is therefore difficult for studies of this type to assess the true effects of meat *per se* even when study investigators have controlled for a wide range of potential confounders.

Studies have also investigated the relationship of other specific dietary patterns with CVD risk. Both the US Health Professionals' Follow-up Study (Hu *et al.* 2000) and the Nurses' Health Study (Fung *et al.* 2001) found that the risk of CHD was 40% higher in individuals in the highest quintile of a dietary pattern derived from a FFQ, termed the 'Western pattern' (high intakes of red meat, processed meat, refined grains, sweets and desserts, French fries and high-fat dairy products) than those in the lowest quintile, which suggest that a Western dietary pattern increases risk of CHD in this cohort. In a study of 72 113 women (Heidemann *et al.* 2008), two major dietary patterns were identified at baseline: high 'prudent' patterns scores (high intakes of vegetables, fruit, legumes, fish, poultry and wholegrains) and high 'Western' pattern scores (high intakes of red meat, processed meat, refined grains, French fries, sweets and desserts). At follow-up (18 years later), the prudent diet was associated with a 28% lower risk of mortality from CVD (95% CI, 13 to 40) when the highest quintile was compared with the lowest quintile. In contrast, the Western pattern was associated with a 22% higher risk of mortality from CVD (95% CI, 1 to 48). The 'prudent' dietary pattern has been shown to improve risk factors for CVD across the globe in Dutch, Italian, Greek and Australian populations (van Dam *et al.* 2003; Harriss *et al.* 2007; Centritto *et al.* 2009; Panagiotakos *et al.* 2009). Among 19 750 randomly selected men and women in the Monitoring Project on Risk Factors and Chronic Diseases in the Netherlands (MORGEN Study), three major food consumption patterns were identified: the cosmopolitan pattern (greater intakes of fried vegetables, salad, rice, chicken, fish and wine), the traditional pattern (greater intakes of red meat and potatoes, and lesser intakes of low-fat dairy and fruit) and the refined foods pattern (greater intakes of French fries, high-sugar beverages and white bread and lesser intakes of wholegrain bread and boiled vegetables). Independent of confounding factors, the traditional pattern score was positively associated with systolic blood pressure [men: $\beta = 0.65$ (95% CI: –0.09, 1.39], women: $\beta = 1.25$ (95% CI: 0.41, 2.09) and HDL-cholesterol concentrations [men: $\beta = 0.03$ (95% CI: 0.02, 0.05)]; women: $\beta = 0.02$ (955 CI: 0.00, 0.04). The cosmopolitan pattern score was positively associated with HDL-cholesterol (men: $\beta = 0.04$ 95% CI: 0.03, 0.06); women: $\beta = 0.00$ (95% CI; –0.02, 0.02) and inversely associated with systolic blood pressure [men: $\beta = -1.44$ (95% CI: –2.12, –0.75)], women: $\beta = -1.92$ (95% CI –2.69, –1.16). The refined foods pattern score was associated with higher total cholesterol, higher systolic blood pressure (in women) and lower HDL-cholesterol and higher plasma glucose levels. However, some studies are not in agreement with the association between a 'Western' dietary pattern and increased risk for developing CVD (Harriss *et al.* 2007; Guallar-Castillon *et al.* 2010). Harriss and colleagues found that the frequent intake of meat was not associated with CVD or ischaemic heart disease (Harriss *et al.* 2007). Similarly, the EPIC-Spain cohort study found that the Westernised pattern was not associated with coronary risk, although the dietary pattern characterised in this study also included foods such as wine, legumes, olive oil and oily fish, among others (Guallar-Castillon *et al.* 2010).

A few randomised-controlled studies have also investigated the effects of dietary patterns that include specifically lean meat on CVD risk factors. For example, 26 men were randomised in an incomplete block design to receive two of three diets – a high-fat diet, a low-fat lacto-ovo-vegetarian diet and a low-fat diet containing lean meat (Kestin *et al.* 1989). The primary aim of this study was to determine the extent to which consumption of lean meat might lower those benefits of a vegetarian diet that relate to cardiovascular risk. Compared with the high-fat diet, both prudent (low fat) diets significantly lowered total and LDL-cholesterol, as well as blood pressure (but significantly increased serum triglycerides – a recognised effect of low-fat diets) (Stanner 2005). The lacto-ovo-vegetarian diet had a significantly greater cholesterol lowering effect (10% reduction in lacto-ovo-vegetarian diet group vs. 5% reduction in lean meat diet group), but blood pressure reductions were similar. The authors concluded that the partial substitution of lean meat for plant protein in a fat-modified diet did not negate the overall cardiovascular risk lowering of the low-fat lacto-ovo-vegetarian diet.

Intakes of red and processed meat and CVD

While studies investigating dietary patterns are useful in assessing multiple risks factors, some studies that analyse dietary patterns do not differentiate between red meat and processed meats (Kelemen *et al.* 2005), and this makes it difficult to isolate the potential effects of red meat alone. In addition, the composition of unprocessed and processed meat differs (see section on processed meats and meat products) such as sodium levels and use of additives (*e.g.* nitrates) that could produce differing effects on cardiometabolic risk. However, some epidemiological studies have investigated the relation of red and processed meat separately for CHD (Whiteman *et al.* 1999; Burke *et al.* 2007; Sinha *et al.* 2009).

A large cohort study of Americans was prospectively followed to investigate meat intakes as risk factors for total mortality and diseases including CVD (Sinha *et al.* 2009). During 10 years of follow-up, both red and processed meats were associated with an increased risk of CVD mortality for men and women in the highest (HR 1.27, 95% CI 1.20–1.35; HR 1.50, 95% CI 1.37–1.65, respectively) quintile compared with the lowest quintile of red and processed meat (HR 1.09, 95% CI

1.03–1.15; HR 1.38, 95% CI 1.26–1.51, respectively) when controlling for known confounders. Another study in Australia among 256 aborigine women and 258 aborigine men found that among other factors, consumption of processed meat greater than once a week (HR 2.21, 95% CI: 1.05, 4.63) was a significant predictor of CHD endpoints (death or first admission to hospital) (Burke *et al.* 2007). No significant associations were observed between CHD outcomes and consumption of traditional sources of meat in this cohort. A recent British cohort study found that consumption of red or processed meat as assessed between 1989 and 1999 did not predict an increased risk of CVD as measured by cholesterol levels or blood pressure (Wagemakers *et al.* 2009). However, red and processed meat intakes in 1989, both separately and combined, had a significant positive association with waist circumference in 1999. Overall, studies investigating the relationships between red and processed meat intakes separately suggest inconsistent findings with studies reporting different effects of both processed and unprocessed (red) meats on CHD risk. Further studies are needed to understand better the effects of both processed and unprocessed meats on CHD risk.

Meat and the metabolic syndrome

Metabolic syndrome is characterised by a cluster of CVD risk factors (*e.g.* central obesity, elevated blood pressure, blood lipids, blood sugar and insulin resistance) that increase the risk of developing various chronic diseases including CVD. A small number of cross-sectional and prospective studies have investigated the relationship between red meat and the risk of metabolic syndrome. Typically, the studies have explored dietary patterns or estimated red meat consumption and risk of metabolic syndrome. Some cross-sectional studies have reported a positive relationship between red meat and risk of metabolic syndrome (Alvarez Leon *et al.* 2006; Azadbakht & Esmaillzadeh 2009), although others have found no association after adjusting for confounding factors (Damiao *et al.* 2006). A recent prospective study in participants at high CVD risk found higher red meat consumption to be associated with greater risk of metabolic syndrome (Babio *et al.* 2010). At 1-year follow-up, subjects in the highest quartile of intake (104 g of red meat/d) had twice the risk of metabolic syndrome (OR < 2.2; 95% CI, 1.3–3.7; *P* for linear trend = 0.034) compared with those subjects in the lowest quartile (21 g of red meat/day). However, intervention studies are needed to explore this association further.

Components of red meat and potential links with CVD

The nutritional and chemical composition of unprocessed meats (fatty acids, protein, iron, B vitamins and selenium) and processed meats (sodium and additives, *e.g.* nitrates and their by-products) have been proposed as factors that may affect CVD risk.

Fatty acids and associated risk of CVD

When examining the effects of fat from meat on CVD risk, it is important to consider the fatty acid profile of red meat in the context of the diet rather than total fat content of meat alone.

Dietary fats are regarded as having an important influence on CVD because of their effects on blood cholesterol levels. A high level of LDL-cholesterol combined with a low level of HDL-cholesterol increases the risk of atherosclerosis, while a low level of LDL and high level of HDL-cholesterol reduces the risk. Laboratory studies have also shown that blood cholesterol can be influenced by the balance of different types of fatty acids in the diet; blood levels of LDL-cholesterol are lowered when some SFAs (*e.g.* myristic and palmitic acids) are replaced by MUFAs, PUFAs or carbohydrate. Fatty acids may also affect CVD risk via other mechanisms. For example, SFAs raise platelet activity and thus increase the tendency of blood to clot, whereas PUFAs have the opposite effect; SFA intake may also be associated with reduced insulin sensitivity, a key factor in the development of the metabolic syndrome (Mozaffarian & Rimm 2006). Dietary recommendations to lower CVD risk have therefore emphasised the need for a reduction in total fat and in particular, the amount of SFAs in the diet.

Although the cholesterol-lowering effect of PUFAs is greater than that of MUFAs, when substituted for SFAs, MUFAs do not reduce the protective HDL-cholesterol to the same extent. MUFAs compared with PUFAs are also less likely to be oxidised both in foods during cooking and processing and in the body (oxidised lipids are implicated in protein and DNA damage and in CVD risk). Moreover, low-fat, high-carbohydrate diets have been shown to increase plasma triglycerides and decrease beneficial HDL-cholesterol levels (Stanner 2005). Intervention studies using high MUFA diets have also shown potential beneficial effects on haemostasis, inflammation and coagulation and favourable outcomes on blood lipids (*e.g.* lowering LDL-cholesterol) (Kelly & Stanner 2003; Allman-Farinelli *et al.* 2005). This is likely to be a contributing factor in the ability of Mediterranean-style diets, which are rich in MUFAs, to protect against CVD. There is therefore considerable support within the scientific community for the idea that a moderate fat diet that is high in unsaturated fatty acids may promote a better lipid profile than a low-fat, high-carbohydrate diet and offer a more effective approach for reducing CVD risk, particularly for people with type 2 diabetes. In addition, such diets are likely to be easier to adhere to for those on weight-reducing diets (Stanner 2005).

The positive relationship between SFAs, blood cholesterol and CHD risk suggests that regular intake of foods high in SFAs and cholesterol may increase risk of CHD. Meat's contribution to SFA intake varies widely between countries (see section on nutrient composition of red meat). Overall, red meat contains similar proportions of MUFAs and SFAs, although the exact proportions of the fatty acids vary depending on its fat content. Lean meat is relatively higher in PUFAs, and lower in SFAs and total fat than untrimmed meat (SACN 2003) (all lean meats contain less than 2 g of SFAs per 100 g, while the visible fat of meat contains over 37 g/100 g). Furthermore, one of the main SFAs in red meat is stearic acid, which has neutral effects on blood cholesterol levels (Yokoyama *et al.* 2007; Manger *et al.* 2010), although red meat also contains smaller amounts of the cholesterol-raising fatty acids (see section on SFAs).

Dietary intervention studies have, however, suggested that while untrimmed meat is cholesterol-raising, this is not true of diets containing fat-trimmed lean meat. In general, studies have demonstrated lean red meat to have similar effects on total, LDL- and HDL-cholesterol or triglyceride levels as white meat or soybean products (Forstermann 2008). Intervention studies investigating the effects of diets containing lean red meat have lowered blood cholesterol compared with diets with a relatively high carbohydrate content in both healthy and hypertensive subjects (O'Dea *et al.* 1990; Hodgson *et al.* 2007; Nowson *et al.* 2009). For example, the Dietary Approaches to Stop Hypertension (DASH) study has demonstrated the potential benefit of inclusion of lean red meat as part of a low-sodium diet. Post-menopausal women with high/normal blood pressure were randomised to a low-sodium DASH-type diet containing 100 g cooked lean red meat [the vitality diet (VD)] for 14 weeks and compared with women receiving a higher acid load 'reference healthy diet' (RHD) (a high-carbohydrate, low-fat diet). At the end of the study, systolic blood pressure fell in the VD group by 5.6 ± 1.3 mm Hg compared with a fall of 2.7 ± 1.0 mm Hg in the RHD (group difference, $P = 0.08$) (Nowson *et al.* 2009). In fact, when the study investigators assessed those subjects taking anti-hypertensive medications, the VD group had a significant fall of 6.5 ± 2.5 mm Hg in systolic blood pressure ($P = 0.02$) and 4.6 ± 1.4 mm Hg in diastolic blood pressure ($P = 0.005$) after the intervention, and their blood pressure was lower than that of the RHD group for the duration of the study ($P < 0.05$). Earlier work has shown that a low-fat diet containing lean red meat can reduce LDL-cholesterol in hypercholesterolaemic subjects. Collectively, these studies suggest that lean meat combined with a healthy diet may, in fact, improve markers of CVD in healthy and at risk groups.

Meat from ruminant animals naturally contains small amounts of *trans* fatty acids as well as CLA (see section on *Trans* fatty acids). *Trans* fatty acids are recognised to have a more potent effect on blood cholesterol than SFAs by raising levels of LDL-cholesterol and lipoprotein (a) and decreasing HDL-cholesterol levels. Increased intakes of *trans* fatty acids have previously been shown to increase the risk of CHD in prospective cohort studies (Appel *et al.* 2005; Preis *et al.* 2010).

In the UK, current intakes are low and well below the population recommendation of no more than 2% of energy with mean intakes of 0.8% of food energy (Bates *et al.* 2010), while intakes in North America are higher. Vaccenic acid (a positional geometric isomer of oleic acid) is the predominant *trans* isomer in ruminant fats. Although some epidemiological and a limited number of human dietary studies have suggested that there is no association between intakes of this *trans* isomer and CHD risk factors (Lee *et al.* 2005; Kirk *et al.* 2008), in its review of the evidence, SACN concluded that there is inadequate data to demonstrate that *trans* fatty acids from different dietary sources have differential effects on CHD risk or lipoprotein profiles (SACN 2007a).

Meat, primarily lean meat, also contains the *n*-3 PUFA ALNA as well as small amounts of the long-chain *n*-3 PUFA EPA, DPA and DHA. Despite being present at low levels, particularly when compared with oil-rich fish, intake of these *n*-3 PUFA from red meat, mainly in the form of ALNA, is significant for the average consumer (see section on PUFAs). Randomised Controlled Trials (RCTs) and cohort studies provide strong evidence that consumption of oily fish and *n*-3 long-chain PUFAs, in particular EPA and DHA, are associated with a reduction in deaths after cardiac events in healthy individuals and in patients with heart disease (Mozaffarian & Rimm 2006). Prospective studies show promising effects on CVD risk factors. For example, in a large study of hypercholesterolaemic patients, Yokoyama and colleagues showed that those receiving a supplement of 1800 mg of EPA together with a statin for 5 years experienced fewer major coronary events (*e.g.* sudden heart attack and fatal and non-fatal myocardial infarction) than those receiving a statin alone (Yokoyama *et al.* 2007). In contrast, among subjects from the Western Norway B vitamin Intervention Trial with coronary artery disease consuming high amounts of fish and supplementary *n*-3 long-chain PUFAs, higher intakes were not found to be associated with a reduced risk of coronary events or mortality (Manger *et al.* 2010).

Sodium, nitrates and blood pressure

The relative risk (RR) of both CHD and stroke increases as blood pressure rises. It is now well established that sodium, and consequently, salt intake, is an important determinant of high blood pressure in the UK population (SACN 2003). In 2003, the SACN reviewed the evidence linking high salt intakes to high blood pressure and concluded that reducing the average salt intake in Britain would confer significant public health benefits by contributing to a reduction in CVD burden. SACN recommend that average salt intakes for adults should not exceed 6 g (2.4 g/100 mmol sodium) per day. Following this review, the FSA and Department of Health set a target to reduce salt intakes in the UK population to meet the SACN target by 2010 (Wyness *et al.* in press). Some progress has been made towards the target; average intake in adults has fallen from 9.5 g/d in 2000/2001 (Henderson *et al.* 2003b) to 8.6 g/d in 2008 (Bates *et al.* 2010). Carcass meat contains very little sodium naturally, but salt is added to meat products for a variety of technical reasons and as such, processed meat products make a substantial contribution to total salt intake. Meat and meat products contribute to 31% (men) and 25% (women) of average daily salt intakes (Bates *et al.* 2010). Salt usage can be an important factor in the safety and quality of certain foods, for example meat products. In these foods, salt reduces the amount of 'available' water present in the food required for microbial growth, causing micro-organisms to grow more slowly or not at all. Consequently, when reducing the level of salt in these foods, manufacturers need to consider potential effects on microbiological safety (Wyness *et al.* in press).

It has been suggested that the higher sodium and nitrate preservative levels in processed meats could potentially contribute to increased CVD (Micha *et al.* 2010). This mechanism may explain the disparity in findings in relation to dietary patterns and risk of CHD referred to earlier and the need to separate processed meats from unprocessed meats in analyses. Experimentally, nitrates have been shown to promote atherosclerosis and vascular dysfunction (Forstermann 2008). However, further work is needed in humans to determine whether nitrates or their metabolites promote atherosclerosis in humans.

Protein and CVD

A limited amount of epidemiological data is available investigating protein intake and CHD risk, and to date, findings are inconsistent. Iso and colleagues found that intake of small amounts of animal protein was associated with a significantly increased risk of haemorrhagic stroke (Iso *et al.* 2001). Bernstein and colleagues followed 84 136 women in the Nurses' Health Study for 26 years, documenting 2210 incident non-fatal infarctions and 952 deaths from CHD (Bernstein *et al.* 2010). When the investigators examined individual protein sources and risk of CHD, higher intakes of red and processed meats combined and red meat excluding processed meat were associated with increased risk; in contrast, higher intakes of fish, nuts and beans were associated with decreased risk. In an earlier analyses of the Nurses' Health study with 14 years of follow-up, the study investigators compared the highest quintile of protein intake (median 24% energy) with the lowest quintile (median 15% energy) and found a RR of CHD of 0.74 (95% CI: 0.59–0.94) after controlling for age, smoking, total energy intake, percentage of energy from specific types of fat and other coronary risk factors (Hu *et al.* 2000). Conversely, no such trend was found when US males were followed prospectively to examine the association between dietary protein and risk of ischaemic heart disease in the Health Professional Follow-Up Study (Preis *et al.* 2010).

Findings for stroke are also inconsistent. A few prospective studies in Japanese populations have suggested that protein, in particular protein from animal sources, may increase stroke risk (Sauvaget *et al.* 2004). However, Preis *et al.* examined the relation between dietary protein and risk of stroke in 43 960 men who participated in the US Health Professional Follow-Up Study and concluded that there was no significant association between total, animal or vegetable protein and risk of stroke in this population (Preis *et al.* 2010). Collectively, given the observational nature of these studies, the likelihood of residual and unmeasured confounding can not be ruled out, and thus, the results must be interpreted with caution.

A limited number of clinical trials have shown that protein may affect blood pressure (Appel *et al.* 2005). The Omniheart study showed that in the context of a healthy diet, partial substitution of carbohydrate with either protein or monounsaturated fat can reduce blood pressure. When the results from the carbohydrate diet [low protein (LP)] were compared with the HP diet, the latter diet further decreased mean systolic blood pressure by 1.4 mm Hg ($P = 0.02$) (Appel *et al.* 2005). Different types of protein affect blood pressure differently, and in general, plant protein, for example soy, has been shown to have a beneficial effect on blood pressure. Other trials have investigated the effects of HP diets on blood lipids. Two recent meta-analyses examined the effects of HP and LP diets on blood lipids. Nordmann and colleagues conducted a meta-analysis of five trials and found that triglyceride and HDL-cholesterol concentrations were decreased more with HP than LP diets after 6 months and total cholesterol and LDL-cholesterol concentrations decreased more with LP than HP diets (Nordmann *et al.* 2006). A recent meta-analysis of 13 studies of subjects with type 2 diabetes proposed that an increase in protein from 15% daily energy to 45% may lead to a 23% decrease in triglyceride concentrations (Kirk *et al.* 2008). In addition, a small number of RCTs suggest a role for dietary protein in glycaemic control. Some studies propose that HP diets may decrease postprandial glucose levels in individuals with type 2 diabetes. Overall, RCTs have yielded mixed findings and larger sample size interventions are needed. However, given the health concerns with very HP protein diets, potential results need to be interpreted carefully before adopting any potential recommendations (see section on meat and satiety).

Iron and associated risk of CVD

Studies investigating haem iron in red meat and CVD have been inconsistent. Some studies have shown a positive association with myocardial infarction and fatal CHD (Lee *et al.* 2005; Sinha *et al.* 2009), while other studies have shown no association (Malaviarachchi *et al.* 2002). A recent draft report on iron and health from SACN concluded that evidence from observational studies of total iron intake or status and CVD do not suggest an association, and limited data are available from prospective studies, suggesting a positive association between high iron intake and increased risk of CVD (SACN 2010). The Committee proposed that more prospective studies of longer duration with more accurate and reliable measures of haem iron intake are needed to investigate this association further.

Homocysteine status and vitamin B_{12}

Observational studies have consistently proposed that blood homocysteine concentration is positively associated with risk of CVD (Clarke *et al.* 1991; Boushey *et al.* 1995; Danesh & Lewington 1998), although it remains unclear whether this is causative. A meta-analysis of prospective studies indicated that after adjustment for other cardiovascular risk factors, a 25% lowering in homocysteine was associated with about a 10% lower risk of CHD and 20% lower risk of stroke (Collaboration 2002). Homocysteine levels can be lowered with folic acid and B_{12} (Homocysteine Lowering Trialists' Collaboration 2005) and it has been suggested that lowering plasma homocysteine concentrations might reduce CVD incidence in individuals. However, a number of large-scale randomised trials in individuals with pre-existing CVD have failed to show any beneficial effects of B-vitamin supplementation on cardiovascular risk (Albert *et al.* 2008; Clarke *et al.* 2010).

Selenium and associated risk of CVD

Although selenium is present in moderate amounts in meat, red meat makes a significant contribution to overall intake (see section on Nutrient composition of red meat). The role of selenium in the activity of specific antioxidant enzymes, particularly glutathione peroxidase, has been well established (Holben & Smith 1999). Low selenium intakes have been reported in areas of China (Keshan region) where soil levels of the nutrient are particularly low and deficiency causes endemic Keshan disease. The acute form of Keshan disease is characterised by sudden onset of cardiac insufficiency (*i.e.* inadequate blood flow to the heart muscles) and the chronic form causes moderate to severe heart enlargement with some degree of cardiac insufficiency. In case control studies, individuals with myocardial infarction have low plasma selenium concentrations, but this could be a consequence of the disease (BNF 2001). Meta-analyses of selenium supplementation trials (dose ranging from 75–200 μg/d) have concluded there is inadequate evidence to suggest a protective role of selenium supplements in CHD (Flores-Mateo *et al.* 2006) or atherosclerosis progression (Bleys *et al.* 2006). However, a more recent review concluded that chronically increased selenium intake in selenium-replete population may actually increase the risk of diabetes or hypercholesterolemia (Navas-Acien *et al.* 2008). In summary, larger high-quality RCTs and observational studies are required across different population groups with varying levels of habitual selenium intake to understand better the relationship between selenium intake and risk of CVD.

Vitamin D and CVD

Meat and meat products make a substantial contribution to the dietary intake of vitamin D in both men and women in the UK (see section on micronutrient composition of red meat). A role of vitamin D in the development of CVD has been speculated, based on observational studies and have found an increase risk of hypertension among people living at higher latitudes (where the angle of the sun is oblique such that most of the UVB photons are absorbed by the ozone layer and hence production of pre-vitamin D3 is halted) (Zittermann *et al.* 2005). This finding led to the premise that reduced sunlight exposure (and a knock on effect on dermal vitamin D synthesis) can be linked to the aetiology of CVD. However, no causal links have been established as yet. A recent RCT in women, with 5 μg vitamin D combined with a calcium supplement, found no association between vitamin D and CVD at 7-year follow-up (Hsia *et al.* 2007). There is a paucity of mechanistic data linking vitamin D to CVD risk; however, this area of vitamin D research is important given the widespread prevalence of poor vitamin D status globally.

Summary

Pooled analyses of prospective studies investigating the effects of dietary patterns on cardiovascular risk have yielded mixed findings. Individuals adopting a vegetarian dietary pattern may be at a lower risk of CVD than those adopting other diets, but such data can not be used to make specific associations about meat consumption. Vegetarians and omnivores differ in a number of respects. Some prospective studies have shown a modestly increased risk of CVD amongst meat eaters compared with low meat eaters, but controlling for potential confounders and other aspects of the diet remains difficult. In recent years, prospective studies have moved towards investigating the evidence for relationships of red (unprocessed) and processed meat separately on risk of CVD. Findings are currently inconclusive and more prospective studies of large population groups are needed to establish whether a relationship really exists.

While red meat contains SFAs, a high intake of which can have adverse effects on CVD risk factors such as blood cholesterol levels, it also contains other fatty acids (*n*-3 PUFAs, MUFAs) and nutrients (*e.g.* B vitamins and selenium) that may offer potential cardio-protective benefits. Intervention studies with lean red meat as part of a healthy diet and in combination with blood pressure-lowering drugs have shown favourable effects on blood cholesterol levels, which suggests that lean red meat can be promoted as part of a healthy diet for primary and secondary CVD prevention.

Type 2 diabetes

The prevalence of diabetes is increasing worldwide, with an estimated 285 million people affected in 2010 and 438 million people expected to be affected by 2030 (Diabetes UK 2010). In the UK, there are currently 2.6 million people who have been diagnosed with diabetes, which is almost twice the number (1.4 million) diagnosed with diabetes in 1996. It is estimated that over four million people in the UK will have diabetes by 2025 and that there are up to half a million more people in the UK who have diabetes but have not been diagnosed. In the UK, 90% of people with diabetes have type 2 diabetes (Diabetes UK 2010). Risk factors for type 2 diabetes include family history; increasing age, which explains some of the increase in prevalence as life expectancy is increasing; overweight and obesity, in particular abdominal obesity as well as dietary factors (Buttriss 2005).

The association between red and processed meat and risk of type 2 diabetes has been investigated in a number of cohort studies and summarised in a recent systematic review (Aune *et al.* 2009). The authors of the review carried out a meta-analysis based on ten cohort studies on the association between red meat intake and type 2 diabetes risk. Overall, study participants in the highest red meat consumption groups (the absolute amount eaten varying between studies or not known, see Table 11) had a significantly increased risk of developing type 2 diabetes compared with those in the lowest consumption groups, with a summary RR of 1.21 (95% CI 1.07–1.38). Meta-analysis of nine cohort studies on the association between processed meat and type 2 diabetes also found a significantly higher risk of developing type 2 diabetes in those in the highest consumption groups (again, the absolute amount eaten varying between studies or not known) compared to those in the lowest consumption groups with a summary RR of 1.41 (95% CI 1.25–1.60). However, the heterogeneity between the studies was significant in both analyses, which means that the results have to be interpreted with caution (Aune *et al.* 2009). Cohort studies included in the meta-analysis by Aune *et al.* (2009), as well as two subsequent cohort studies, are presented in Table 11. A significantly increased risk of type 2 diabetes in the highest vs. lowest red meat consumers was observed in three out of 11 cohort studies with RRs ranging from 1.30–1.64; in five studies, a non-significant increase in risk (RRs 1.17–1.44) was reported, and in three studies, no association was reported (RRs 0.94–1.05). However,

Table 11 Red and processed meat consumption and diabetes: prospective cohort studies (highest vs. lowest consumption; adapted from (Aune *et al.* 2009) and including results from two subsequent cohort studies (Mannisto *et al.* 2010; Steinbrecher *et al.* 2010)

Author, year published	Country/cohort details	Number in cohort (age)	Follow-up years (mean)	Number of cases type 2 DM	Red meat (fresh plus processed) Quantity (highest vs. lowest)	Red meat (fresh plus processed) Relative risk (95% CI)	Processed meat Quantity (highest vs. lowest)	Processed meat Relative risk (95% CI)	Adjustments
(van Dam *et al.* 2002)	Health Professionals' Follow-up Study, USA	42 04 men (40–75 y)	1986–1998 (11 y)	1321	Highest vs. lowest quintile[§†]	1.05 (0.85–1.30)	≥5 times/week vs. 1 time/month	1.46 (1.14–1.86)*	Age, total energy, time period, physical activity, cigarette smoking, alcohol, HC, hypertension, FH-DM, cereal fibre, Mg, BMI
(Schulze *et al.* 2003)	Nurses' Health Study II, USA	91 246 women (26–46 y)	1991–1999 (7.8 y)	741	≥5 vs. <1 time/week	1.44 (0.92–2.24)	≥5 vs. <1 time/week	1.82 (1.34–2.46)*	Age, BMI, FH-DM, alcohol, HC, smoking, menopausal status, dietary energy, hypertension, physical activity, HRT, OC, cereal fibre, Mg, caffeine, GI
(Lee *et al.* 2004)	Iowa Women's Health Study, USA	35 698 women (55–69 y)	1986–1997 (9.3 y)	1921	Highest vs. lowest quintile[§]	1.19 (0.97–1.45)	NA	NA	Age, total energy, WHR, BMI, physical activity, HRT, cigarette smoking, alcohol, education, marital status, residential area, animal fat, vegetable fat, cereal fibre, Mg
(Fung *et al.* 2004)	Nurses' Health Study, USA	69 554 women (38–63 y)	1984–1998 (12.8 y)	2699	0.96 vs. 0.21 servings/d[†]	1.36 (1.18–1.56)*	0.55 vs. 0.04 servings/d	1.60 (1.39–1.83)*	Age, FH-DM, HB, smoking, menopausal status, energy intake, hypertension, physical activity, alcohol, BMI
(Song *et al.* 2004)	Women's Health Study, USA	37 309 women (≥45 y)	1993–2003 (8.8 y)	1558	≥5 vs. <1 time/week; Highest vs. lowest quintile of intake (median servings/d 1.42 and 0.13)	1.25 (0.94–1.67) 1.24 (1.00–1.54)	≥5 vs. <1 time/week; Highest vs. lowest quintile of intake (median servings/d 0.56 and 0)	1.38 (1.11–1.71)* 1.19 (1.00–1.42)	Age, BMI, total energy, smoking, exercise, alcohol, FH-DM, fibre intake, GL, Mg, total fat
(Montonen *et al.* 2005)	Finnish Mobile Clinic Health Examination Survey, Finland	4303 men and women (40–69 y)	1967–1990	383	≥101 vs. <41 g/d	0.99 (0.72–138)	≥69 vs. <18 g/d	1.22 (0.89–1.69)	Age, sex, BMI, energy intake, smoking, FH-DM, geographic area

Table 11 *Continued*

Author, year published	Country/cohort details	Number in cohort (age)	Follow-up years (mean)	Number of cases type 2 DM	Red meat (fresh plus processed)		Processed meat		Adjustments
					Quantity (highest vs. lowest)	Relative risk (95% CI)	Quantity (highest vs. lowest)	Relative risk (95% CI)	
(Villegas *et al*. 2006)	Shanghai Women's Health Study, China	70 609 women (40–70 y)	1997–2000–2004 (4.6 y)	1972	≥68 vs. 25 g/d[†]	0.94 (0.79–1.12)	Consumers vs. non-consumers	1.18 (0.99–1.37)	Age, energy, BMI, WHR, smoking, alcohol, physical activity, vegetable intake, income level, education level, occupation status, hypertension
(Hodge *et al*. 2007)	Melbourne Collaborative Cohort Study, Australia	31 641 men and women (27–75 y)	1990–1994	365	8 vs. 2 servings/week	1.17 (0.83–1.66)	8 vs. 2 servings/ week	1.11 (0.79–1.56)	Age, energy intake, FH-DM, country of birth, BMI, WHR
(Schulze *et al*. 2007)	EPIC-Potsdam Cohort Study, Germany	25 167 men and women (35–65 y)	1994–1998–2005 (7.0 y)	849	Per 150 g/d	1.64 (1.23–2.19)*	NA	NA	Age, height, alcohol, waist circumference, hypertension, coffee, wholegrains, sports, biking, gardening, smoking
(Vang *et al*. 2008)	Adventist Mortality Study, Adventist Health Study, USA	8401 men and women (45–88 y)	1960–1976	543	NA	NA	Weekly vs. never	1.38 (1.05–1.82)*	Age, sex
(Steinbrecher *et al*. 2010)	Multiethnic Cohort Study, Hawaii	36 256 men and 39 256 women (45–75 y)	1993–2007 (13.5 y)	8587	Men: 35.6 vs. 5.4 g/4.2 MJ and day Women: 31.8 vs. 4.0 g/ 4.2 MJ and day	Men: 1.43 (1.29–1.59)* Women: 1.30 (1.17–1.45)*	Men: 17.1 vs. 1.7 g/ 4.2 MJ and day Women: 13.9 vs. 1.1 g/4.2 MJ and day	1.57 (1.42–1.75)* Women: 1.45 (1.30–1.62)*	Age, ethnicity, education, BMI, physical activity, total energy
(Mannisto *et al*. 2010)	Alpha-tocoperol, Beta-carotene Cancer Prevention Study, Finland	25 943 men (50–69 y)	1985–1993	1098	Highest vs. lowest quintile of intake (median intake 106 vs. 33 g/d)	1.22 (0.97–1.53)	Highest vs. lowest quintile of intake (median intake 142 vs. 22 g/d)	1.37 (1.11–1.71)*	Age, BMI, smoking, blood pressure, serum total cholesterol, serum HDL cholesterol, physical activity, intakes of alcohol, energy, fruits, vegetables, rye, milk and coffee

[†]Excluding processed meat.
*Statistically significant outcome.
[§]No absolute amounts given.
HC, hypercholesterolaemia; FH-DM, family history of diabetes mellitus; Mg, magnesium; BMI, body mass index; HRT, hormone replacement therapy; OC, oral contraception; GI, glycaemic index; WHR, waist-to-hip ratio; GL, glycaemic load.

seven out of ten studies found a significantly increased risk of type 2 diabetes in the highest compared with the lowest consumers of processed meat, with RRs ranging from 1.37–1.82; three studies found a non-significant increase in risk (RRs 1.11–1.22).

Another recent meta-analysis using slightly different inclusion criteria (*e.g.* the exclusion of studies looking at vegetarians vs. non-vegetarians) found a non-significant association between red meat intake and type 2 diabetes based on five cohort studies (RR 1.16, 95% CI 0.92–1.46) and found a significant association between intake of processed meat and diabetes based on seven cohort studies (RR of 1.19, 95% CI 1.11–1.27) (Micha *et al.* 2010).

Summary

Cohort studies have generally found 'high' consumers of red meat to have a higher risk of type 2 diabetes compared with those defined as 'low consumers', although the results of most individual studies have been non-significant. Meta-analyses of these cohorts have supported a positive association with 'high' intakes. However, major inconsistencies regarding consumption levels between the studies, with variable intakes for both 'high' and 'low' consumers and no indication of absolute amounts of intake in some studies, prevent any conclusions about the amount of meat that may potentially be associated with higher risk.

There is no evidence to suggest that lean red meat can not be recommended as part of a healthy balanced diet in respect to preventing type 2 diabetes as well as for people with established type 2 diabetes. Intake of protein has been associated with increased satiety. Therefore, lean meat could aid appetite and weight control, including among those with established type 2 diabetes (see section on meat and satiety).

Meat and cancer

A large number of studies have looked at the association between environmental and lifestyle factors, including dietary factors, and risk of cancer. The first evidence suggesting that cancer is a largely preventable disease came from studies describing changes in the rates of different cancers in genetically identical populations that migrate from their native countries to other countries [World Cancer Research Fund (WCRF) and American Institute for Cancer Research (AICR) 2007]. Overall, evidence on the relationship between diet and cancer mainly comes from prospective cohort studies or case control studies. The type of evidence included in this review is discussed earlier (see section on evidence base). This section will focus mainly on prospective cohort studies as these are generally less prone to bias than other epidemiological study designs. The most studied cancer in relation to red and processed meat intake is colorectal cancer (CRC). Other cancer sites that have been investigated in relation to meat include the oesophagus, stomach (gastric), lung, pancreas, endometrium and breast.

Colorectal cancer (CRC)

Data show that CRC is the third most common cancer in men (663 000 cases in 2008, 10.0% of the total) and the second most common cancer in women (570 000 cases, 9.4% of the total) worldwide. Incidence rates vary ten-fold in both sexes worldwide, the highest rates being estimated in Australia/New Zealand and Western Europe, the lowest in Africa (except Southern Africa) and South Central Asia. In 2008, approximately 608 000 deaths from CRC estimated worldwide, accounting for 8% of all cancer deaths, making it the fourth most common cause of death from cancer (Ferlay *et al.* 2010). However, estimation of cancer incidence in many developing countries proves difficult and often only data from certain regions or bigger cities are available (Ferlay *et al.* 2010). Therefore, lower rates in developing countries may partly be explained by cancer cases remaining undiagnosed. However, the varying incidence rates are likely to also reflect findings that show that most cases of CRC are sporadic rather than genetic and seem to be influenced by environmental and lifestyle factors, such as diet and physical activity. Around 5% to 10% of CRCs are a consequence of recognised hereditary conditions and a further 20% of cases occur in people who have a family history of CRC (WCRF and AICR 2007). Therefore, lifestyle-related factors are suggested to have a significant impact on the risk of CRC and many cases may be avoided by choosing a healthier lifestyle.

Increasing age has been suggested to be the biggest single risk factor of CRC, with 84% of cases being diagnosed in people aged 60 years or over (Cancer Research UK 2010). Therefore, with the increase in life expectancy, the incidence of CRC is also increasing. Another major factor impacting on CRC mortality is screening. It has been suggested that recent decreases in CRC mortality rates in several European countries are likely to be caused by improvements in (early) diagnosis and treatment, with a consequent higher survival rate from the disease (Bosetti *et al.* 2010). In the UK, a National Bowel Cancer Screening Programme has been

Box 2 WCRF criteria for grading evidence
Convincing: evidence is strong enough to support a judgement of a convincing causal relationship, which justifies goals and recommendations designed to reduce the incidence of cancer. A convincing relationship should be robust enough to be highly unlikely to be modified in the foreseeable future as new evidence accumulates.
Probable: evidence is strong enough to support a judgement of a probable causal relationship, which would generally justify goals and recommendations designed to reduce the incidence of cancer.
Limited – suggestive: evidence is too limited to permit a probable or convincing causal judgement. The evidence may have methodological flaws, or be limited in amount, but shows a generally consistent direction of effect.
Limited – no conclusion: evidence is so limited that no firm conclusion can be made.
Source: (WCRF and AICR 2007).

set up following outcomes from pilot studies showing a high detection rate of bowel cancer with a highly significant shift towards earlier stage disease in the screened group (Ellul *et al*. 2010; National Health Service 2010).

The 2007 expert report of the WCRF and the AICR concluded that there was *convincing* evidence (see Box 2) that physical activity decreased the risk of CRC and that intake of red meat, processed meat, alcoholic drinks (men), body fatness and abdominal fatness increased the risk of CRC. They also concluded that a decrease in CRC risk was *probable* for foods containing dietary fibre, garlic, milk and calcium, and an increase in CRC risk was probable for alcoholic drinks in women. There was only *limited* evidence that suggested that fruits, vegetables and fish have a protective effect on CRC (WCRF and AICR 2007).

A meta-analysis of 15 prospective studies investigating the association between red meat consumption and the risk of CRC found a significantly increased risk of CRC in the highest category of red meat consumption vs. the lowest category (see studies in Table 12); the RR was 1.28 (95% CI, 1.15–1.42), which means that the risk of developing CRC was 28% higher in the highest compared to the lowest category of red meat consumption (Larsson & Wolk 2006). The association with red meat was stronger for rectal cancer (RR 1.56; 95% CI, 1.25–1.95) than for colon cancer (RR 1.21; 95% CI 1.05–1.40). Processed meat was also associated with a higher risk of CRC, with a RR of the highest consumption category vs. the lowest category of 1.20 (95% CI, 1.11–1.31, based on 14 studies), the risk being similar for colon and rectum (Larsson & Wolk 2006). Two subsequently published prospective cohort studies showed conflicting results. The National Institutes of Health (NIH-AARP) Diet and Health Study found a significantly increased CRC risk in the highest quintile compared with the lowest quintile of red meat intake (RR 1.24, 95% CI 1.12–1.36, P for trend <0.001; Table 12) (Cross *et al*. 2007), but no clear association was found between red meat intake and either adenocarcinoma (HR 1.41, 95% CI 0.66–3.01 in the highest vs. lowest tertile of intake) or carcinoid tumours (HR 1.44, 95% CI 0.78–2.69 in the highest vs. lowest tertile of intake) of the small intestine (Cross *et al*. 2008). The Canadian National Breast Screening Study did not observe a significantly increased risk of CRC in the highest compared with the lowest quintile of red meat intake, although there was a significantly increased risk of rectal cancer (RR 1.95, 95% CI 1.21–3.16; Table 12) (Kabat *et al*. 2007).

The WCRF and the AIRC have also conducted systematic reviews of the literature on the association between diet and physical activity and cancer. Based on their findings from evidence on red meat and CRC, the expert panel concluded that there was *convincing* evidence that red meat consumption, as well as processed meat consumption, were associated with an increased risk of CRC. A meta-analysis of studies reporting on frequency of red meat consumption showed similar findings to the meta-analysis by Larsson and Wolk (2006) with a RR of 1.43 (95% CI, 1.05–1.94) with each occasion of red meat consumption per day. A meta-analysis of studies reporting an increase in RR per 100 g/d also showed a significant increase in CRC risk with an estimated summary risk of 1.29 (95% CI, 1.04–1.60) (WCRF and AICR 2007).

There has been much debate on the conclusions of WCRF/AIRC (Harland 2010; Truswell 2009). The SACN, for example, have also looked at the evidence around red meat intake and the risk of CRC in the course of their work on a report on the role of iron in the diet (final report to be published early 2011). In their latest preliminary draft of the Iron Report,[3]

[3]Available at http://www.sacn.gov.uk/ (2010).

Table 12 Red and processed meat consumption and colorectal cancer: prospective cohort studies (highest versus lowest consumption; adapted from (Larsson & Wolk 2006) and including results from two subsequent cohort studies (Cross *et al*. 2007; Kabat *et al*. 2007)

					Red meat (fresh plus processed)		Processed meat		
Author, year published	Country/cohort details	Number in cohort (age)	Follow-up years (mean)	Number of cases CRC	Quantity (highest vs. lowest)	Relative risk (95% CI)	Quantity (highest vs. lowest)	Relative risk (95% CI)	Adjustments
(Bostick *et al*. 1994)	Iowa Women's Health Study, USA	35 215 women (55–69 y)	1986–1990 (4 y)	212 CC	>11 vs. <4 servings/ week	1.04 (0.62–1.76)	>3 servings/ week vs. none	1.51 (0.72–3.17)	Age, height, parity, vitamin A supplement use, intakes of energy and total vitamin E
(Kato *et al*. 1997)	New York University Women's Health Study, USA	14 727 women (34–65 y)	1985–1994 (7.1 y)	100 CRC	Highest vs. lowest quintile[§]	1.23 (0.68–2.22)	Highest vs. lowest quintile[§]	1.09 (0.59–2.02)	Age, place of enrolment, education, energy intake
(Hsing *et al*. 1998)	Lutheran Brotherhood Study, USA	17 633 men (≥35 y)	1966–1986	145 CRC deaths[†] 120 CC deaths[†]	≥60 vs. <15 times/month	1.9 (0.9–4.3) 1.8 (0.8–4.4)	NA	NA	Age, smoking, alcohol, energy intake
(Singh & Fraser 1998)	Adventist Health Study, USA	32 051 women and men (≥25 y)	1976–1982	157 CC	≥1 time/week vs. never	1.41 (0.90–2.21)	NA	NA	Age, sex, family history, smoking, BMI, physical activity, aspirin use, alcohol
(Pietinen *et al*. 1999)	Alpha-tocopherol, Beta-carotene Cancer Prevention Study, Finland	27 111 male smokers (50–69 y)	1988–1995 (8 y)	185 CRC	Highest vs. lowest quartile (median intake 99 vs. 35 g/d)[¶]	0.8 (0.5–1.2)	Highest vs. lowest quartile (median intake 122 vs. 26 g/d)	1.2 (0.7–1.8)	Age, supplement group, education, smoking years, BMI, physical activity, alcohol, calcium intake
(Järvinen *et al*. 2001)	Finnish Mobile Clinic Health Examination Survey, Finland	9959 women and men (15–99 y)	1966–1999	109 CRC 63 CC 46 RC	>206 vs. <94 g/d in men; >134 vs. <61 g/d in women	1.50 (0.77–2.94) 1.34 (0.57–3.15) 1.82 (0.60–5.52)	NA	NA	Age, sex, occupation, geographic area, smoking, BMI, intakes of energy, vegetables, fruits and cereals
(Flood *et al*. 2003)	Breast Cancer Detection Demonstration Project, USA	45 496 women (40–93 y)	1987–1998 (8.5 y)	487 CRC	Highest vs. lowest quintile (median 52.2 vs. 6.1 g/ 1000 kcal)	1.10 (0.83–1.45)	Highest vs. lowest quintile (median 22.2 vs. 0.02 g/ 1000 kcal)	1.00 (0.76–1.31)	Age, energy intake
(Wei *et al*. 2004)	Nurses' Health Study, USA	87 733 women (30–55 y)	1980–2000	876 CRC 672 CC 204 RC	≥5 times/week vs. never[††]	1.21 (0.72–2.03) 1.31 (0.73–2.36) 0.92 (0.31–2.71)	≥5 times/week vs. never	1.10 (0.64–1.88) 1.32 (0.95–1.83) 0.72 (0.33–1.59)	Age, history of endoscopy, family history, smoking, height, BMI, physical activity, intakes of alcohol, calcium and folate
(Wei *et al*. 2004)	Health Professionals Follow-Up Study, USA	46 632 men (40–75 y)	1986–1999	602 CRC 467 CC 135 RC	≥5 times/week vs. never[††]	1.24 (0.78–1.96) 1.35 (0.80–2.27) 0.90 (0.34–2.45)	≥5 times/week vs. never	1.23 (0.87–1.73) 1.27 (0.87–1.85) 1.06 (0.48–2.33)	Age, history of endoscopy, family history, smoking, height, BMI, physical activity, intakes of alcohol, calcium and folate
(English *et al*. 2004)	Melbourne Collaborative Cohort Study, Australia	37 112 women and men (40–69 y)	1990–2002 (9 y)	451 CRC 283 CC 169 RC	≥6.5 vs. <3 times/week	1.4 (1.0–1.9) 1.1 (0.7–1.6) 2.3 (1.2–4.2)*	≥4 vs. 1.5 times/week	1.5 (1.1–2.0)* 1.3 (0.9–1.9) 2.0 (1.1–3.4)*	Age, sex, country of birth, intakes of energy, fat and cereals

Table 12 *Continued*

Author, year published	Country/cohort details	Number in cohort (age)	Follow-up years (mean)	Number of cases CRC	Red meat (fresh plus processed): Quantity (highest vs. lowest)	Red meat (fresh plus processed): Relative risk (95% CI)	Processed meat: Quantity (highest vs. lowest)	Processed meat: Relative risk (95% CI)	Adjustments
(Larsson *et al.* 2005)	Swedish Mammography Cohort, Sweden	61 433 women (40–75 y)	1987–2003 (13.9 y)	733 CRC	≥94 vs. <50 g/d (median 114 vs. 37 g/d)	1.32 (1.03–1.68)*	≥32 vs. <12 g/d (median 41 vs . 6 g/d)	1.07 (0.85–1.33)	Age, education, BMI, intakes of energy, alcohol, saturated fat, calcium, folate, fruits, vegetables and wholegrain foods
				389 CC		1.41 (0.92–2.16)		1.06 (0.83–1.35)	
				230 RC		1.28 (0.83–1.98)		0.90 (0.60–1.34)	
(Chao *et al.* 2005)	Cancer Prevention Study II Nutrition Cohort, USA	148 610 women and men (50–74 y)	1992–2001	1667 CRC 1197 CC 470 RC	men: >800 vs. ≤180 g/week (median 999 vs. 100 g/week); women: >560 vs. ≤90 g/week (median 712 vs. 43 g/week)	1.36 (0.93–2.00) 1.15 (0.90–1.46) 1.71 (1.15–2.52)*	men: Highest vs. lowest quintile (median 283 vs. 10 g/week); women: median 145 vs. 0 g/week)	1.16 (0.96–1.40) 1.13 (0.91–1.41) 1.26 (0.86–1.83)	Age, sex, smoking, education, hormone therapy use (women), BMI, physical activity, multivitamin use, aspirin use, intakes of energy, alcoholic beverages, fruits, vegetables and high-fibre grain foods
(Norat *et al.* 2005)	EPIC, Europe[‡]	478 040 women and men (35–70 y)	1992–1998 (4.8 y)	1329 CRC	≥80 vs. <10 g/d	1.35 (0.96–1.88)	≥80 vs. <10 g/d	1.42 (1.09–1.86)*	Age, sex, centre, smoking, height, weight, physical activity, intakes of alcohol and energy
				855 CC		1.17 (0.78–1.77)		1.30 (0.92–1.84)	
				474 RC		1.75 (0.98–3.10)		1.62 (1.04–2.50)*	
(Cross *et al.* 2007)	National Institutes of Health (NIH-AARP) Diet and Health Study, USA	494 036 women and men (50–71 y)	1995–2003	5107 CRC	Highest vs. lowest quintile (median 62.7 vs. 9.8 g/1000 kcal)	1.24 (1.12–1.36)*	Highest vs. lowest quintile (median 22.6 vs. 1.6 g/1000 kcal)	1.20 (1.09–1.32)*	Age, sex, education, marital status, family history of cancer, race, BMI, smoking, physical activity, intakes of energy, alcohol, fruits and vegetables
(Kabat *et al.* 2007)	Canadian National Breast Screening Study, Canada	49 654 women (40–59 y)	1982–2000 (16.4 y)	617 CRC	≥40.3 vs. <14.3 g/d	1.12 (0.86–1.46)	NA	NA	Age, BMI, menopausal status, oral contraceptive use, hormone replacement use, smoking, education, physical activity, intakes of fat, fibre, folic acid, energy and alcohol
				428 CC		0.88 (0.64–1.21)			
				195 RC		1.95 (1.21–3.16)*			

*Statistically significant.
[†]Deaths.
[‡]EPIC includes subjects from 10 European countries: Denmark, France, Germany, Greece, Italy, Netherlands, Norway, Spain, Sweden and UK.
[§]No absolute amounts given.
[¶]excl. processed meat (beef, pork and lamb).
[††]excl. processed meat (beef, pork and lamb as a main dish).
N, number; NA, not available; CRC, colorectal cancer; CC, colon cancer; RC, rectal cancer; BMI, body mass index.

SACN also reported that results from prospective cohort studies have consistently suggested that high intakes of red and processed meat are associated with increased CRC risk. They stated that although the increased risk was not statistically significant in most studies, this may be caused by lack of statistical power owing to too small cohorts. However, SACN concluded that the available evidence suggested that red and processed meat intake was *probably* associated with increased CRC risk. Recent reviews of prospective epidemiological studies have concluded that the currently available evidence is not sufficient to support a clear positive association between red meat consumption (Alexander & Cushing 2010) or processed meat cosumption (Alexander *et al.* 2010), and colorectal cancer. SACN argues that there are a number of methodological inconsistencies between the different studies that make comparisons difficult. For example, there have been considerable inconsistencies between studies in categorisation and definition of red and processed meat, some including only fresh meat whereas others also include processed meat, such as burgers and sausages, in the red meat category. The way that quintiles of red and processed meat intake were reported (for example, frequencies or total amounts) was variable, and there were large differences in the quintiles of intake between studies so that the amounts in the lowest quintiles in some studies are higher than the top quintiles in others. Many studies also did not adjust for foods and nutrients that have been associated with a decreased risk of CRC, such as fruits, vegetables and fibre (SACN 2010).

Most prospective cohort studies use FFQs to estimate the intake of different foods, which are less accurate in estimating food intake than other methods (see section on Evidence Base). An analysis of standardised individual dietary data from food diaries in case control studies nested within seven prospective cohorts in the UK (EPIC Norfolk, EPIC Oxford, Guernsey Study, Medical Research Council (MRC) National Survey of Health and Development, Oxford Vegetarian Study, Whitehall II, UK Women's Cohort Study), which also quantified disaggregated intakes of meat from mixed dishes, found that neither red meat nor processed meat were associated with an increased risk of CRC. The odds ratio (OR) for people eating, on average, more than 50 g red meat per day vs. those who ate less than 5 g per day was 0.91 (95% CI 0.66–1.24) and the OR for people eating, on average, more than 30 g processed meat per day vs. those who ate less than 5 g per day was 0.76 (95% CI 0.56–1.03) (Spencer *et al.* 2010).

Proposed mechanisms

A number of plausible mechanisms for the association between red and processed meat intake and CRC incidence have been suggested. However, so far, none of these potential mechanisms has been definitively established (Key *et al.* 2002). The most plausible mechanisms identified so far to explain why red meat intake may be a risk factor for colorectal carcinogenesis involve the meat-related mutagens heterocyclic amines, polycyclic aromatic hydrocarbons and *N*-nitroso compounds (Cross & Sinha, 2004; Tabatabaei *et al.* 2010). These are discussed in more detail in Williamson *et al.* (2005).

Oesophageal cancer

Oesophageal cancer is the eighth most common cancer worldwide, with 481 000 new cases (3.8% of the total) estimated in 2008 and the sixth most common cause of death from cancer with 406 000 deaths (5.4% of the total). More than 80% of the cases and of the deaths occur in developing countries (Ferlay *et al.* 2010). Major risk factors for oesophageal cancer are smoking and gastric reflux, but also, exposure to food and drink can play a role (WCRF and AICR 2007). The WCRF and AIRC reported on 12 case-control studies examining the impact of red meat consumption on the risk of oesophageal cancer (WCRF and AICR 2007). Eight studies reported increased risk for the highest intake group when compared with the lowest, which was statistically significant in five. Three studies reported non-significantly decreased risk, and one study reported no significant effect on risk but did not provide further details. The WCRF/AIRC concluded that 'there is *limited* evidence, from case control studies, some of which were poor quality, suggesting that red meat is a cause of oesophageal cancer' (WCRF and AICR 2007). Results from two prospective cohort studies have been published since. Findings from the EPIC study, including around 500 000 subjects from ten different European countries, showed a non-significant increased risk of oesophageal cancer in the highest tertile of red meat intake (≥73 g/d in men and ≥51 g/d in women; including fresh, minced and frozen red meat, excluding processed meat) compared with the lowest tertile (≤34 g/d in men and 23 g/d in women; HR 1.67, 95% CI 0.75–3.72), but there was a significantly increased risk in the highest tertile of processed meat intake (≥49 g/d in men and ≥30 g/d in women) compared with the lowest tertile (≤22 g/d in men and ≤13 g/d in women; HR 3.54, 95% CI 1.57–7.99) (Gonzalez *et al.* 2006). The findings of the NIH-

AARP Diet and Health Study, also entailing in and around 500 000 subjects, showed that the highest quintile of red meat intake (64.8 g/1000 kcal; including processed meat) was associated with a significantly increased risk of oesophageal squamous cell carcinoma compared to the lowest quintile (10.0 g/1000 kcal; HR 1.79, 95% CI 1.07–3.01), but no association could be observed for red meat intake and oesophageal adenocarcinoma (Cross *et al.* 2010). In contrast to the findings of Gonzalez *et al.* (2006), no increased risk of either form of oesophageal cancer was observed for processed meat (Cross *et al.* 2010).

Stomach cancer

Stomach cancer is currently the fourth most common malignancy in the world behind cancers of the lung, breast and colorectum. More than 70% of cases (712 000 cases) occur in developing countries (466 000 in men, 246 000 in women), and half the world total occurs in Eastern Asia (mainly in China). Age-standardised incidence rates are about twice as high in men as in women. Stomach cancer is the second leading cause of cancer death in both sexes worldwide (737 000 deaths, 9.7% of the total) (Ferlay *et al.* 2010). Risk factors include age, exposure to carcinogenic substances in food and environment, genetic predisposition and infection with *Helicobacter pylori* (WCRF and AICR 2007). It has been estimated that *H. pylori* is responsible for 63% of all gastric cancers worldwide (Parkin 2002). There are a number of studies available on the association of processed meat consumption and stomach cancer, but there is very limited evidence around the effect of red meat in general (fresh only or fresh including processed). A meta-analysis summarising the findings from studies on processed meat and stomach cancer reported a non-significantly increased risk for individuals in the highest vs. the lowest category of processed meat consumption (RR 1.24, 95% CI 0.98–1.56) based on seven cohort studies and a significantly increased risk (RR 1.63, 95% CI 1.31–2.01) based on 12 case-control studies. The estimated summary RR of stomach cancer for an increase in processed meat consumption of 30 g/d was 1.15 (95% CI 1.04–1.27) for cohort studies and 1.38 (95% CI 1.19–1.60) for case control studies (Larsson *et al.* 2006). A meta-analysis of eight cohort studies carried out by the WCRF and AIRC resulted in a summary effect estimate of 1.02 (95% CI 1.00–1.05) per 20 g/d processed meat, and a meta-analysis of nine case control studies resulted in a summary effect estimate of 1.13 (95% CI 1.01–1.25) per 20 g/d. The WCRF and AICR concluded that 'the evidence is inconsistent. There is *limited* evidence suggesting that processed meat is a cause of stomach cancer' (WCRF and AICR 2007).

One of the cohort studies included in both meta-analyses also reported on the association between red meat (including fresh, minced and frozen red meat, excluding processed meat) and stomach cancer. The authors reported a significantly increased risk in the highest quartile of red meat intake (average intake 85 g/d in men and 53 g/d in women) compared with the lowest quartile (average intake 34 g/d in men and 23 g/d in women; RR 1.50, 95% CI 1.02–2.22; $P = 0.05$), which is slightly lower than the reported risk in the highest quartile of processed red meat intake (average intake 86 g/d in men and 45 g/d in women) compared with the lowest quartile (average intake 19 g/d in men and 13 g/d in women; RR 1.62, 95% CI 1.08–2.41, $P = 0.02$) (Gonzalez *et al.* 2006). In a subsequent study, intakes of red meat (including processed meat) and processed meat alone were not associated with an increased risk of stomach cancer. The HR for cancer of the gastric cardia in the highest quintile of red meat intake (median intake 64.8 g/1000 kcal) compared with the lowest quintile (median intake 10.0 g/1000 kcal) was 1.04 (95% CI 0.72–1.51) and the HR for cancer of the non-cardia stomach was 0.77 (95% CI 0.56–1.06). The respective numbers for the highest quintile of processed meat consumption (median intake 23.2 g/1000 kcal) compared with the lowest quintile (median intake 1.7 g/1000 kcal) were 0.82 (95% CI 0.59–1.14) for cancer of the gastric cardia and 1.09 (95% CI 0.81–1.48) for gastric non-cardia (Cross *et al.* 2010).

Lung cancer

Lung cancer has been the most common cancer in the world for several decades and by 2008, there were an estimated 1.61 million new cases, representing 12.7% of all new cancers. It was also the most common cause of death from cancer in 2008, with 1.38 million deaths (18.2% of total cancer deaths). Tobacco smoking is the main cause for lung cancer, being responsible for approximately 85% of all lung cancer cases and up to 90% in populations with a high proportion of smokers. Other causes are exposure to various environmental carcinogens, including asbestos and some pollutants (WCRF and AICR 2007). Diet has also been suggested to impact on the risk of developing lung cancer. The WCRF and AIRC reviewed the evidence around red meat consumption and risk of lung cancer. Based on only one cohort study and nine case control studies, the expert panel suggested that 'there is *limited* evidence, mostly from inconsistent case-control studies, suggesting that red meat is a cause of lung cancer' (WCRF and AICR 2007). The single cohort study included in the WCRF/AIRC report showed an increased risk for the highest red meat intake group (>6.6 servings/week) when compared with the lowest (0–2.3 servings/week; RR 1.6, 95% CI 1.0–2.6, *P* for trend <0.014) but found no increased risk in the highest intake group of processed meat (>3 servings/week) compared with the lowest intake group (0–0.5 servings/week) (Breslow *et al.* 2000). One subsequent cohort study found a significantly increased risk of lung cancer in the highest quintile of red meat intake (median intake 62.7 g/1000 kcal) and processed meat intake (median intake 22.6 g/1000 kcal) compared with the lowest quintile (median intake 9.8 and 1.6 g/1000 kcal, respectively; RR 1.20, 95% CI 1.10–1.31 and RR 1.16, 95% CI 1.06–1.26, respectively). Sensitivity analysis excluding processed meats from the red meat variable did not change the positive association for red meat and lung cancer (data not given) (Cross *et al.* 2007). Two subsequent case control studies found an increased risk of lung cancer in the highest compared to the lowest consumers of red meat. In a study carried out in Italy, the OR was 1.8 (95% CI 1.5–2.2, *P* for trend <0.001) in the highest tertile (median 3.7 times/week in men and 3.5 times/week in women) compared with the lowest tertile of consumers of fresh red meat (median 0.6 times/week in men and 0.4 times/week in women) (Lam *et al.* 2009). A study in Uruguayan men showed an OR of 2.33 (95% CI 1.63–3.32) for those in the highest quartile of consumers of red meat (>9 servings/week of beef and lamb) compared with the lowest (≤5.0 servings/week) (De Stefani *et al.* 2009).

Pancreatic cancer

Pancreatic cancer is more prevalent in high-income countries (10–15 per 100 000 people in Europe) compared with poorer regions in the world (<1 per 100 000 in areas of Africa and Asia. The risk of pancreatic cancer increases with age, with most diagnoses made in people aged between 60 and 80 years. Established causes of pancreatic cancer include chronic pancreatitis and tobacco use, and it is suggested that type 2 diabetes and increased body fatness are also associated with a higher risk of this cancer (WCRF and AICR 2007). The expert panel of the WCRF and AIRC included seven cohort studies and four case control studies in their systematic review. Six cohort studies showed an increased risk for the highest intake group of red meat when compared with the lowest, but the increase was significant in only one study. All four case-control studies showed increased risk for the highest red meat intake group when compared with the lowest, which was statistically significant in three. Meta-analysis of three case-control studies gave a summary effect estimate of 1.11 (95% CI 1.08–1.15) per 20 g/d. The expert panel concluded that 'evidence from cohort studies is less consistent than that from case control studies'. There is *limited* evidence suggesting that red meat is a cause of pancreatic cancer' (WCRF and AICR 2007). One subsequent cohort study found that red meat intake was associated with a significantly increased risk of pancreatic cancer in the highest quintile of red meat intake (median intake 62.7 g/1000 kcal) compared with the lowest quintile (median intake 9.8 g/1000 kcal) in men (HR 1.43, 95% CI 1.11–1.83, *P* for trend = 0.001), but there was no effect on the risk in the female population. The respective results for processed meat (median intake 22.6 vs. 1.6 g/1000 kcal) were HR 1.31 (95% CI 1.01–1.68) for men and HR 0.86 (95% CI 0.60–1.22) for women. Sensitivity analysis excluding processed meats from the red meat variable resulted in no association between red meat intake and pancreatic cancer (data not given) (Cross *et al.* 2007).

Endometrial cancer

This form of cancer is more prevalent in higher than in middle- to low-income countries. Age-adjusted incidence rates range from more than 15 per 100 000 women in North America and parts of Europe to less than five per 100 000 in most of Africa and Asia. Risk factors for endometrial cancer are not bearing children, late natural menopause, body fatness and estrogen-only hormone replacement therapy (WCRF and AICR 2007). The expert panel of the WCRF and AICR identified one cohort study and seven case control studies investigating red meat and endometrial cancer. The cohort study showed no effect on endometrial cancer risk for the highest intake group when compared with the lowest, with an effect estimate of 1.10 (95% CI 0.70–1.73) (WCRF and AICR 2007). Five of the seven case-control studies showed increased risk for the highest intake group vs. the lowest, which was statistically significant in two. Two studies showed a non-significantly reduced risk. Meta-analysis of six case-control studies showed a summary effect estimate of 1.20 (95% CI 1.03–1.39) per 50 g red meat/d. Based on the current evidence, the panel concluded that 'the evidence, mostly from case-control studies, is sparse. There is *limited* evidence suggesting that red meat is a cause of endometrial cancer'

(WCRF and AICR 2007). A meta-analysis carried out by a different group of experts found a summary effect of 1.51 (95% CI 1.19–1.93) per 100 g red meat/d and a summary effect of 1.48 (95% CI 1.22–1.80) for the highest intake category compared with the lowest intake category (Bandera *et al.* 2007). Two subsequent prospective cohort studies found no increased risk of endometrial cancer with red meat intake (Cross *et al.* 2007; Kabat *et al.* 2008). Findings from the NIH-AARP Diet and Health Study even found a significantly decreased risk of endometrial cancer in the highest quintile of red meat intake compared with the lowest; HR 0.75, 95% CI 0.62–0.91, *P* for trend = 0.02). The authors reported that this association was not attenuated by adjustment for known risk factors, such as BMI or menopausal hormone therapy or by controlling for smoking. No effect of processed meat on endometrial cancer was found (Cross *et al.* 2007). Also, the Canadian National Breast Screening Study found a non-significant reduced risk of endometrial cancer in the highest quintile of red meat intake (≥109 g/d) compared with the lowest (<48.5 g/d; HR 0.86, 95% CI 0.61–1.22) (Kabat *et al.* 2008).

Breast cancer

Breast cancer is by far the most frequent cancer among women with an estimated 1.38 million new cancer cases diagnosed in 2008 (23% of all cancers) and ranks second overall (10.9% of all cancers in men and women). Incidence rates are high (greater than 80 per 100 000) in developed regions of the world (except Japan) and low (less than 40 per 100 000) in most of the developing regions. Risk factors of breast cancer include lifetime exposure to estrogen, influenced by early menarche, late natural menopause, not bearing children and late (over 30) first pregnancy, ionising radiation exposure, hormone replacement therapy, alcoholic drinks and body fatness. The expert panel of the WCRF and AIRC categorised evidence on meat intake and breast cancer as *limited/no conclusion* (WCRF and AICR 2007). Six prospective cohort studies reporting on red meat and breast cancer have been published in the past few years. One out of the six cohort studies found a significantly increased risk of breast cancer in high consumers of red meat (>103 g/d) compared with non-consumers of red meat (HR 1.41, 95% CI 1.11–1.81) (Taylor *et al.* 2007). Two studies reported a non-significant increase in breast cancer risk in the highest vs. the lowest meat consumption group with estimated risks of 1.27 (95% CI 0.96–1.67, *P* for trend = 0.28; for those consuming >1.5 servings/d compared with ≤3 servings/week) (Cho *et al.* 2006) and 1.23 (95% CI 1.00–1.51, *P* for trend = 0.22; for those in the highest quintile with an average intake of 57.1 g/1000 kcal compared with lowest quintile with an average intake of 8.8 g/1000 kcal) (Ferrucci *et al.* 2009). Three studies showed no, or virtually no, effect of red meat consumption on breast cancer risk with estimated risks of 1.02 (95% CI 0.93–1.12; for highest quintile compared with lowest quintile) (Cross *et al.* 2007), 1.06 (95% CI 0.98–1.14; for highest quintile with median intake of 84.6 g/d compared with lowest quintile with median intake of 1.4 g/d) (Pala *et al.* 2009) and 0.98 (95% CI 0.86–1.12; ≥98 g/d vs. <46 g/d) (Larsson *et al.* 2009).

Summary

Most of the evidence of the association between red meat and CRC shows an increase in risk of CRC in the highest consumers of red meat compared with the lowest consumers, although findings of most studies have not reached statistical significance. This could be caused by too small cohorts and other issues such as inappropriate/inadequate adjustment for confounders. However, there have been considerable inconsistencies between studies in the categorisation and definition of red meat and in the ways that quantities of red meat intake were reported. Based on the current evidence, it is not possible to make any conclusions on amounts of meat consumed that may potentially be associated with CRC risk, and more evidence from large cohort studies will be needed to draw clear conclusions. An expert panel from WCRF/AIRC is currently working on updating the section on CRC; the update is to be published early this year (2011). Any association between red meat intake and cancer at other sites remains inconclusive.

Establishing associations between dietary factors and cancer is difficult. Cancer develops over a long period of time so it is not possible to assess immediate effects of certain foods on the risk of cancer. When looking at past dietary patterns, changes throughout life are difficult to account for. Furthermore, it is difficult to assign estimated risks to certain foods as the intake of all foods is likely to have an impact on cancer risk, in addition to other lifestyle factors including physical activity or drinking habits. It is difficult to account for these and numerous potential other confounding factors that are not lifestyle-related (*e.g.* pollutants, passive smoking) Bearing this in mind, findings of associations between foods and cancer always have to be considered with caution as it is impossible to account for all possible factors influencing the risk of disease.

Bone health

Protein from animal sources is richer in sulphur amino acids (*e.g.* methionine and cysteine) than vegetable protein and as a result, produces more sulphuric acid. Consequently, for a number of years, diets high in meat have been suggested to have a negative effect on bone health because of the observations that high protein intakes can increase renal acid and urinary calcium excretion (Zemel, 1988; Sellmeyer *et al.* 2001).

However, not all studies are in agreement with these findings. Short-term high meat diets have been shown not to affect whole body calcium retention (Roughead *et al.* 2003) and high intakes of animal protein have been shown to significantly increase bone mineral density (Promislow *et al.* 2002, Hannan *et al.* 2000). The inconsistent findings suggest that other components of the diet are important and may also influence calcium excretion, such as calcium, potassium, phosphorous, salt, phytates and other components.

A recent review by Cao *et al.* (2010) concluded that evidence is lacking that shows high protein intakes, including from animal sources, affects whole body calcium balance or contributes to osteoporosis development or fracture risk (Cao *et al.* 2010).

Meat as part of a sustainable diet

There is increasing pressure on the global livestock sector to increase production and productivity to meet the rising demand for meat and dairy products, particularly in developing countries, and to feed the growing global population. At the same time, focus on the adverse aspects of the contribution of meat production, particularly beef and sheep meat, to climate change has raised questions among experts about whether meat can be part of a sustainable diet. According to the Food and Agriculture Organization (FAO), the agriculture sector is the world's largest user and steward of natural resources such as land and water, and it is estimated that livestock production accounts for 18% of global greenhouse gas emissions (FAO 2009). In the UK, the contribution of livestock production to green house gases emissions is lower at approximately 8% of total UK emissions (Millward & Garnett 2010), which reflects the production efficiencies that have been achieved by UK beef and sheep farmers in recent years. Nevertheless, it needs to be recognised that sheep in particular often graze on upland and arid land that could not be used to grow other food crops and that pasture is itself an effective carbon sink. Furthermore, there is now evidence that methane emissions by cattle can be lessened through nutritional management of animal feed (Beauchemin *et al.* 2008).

In the UK, the livestock sector, the government and many others are currently exploring opportunities to reduce the impact of livestock production on the environment. The UK Low Carbon Transition Plan aims to reduce greenhouse gas emissions from livestock production by 11% from current levels by 2020 (HM Government 2009). Breeding, feeding and management have been identified as areas where improvements could be made in beef and sheep meat production [English Beef and Lamb Executive (EBLEX) 2009; (EBLEX) 2010]. Although livestock production has its environmental challenges, it can also make positive contributions to other aspects of the environment (as mentioned earlier), society and the economy (*e.g.* provides a livelihood for many people living in rural areas).

Factors relating to meat consumption may also contribute to the impact of livestock on the environment. Various options have been suggested in order to reduce this impact, including that people eat less meat or no meat at all and that beef and sheep meat should be substituted with chicken or pork meat (as greenhouse gas emissions are lower for the production of these non-ruminant meats). However, as discussed earlier in the paper, red meat contains many important nutrients for good health and is an important contributor of these nutrients in the UK diet. Therefore, it is important that all potential impacts (positive and negative) of reduced meat consumption or change in meat type on nutrient intakes and health are thoroughly considered before making recommendations for changes in consumption.

There are many examples of competing pressures in the food system, but there is a potential 'win–win' situation relating to meat consumption and the environment. A recommendation for high consumers of red meat and processed meat to reduce their intakes may have a positive impact on both the environment and their health. Such a recommendation was proposed by the SACN in its draft report on Iron and Health as high consumption of these foods may be associated with some adverse health outcomes such as increased risk of CRC (SACN 2009). However, this message would need to be targeted to high consumers only to prevent already low consumers reducing their intakes, which may increase their risk of inadequate nutrient intakes.

While the current focus for livestock production in the UK is to reduce its impact on the environment, the priority may be different for rapidly developing countries such as China, India and Brazil. In these and in less developed countries, social and economic factors may prevail over concerns about the environment. In addition, from a nutritional perspective, people in developing countries who have low energy and/or protein intakes may actually benefit nutritionally from increasing their intakes of livestock products.

Therefore, the challenge ahead is to find a balance for livestock as part of a healthy and sustainable diet at both a UK and global level. We need robust evidence to weigh decisions and evaluate trade-offs, including evidence on the likely impacts (positive and negative) of dietary changes on nutrition and health. The challenge will then be to communicate tailored, practical and consistent messages to consumers about sustainable eating patterns and food choices.

Discussion

Trends in consumption of red meat vary widely between countries and between subgroups of the population within countries. There has been a general decrease in the amount of red meat consumed in the UK during the last few decades because of an increased preference for white meat such as poultry. Globally, men tend to have higher total and red meat intakes than women.

The average daily intake of red meat among adults in the UK is 96 g for men and 57 g for women. Current expert advice is that individuals' consumption of red and processed meat should not rise and that those consuming large amounts (more than 140 g per day) should consider a reduction in intake (SACN 2009).

There are many challenges when comparing data from different studies because of the lack of a standardised definition of red meat and because different methodologies were used to collect the data. Prospective cohort studies generally provide more meaningful findings than other types of epidemiological studies as there is less chance of bias in reporting of food intakes.

Red meat contains important nutrients including high quality protein, long-chain *n*-3 fatty acids, iron, zinc, selenium, vitamin D and vitamins B_3 and B_{12}. The fatty acid composition of red meat varies according to, for example, the type of meat, the cut and the degree of trimming. Lean red meat contains similar proportions of SFAs and MUFAs and a total fat content of about 5–10 g/100 g. Although meat contains only a small amount of long-chain *n*-3 PUFAs, as there are very few dietary sources apart from oily fish, meat can make a useful contribution to intakes of long-chain *n*-3 PUFAs for those who consume little or no oily fish. Advances in food processing technologies, animal husbandry and butchery techniques over the last 20 years have resulted in a reduction in the fat content of carcass meat by 10–30%. Further reductions in fat content of meat can be made by consumers using preparation and cooking methods such as dry frying or grilling and by trimming the visible fat off meat. Red meat contributes approximately 17% of total dietary iron intake in the UK and is present in the more readily absorbed haem form in red meat. Low iron intakes are common in many developed countries, particularly among women and infants. In the UK, almost 50% of women of childbearing age have iron intakes below the LRNI. Low intakes of zinc are also a concern for some population groups in the UK, such as young girls and infants and children. Red meat contains a substantial amount of zinc, which importantly is available in a form that is readily absorbed by the body. Modelling by SACN suggests that a reduction in meat intake may have an even greater impact on zinc intake than on iron intake. Meat is now of more importance as a source of selenium in the UK and European diets owing to the decreased selenium levels in the wheat consumed (the majority of wheat used to make bread is now European rather than imported from North America, where selenium concentrations are higher). Red meat contains a variety of vitamins, including a range of B vitamins, and in particular, red meat is a *rich source* of vitamin B_3 (niacin) and B_{12} using the categorisation defined in EU labelling legislation. As vitamin B_{12} is only found naturally in foods of animal origin, people who do not consume meat or other animal products may have inadequate intakes. Low vitamin D status is now common in the UK. For those who do not get enough vitamin D through sunlight exposure (the main route for most people), red meat is an important dietary source, as the vitamin D found in meat is thought to be more easily utilised than the vitamin D found in some other foods.

Identifying good, robust scientific evidence to demonstrate effects of particular foods, such as red meat, on risk of diseases (such as cancer and CVD) is challenging. Current evidence suggests a possible link between intake of meat (especially processed meats) and an increased risk of CRC. With respect to CRC risk, the currently available evidence suggests that the UK guidelines set by the Committee on Medical Aspects of Food and Nutrition Policy are still appropriate, which is that individuals' consumption of red and processed meat should not rise and that higher consumers (>140 g per day or 12–14 portions per week) should consider a reduction in intake (Department of Health, UK, 1998). Average daily intakes of red and processed meat in most countries remain below this level. For example, average intakes of red meat in Europe (in men) range from 40 g per day in the UK to 74 g per day in Spain, while average intakes of processed meat (in men) range from 10 g per day in Greece to 83.2 g per day in Germany (see Table 3), and therefore, it is only the small proportion of high consumers of meat and meat products that may need to consider a reduction in consumption.

While some cohort studies have shown increased risk of CVD amongst meat eaters compared with vegetarians or low meat eaters, controlling for the wide range of potential confounders is difficult. Although recent studies have attempted to investigate the specific effects of unprocessed and processed meats on CVD risk, these have provided conflicting results and further research is needed to clarify matters. While meat products are a significant contributor to SFA and sodium intakes, unprocessed lean red meat has a relatively low SFA and sodium content. Intervention studies have indeed demonstrated favourable effects of lean red meat on traditional CVD risk factor such as blood cholesterol levels and blood pressure.

Recent research has, however, suggested that a high consumption of processed meat may increase the risk of developing type 2 diabetes but further research is needed to determine the level of intake associated with higher risk and whether it is the meat *per se* in these products or other ingredients such as pastry or additives used.

Several studies have shown meat eaters to have a higher BMI compared with vegetarians, but it is impossible to attribute this to any individual lifestyle or dietary factor because vegetarians tend to be more health-conscious. The energy content of meat varies widely depending on its fat content, which is influenced by the type of meat, the cut and how much of the fat has been trimmed off. However, lean red meat may be a useful component of weight loss diets because of the satiating effect of its high protein content, although evidence suggests that the energy density of a food or diet may be more important in determining its satiating effect than its protein content *per se*. Whether the protein is from meat or non-meat sources does not appear to have a significant effect on satiety in the context of a normal diet.

With increasing demand for food, increasing pressure on natural resources and the associated impact on the environment, the question of whether meat can be part of a sustainable diet is currently being debated. There are many issues to consider when defining the attributes of a sustainable and healthy diet that has a low impact on the environment. Socio-economic and environmental issues need to be considered along with the nutritional contribution that foods such as lean meat can make to a healthy diet.

Around the world, moderate intake of lean red meat is widely recognised to play an important part in a healthy balanced diet. For example, the UK FSA recommends choosing lean cuts of meat and leaner mince, to cut the fat off meat and to grill meat instead of frying. Consumers should be aware that the type of cut or the meat product chosen and how it is cooked can make a big difference to the fat content. There is no evidence that a moderate intake of lean red meat, when consumed as part of a healthy balanced diet, has any negative health effects.

Conflict of interest

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References

Albert CM, Cook NR, Gaziano JM *et al.* (2008) Effect of folic acid and B vitamins on risk of cardiovascular events and total mortality among women at high risk for cardiovascular disease: a randomized trial. *JAMA* **299**: 2027–36.

Allman-Farinelli MA, Gomes K, Favaloro EJ *et al.* (2005) A diet rich in high-oleic-acid sunflower oil favorably alters low-density lipoprotein cholesterol, triglycerides, and factor VII coagulant activity. *Journal of the American Dietetic Association* **105**: 1071–9.

Alexander DD & Cushing CA (2010) Red meat and colorectal cancer: a critical summary of prospective epidemiologic studies. *Obesity Reviews*: DOI: 10.1111/j.1467-789X.2010.00785.x.

Alexander DD, Miller AJ, Cushing CA, Lowe KA (2010) Processed meat and colorectal cancer: a quantitative review of prospective epidemiological studies. *European Journal Cancer Prevention* **19**: 328–41.

Alvarez Leon EE, Henriquez P & Serra-Majem L. (2006) Mediterranean diet and metabolic syndrome: a cross-sectional study in the Canary Islands. *Public Health Nutrition* **9**: 1089–98.

Appel LJ, Sacks FM, Carey VJ *et al.* (2005) Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA* **294**: 2455–64.

Appleby PN, Davey GK & Key TJ (2002) Hypertension and blood pressure among meat eaters, fish eaters, vegetarians and vegans in EPIC-Oxford. *Public Health Nutrition* **5**: 645–54.

Ashaye A, Gaziano J & Djousse L (2010) Red meat consumption and risk of heart failure in male physicians. *Nutrition, Metabolism, and Cardiovascular Diseases*, DOI: 10.1016/jnumecd.2010.03.009.

Aune D, Ursin G & Veierod MD (2009) Meat consumption and the risk of type 2 diabetes: a systematic review and meta-analysis of cohort studies. *Diabetologia* **52**: 2277–87.

Azadbakht L & Esmaillzadeh A (2009) Red meat intake is associated with metabolic syndrome and the plasma C-reactive protein concentration in women. *Journal of Nutrition* **139**: 335–9.
Babio N, Sorli M, Bullo M *et al.* (2010) Association between red meat consumption and metabolic syndrome in a Mediterranean population at high cardiovascular risk: cross-sectional and 1-year follow-up assessment. *Nutrition, Metabolism, and Cardiovascular Diseases*, DOI: 10.1016/jnumecd.2010.06.11.
Bandera EV, Kushi LH, Moore DF *et al.* (2007) Consumption of animal foods and endometrial cancer risk: a systematic literature review and meta-analysis. *Cancer Causes and Control* **18**: 967–88.
Bates B, Lennox A & Swan G (2010) *National Diet and Nutrition Survey. Headline Results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency/Department of Health: London.
Beauchemin KA, Kreuzer M, O'mara F *et al.* (2008) Nutritional management for enteric methane abatement: a review. *Australian Journal of Experimental Agriculture* **48**: 21–7.
Benelam B (2009) Satiation, satiety and their effects on eating behaviour. *Nutrition Bulletin* **34**: 126–73.
Berkow SE & Barnard N (2006) Vegetarian diets and weight status. *Nutrition Reviews* **64**: 175–88.
Bernstein AM, Sun Q, Hu FB *et al.* (2010) Major dietary protein sources and risk of coronary heart disease in women. *Circulation* **122**: 876–83.
Berti C, Riso P & Porrini M (2008) Satiating properties of meat preparations: role of protein content and energy density. *Journal of the American College of Nutrition* **27**: 244–52.
Bleys J, Miller ER 3rd, Pastor-Barriuso R *et al.* (2006) Vitamin–mineral supplementation and the progression of atherosclerosis: a meta-analysis of randomized controlled trials. *American Journal of Clinical Nutrition* **84**: 880–7. Quiz 954–885.
BNF (British Nutrition Foundation) (1995) *Task Force Report, Iron: Nutritional and Physiological Significance*. British Nutrition Foundation: London.
BNF (British Nutrition Foundation) (2001) *Selenium and Health*. British Nutrition Foundation: London.
Borzoei S, Neovius M & Brakeling B (2006) A comparison of effects of fish and beef protein on satiety in normal weight men. *European Journal of Clinical Nutrition* **60**: 897–902.
Bosetti C, Levi F, Rosato V *et al.* (2010) Recent trends in colorectal cancer mortality in Europe. *International Journal of Cancer*, DOI: 10.1002/ijc.25653.
Bostick RM, Potter JD, Kushi LH *et al.* (1994) Sugar, meat, and fat intake, and non-dietary risk factors for colon cancer incidence in Iowa women (United States). *Cancer Causes and Control* **5**: 38–52.
Boushey CJ, Beresford SA, Omenn GS *et al.* (1995) A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. Probable benefits of increasing folic acid intakes. *JAMA* **274**: 1049–57.
Breslow RA, Graubard BI, Rashmi S *et al.* (2000) Diet and lung cancer mortality: a 1987 National Health Interview Survey cohort study. *Cancer Causes and Control* **11**: 419–31.
British Heart Foundation (BHF) (2010) *UK coronary heart disease statistics 2009–10*. Available at: http://www.bhf.org.uk/publications/view-publication.aspx?ps=1001141
Burke V, Zhao Y, Lee AH *et al.* (2007) Health-related behaviours as predictors of mortality and morbidity in Australian Aborigines. *Preventive Medicine* **44**: 135–42.
Butland B, Jebb S, Kopelman P *et al.* (2007) Foresight Tackling Obesities: Future Choices – Project report. Government Office for Science, Department of Innovation. Universities and Skills: London.
Buttriss J (2005) Taking the science forward: public health implications. In: *Healthy Ageing. The Role of Nutrition and Lifestyle*, (S Stanner, R Thompson, J Buttriss eds), pp. 246–94. Wiley-Blackwell: London.
Cancer Research UK (2010) *High risk groups of bowel cancer*. Available at: http://www.cancerhelp.org.uk/type/bowel-cancer/about/risks/high-risk-groups-for-bowel-cancer (accessed 3 December 2010).
Cao JJ & Niesen FH (2010) Acid diet (high meat protein) effects on calcium metabolism and bone health. *Current Opinion in Clinical Nutrition and Metabolic Care* **13**: 698–702.
Centritto F, Iacoviello L, Di Giuseppe R *et al.* (2009) Dietary patterns, cardiovascular risk factors and C-reactive protein in a healthy Italian population. *Nutrition, Metabolism, and Cardiovascular Diseases* **19**: 697–706.
Chan W, Brown J & Church S (1995) *Meat, Poultry and Game. Supplement to McCance and Widdowson's the Composition of Foods*. MAFF: London.
Chao A, Thun MJ, Connell CJ *et al.* (2005) Meat consumption and risk of colorectal cancer. *JAMA* **293**: 172–82.
Cho E, Chen WY, Hunter DJ *et al.* (2006) Red meat intake and risk of breast cancer among premenopausal women. *Archives of Internal Medicine* **166**: 2253–9.
Clarke R, Daly L, Robinson K *et al.* (1991) Hyperhomocysteinemia: an independent risk factor for vascular disease. *New England Journal of Medicine* **324**: 1149–55.
Clarke R, Halsey J, Lewington S *et al.* (2010) Effects of lowering homocysteine levels with B vitamins on cardiovascular disease, cancer, and cause-specific mortality: meta-analysis of 8 randomized trials involving 37 485 individuals. *Archives of Internal Medicine* **170**: 1622–31.
Clausen I & Ovesen L (2005) Changes in fat content of pork and beef after pan-frying under different conditions. *Journal of Food Composition and Analysis* **18**: 201–11.
Collaboration HS (2002) Homocysteine and risk of ischemic heart disease and stroke: a meta-analysis. *JAMA* **288**: 2015–22.
Cosgrove M, Flynn A & Kiely M (2004) Impact of disaggregation of composite foods on estimate of intakes of meat and meat products in Irish adults. *Public Health Nutrition* **8**: 327–37.
Cosgrove M, Flynn A & Kiely M (2005) Consumption of red meat, white meat and processed meat in Irish adults in relation to dietary quality. *British Journal of Nutrition* **93**: 933–42.
Cross AJ & Sinha R (2004) Meat-related mutagens/carcinogens in the etiology of colorectal cancer. *Environmental and Molecular Mutagenesis* **44**: 44–55.
Cross AJ, Leitzmann MF, Gail MH *et al.* (2007) A prospective study of red and processed meat intake in relation to cancer risk. *PLoS Medicine* **12**: 1973–84.
Cross AJ, Leitzmann MF, Subar AF *et al.* (2008) A prospective study of meat and fat intake in relation to small intestinal cancer. *Cancer Research* **68**: 9274–9.

Cross AJ, Freeman ND, Ren J *et al*. (2010) Meat consumption and risk of esophageal and gastric cancer in a large prospective study. *American Journal of Gastroenterology*, DOI: 10.1038/ajg.2010.415.

Daley CA, Abbott A, Doyle PS *et al*. (2010) A review of fatty acid profiles and antioxidant content in grass-fed and grain-fed beef. *Nutrition Journal* **9**: 10.

van Dam RM, Willet WC, Rimm EB *et al*. (2002) Dietary fat and meat intake in relation to risk of type 2 diabetes in men. *Diabetes Care* **25**: 417–24.

van Dam RM, Grievink L, Ocke MA *et al*. (2003) Patterns of food consumption and risk factors for cardiovascular disease in the general Dutch population. *American Journal of Clinical Nutrition* **77**: 1156–63.

Damiao R, Castro TG & Cardoso MA *et al*. (2006) Dietary intakes associated with metabolic syndrome in a cohort of Japanese ancestry. *British Journal of Nutrition* **96**: 532–8.

Danesh J & Lewington S (1998) Plasma homocysteine and coronary heart disease: systematic review of published epidemiological studies. *Journal of Cardiovascular Risk* **5**: 229–32.

De Stefani E, Boffetta P, Deneo-Pellegrini H *et al*. (2009) Meat intake, meat mutagens and risk of lung cancer in Uruguayan men. *Cancer Causes and Control* **20**: 1635–43.

Department for Environment Food and Rural Affairs (Defra) (2010) *Family Food datasets*. Available at: http://www.defra.gov.uk/evidence/statistics/foodfarm/familyfood/documents/index.htm (accessed 11 January 2011).

Department of Health (1994) *Report on Health and Social Subjects 48. Nutritional Aspects of the Development of Cardiovascular Disease: Report of the Working Group on Diet and Cardiovascular Disease of the Committee on Medical Aspects of Food and Nutrition Policy*. The Stationary Office: London.

Department of Health (1998) *Report on Health and Social Subjects 48. Nutritional Aspects of the Development of Cancer: Report of the Working Group on Diet and Cancer of the Committee on Medical Aspects on Food and Nutrition Policy*. The Stationary Office: London.

Diabetes UK (2010) *Diabetes in the UK 2010: Key Statistics on Diabetes*. Diabetes UK: London.

Dunnigan M & Henderson J (1997) An epidemiological model of privational rickets and osteomalacia. *Proceedings of the Nutrition Society* **56**: 939–56.

Eisentstein J, Roberts SB & Dallal G *et al*. (2002) High- protein weight-loss diets: are they safe and do they work? A review of the experimental and epidemiologic data. *Nutrition Reviews* **60**: 189–200.

Ellul P, Fogden E, Simpson CL *et al*. (2010) Downstaging of colorectal cancer by the national bowel cancer screening programme in England: first round data from the first centre. *Colorectal Disease* **12**: 420–2.

Elmadfa I, Meyer A, Nowak V *et al*. (2009) *European Nutrition and Health Report 2009*. Karger: Basel.

English Beef and Lamb Executive (EBLEX) (2009) *Change in the air: the English beef and sheep production roadmap – phase 1*. Available at: http://www.eblex.org.uk/documents/content/news/p_cp_changeintheairtheenglishbeefandsheepproductionroadmap.pdf (accessed January 2011).

English Beef and Lamb Executive (EBLEX) (2010) *Testing the water: the English beef and sheep production environmental roadmap – phase 2*. Available at: http://www.eblex.org.uk/documents/content/publications/p_cp_testingthewater061210.pdf (accessed 11 January 2011).

English DR, MacInnis RJ, Hodge AM *et al*. (2004) Red meat, chicken, and fish consumption and risk of colorectal cancer. *Cancer Epidemiology, Biomarkers & Prevention* **13**: 1509–14.

Ferlay J, Shin HR & Bray F *et al*. (2010) *GLOBOCAN 2008: Cancer Incidence and Mortality Worldwide: IARC Cancer Base No. 10 [Internet]*. International Agency for Research on Cancer: Lyon.

Fernández-Ginés JM, Fernández-López J, Sayas-Barberá E *et al*. (2005) Meat products as functional foods: a review. *Journal of Food Science* **70**: R37–43.

Ferrucci LM, Cross AJ, Graubard BI *et al*. (2009) Intake of meat, meat mutagens, and iron and the risk of breast cancer in the prostate, lung, colorectal, and ovarian cancer screening trial. *British Journal of Cancer* **101**: 178–84.

Fitt E, Mak TN, Stephen AM *et al*. (2010) Disaggregating composite food codes in the UK national diet and nutrition survey food composition databank. *European Journal of Clinical Nutrition* **64**: S32–6.

Flood A, Velie EM, Sinah R *et al*. (2003) Meat, fat, and their subtypes as risk factors for colorectal cancer in a prospective cohort of women. *American Journal of Epidemiology* **158**: 59–68.

Flores-Mateo G, Navas-Acien A, Pastor-Barriuso R *et al*. (2006) Selenium and coronary heart disease: a meta-analysis. *American Journal of Clinical Nutrition* **84**: 762–73.

Food and Agriculture Organization (FAO) (2009) *The state of food and agriculture: livestock in the balance*. Available at: http://www.fao.org/publications/sofa/en (accessed 11 January 2011).

Food Standards Agency (FSA) (2002) *McCance and Widdowson's the Composition of Foods* Royal Society of Chemistry: Cambridge.

Food Standards Agency (FSA) (2005) *Agency issues new advice on vitamin A to at risk groups*. Available at: http://www.food.gov.uk/news/pressreleases/2005/sep/newvitaminaadvicepress (accessed 29 October 2010).

Food Standards Agency (FSA) (2010) *Food standards agency. Salt campaign site*. Available at: http://www.salt.gov.uk (accessed 15 July 2010).

Forstermann U (2008) Oxidative stress in vascular disease: causes, defense mechanisms and potential therapies. *Nature Clinical Practice. Cardiovascular Medicine* **5**: 338–49.

Fung TT, Willett WC, Stampfer MJ *et al*. (2001) Dietary patterns and the risk of coronary heart disease in women. *Archives of Internal Medicine* **161**: 1857–62.

Fung TT, Schulze MB, Manson JE *et al*. (2004) Dietary patterns, meat intake, and the risk of type 2 diabetes in women. *Archives of Internal Medicine* **164**: 2235–40.

Fung T, van Dam R, Hankinson SE *et al*. (2010) Low-carbohydrate diets and all-cause and cause-specific mortality: two cohort studies. *Annals of Internal Medicine* **153**: 289–98.

Gardner CD, Kim S, Bersamin A *et al*. (2010) Micronutrient quality of weight-loss diets that focus on macronutrients: results from the A to Z study. *American Journal of Clinical Nutrition* **92**: 304–12.

Gerber N, Scheeder MRL & Wenk C (2009) The influence of cooking and fat trimming on the actual nutrient intake from meat. *Meat Science* **81**: 148–54.

Givens DI (2005) The role of animal nutrition in improving the nutritive value of animal-derived foods in relation to chronic disease. *Proceedings of the Nutrition Society* **64**: 395–402.

Givens DI & Shingfield KJ (2004) Foods derived from animals: the impact of animal nutrition on their nutritive value and ability to sustain long-term health. *Nutrition Bulletin* **29**: 325–32.

Gonzalez CA, Jakszyn P, Pera G *et al.* (2006) Meat intake and risk of stomach and esophageal adenocarcinoma within the European Prospective Investigation into Cancer and Nutrition (EPIC). *Journal of the National Cancer Institute* **98**: 345–54.

Greenfield H, Arcot J, Barnes JA *et al.* (2009) Nutrient composition of Australian retail pork cuts 2005/2006. *Food Chemistry* **117**: 721–30.

Gregory J, Lowe S, Bates C *et al.* (2000) *National Diet and Nutrition Survey: Young People Aged 4 to 18 Y*. The Stationery Office: London.

Groff J, Gropper S & Junt S (1995) *Advanced Nutrition and Human Metabolism*. West Publishing Co.: Minneapolis/St Paul, MN.

Guallar-Castillon P, Rodriguez-Artalejo F, Tormo MJ *et al.* (2010) Major dietary patterns and risk of coronary heart disease in middle-aged persons from a Mediterranean country: the EPIC-Spain cohort study. *Nutrition, Metabolism, and Cardiovascular Diseases*, DOI: 10.1016/j.numecd.2010.06.004.

Halton TL & Hu FB (2004) The effects of high protein diets on thermogenesis, satiety and weight loss: a critical review. *Journal of the American College of Nutrition* **23**: 373–85.

Hannan MT, Tucker KL, Dawson-Hughes B *et al.* (2000) Effect of dietary protein on bone loss in elderly men and women: the Framingham Osteoporosis Study. *Journal of Bone and Mineral Research* **15**: 2501–12.

Harland J (2010) Colorectal cancer and red meat: what does the evidence say? *Complete Nutrition* **10**: 46–9.

Harriss LR, English DR, Powles J *et al.* (2007) Dietary patterns and cardiovascular mortality in the Melbourne collaborative cohort study. *The American Journal of Clinical Nutrition* **86**: 221–9.

Heidemann C, Schulze MB, Franco OH *et al.* (2008) Dietary patterns and risk of mortality from cardiovascular disease, cancer, and all causes in a prospective cohort of women. *Circulation* **118**: 230–7.

Henderson L, Gregory J, Irving K *et al.* (2003a) *The National Diet and Nutrition Survey: Adults Aged 19–64 Y. Volume 2 Energy, Protein, Carbohydrate, Fat and Alcohol Intake*. The Stationery Office: London.

Henderson L, Irving K, Gregory J *et al.* (2003b) *The National Diet and Nutrition Survey: Adults Aged 19–64 Y. Volume 3 Vitamin and Mineral Intake and Urinary Analytes*. The Stationery Office: London.

Higgs JD (2000) The changing nature of red meat: 20 y of improving nutritional quality. *Trends in Food Science & Technology* **11**: 85–95.

HM Government (2009) *The UK Low Carbon Transition Plan*. Available at: http://www.decc.gov.uk/assets/decc/White%20Papers/UK%20Low%20Carbon%20Transition%20Plan%20WP09/1_20090724153238_e_@@_lowcarbontransitionplan.pdf (accessed 11 January 2011).

Hoare J, Henderson L, Bates CJ *et al.* (2004) *The National Diet and Nutrition Survey: Adults Aged 19 to 64 Y. Summary Report*. TSO: London.

Hodge AM, English DR, O'Dea K *et al.* (2007) Dietary patterns and diabetes incidence in the Melbourne collaborative cohort study. *American Journal of Epidemiology* **165**: 603–10.

Hodgson JM, Ward NC, Burke V *et al.* (2007) Increased lean red meat intake does not elevate markers of oxidative stress and inflammation in humans. *Journal of Nutrition* **137**: 363–7.

Holben DH & Smith AM (1999) The diverse role of selenium within selenoproteins: a review. *Journal of the American Dietetic Association* **99**: 836–43.

Homocysteine Lowering Trialists' Collaboration (2005) Dose-dependent effects of folic acid on blood concentrations of homocysteine: a meta-analysis of the randomized trials. *American Journal of Clinical Nutrition* **82**: 806–12.

Hsia J, Heiss G, Ren H *et al.* (2007) Calcium/vitamin D supplementation and cardiovascular events. *Circulation* **115**: 846–54.

Hsing AW, McLaughlin JK, Chow WH *et al.* (1998) Risk factors for colorectal cancer in a prospective study among U.S. white men. *International Journal of Cancer* **77**: 549–53.

Hu FB, Rimm EB, Stampfer MJ *et al.* (2000) Prospective study of major dietary patterns and risk of coronary heart disease in men. *American Journal of Clinical Nutrition* **72**: 912–21.

Hughes H, Arrowsmith H & Mulvey L (2010) *Reducing the Fat Content of Meat Products: A Review of Fat Replacement Ingredients. Review No.65*. Campden BRI: Gloucestershire.

Hunter JE, Zhang J & Kris-Etherton PMK (2010) Cardiovascular disease risk of dietary stearic acid compared with *trans*, other saturated, and unsaturated fatty acids: a systematic review. *American Journal of Clinical Nutrition* **91**: 46–63.

Iso H, Stampfer MJ, Manson JE *et al.* (2001) Prospective study of fat and protein intake and risk of intraparenchymal hemorrhage in women. *Circulation* **103**: 856–63.

Järvinen R, Knekt P, Hakulinen T *et al.* (2001) Dietary fat, cholesterol and colorectal cancer in a prospective study. *British Journal of Cancer* **85**: 357–61.

Johnson JM & Walker PM (1992) Zinc and iron utilization in young women consuming a beef-based diet. *Journal of the American Dietetic Association* **92**: 1474–8.

Johnstone AM, Horgan GW, Murison SD *et al.* (2008) Effects of a high-protein ketogenic diet on hunger, appetite and weight loss in obese men feeding *ad libitum*. *American Journal of Clinical Nutrition* **87**: 44–55.

Kabat GC, Miller AB, Jain M *et al.* (2007) A cohort study of dietary iron and heme iron intake and risk of colorectal cancer in women. *British Journal of Cancer* **97**: 118–22.

Kabat GC, Miller AB, Jain M *et al.* (2008) Dietary iron and haem iron intake and risk of endometrial cancer: a prospective cohort study. *British Journal of Cancer* **98**: 194–8.

Kato I, Akhmedkhanov A, Koenig K *et al.* (1997) Prospective study of diet and female colorectal cancer: the New York University Women's Health Study. *Nutrition and Cancer* **28**: 276–81.

Kelemen LE, Kushi LH, Jacobs DR *et al.* (2005) Associations of dietary protein with disease and mortality in a prospective study of postmenopausal women. *American Journal of Epidemiology* **161**: 239–49.

Kelly CN & Stanner SA (2003) Diet and cardiovascular disease in the UK: are the messages getting across? *Proceedings of the Nutrition Society* **62**: 583–9.

Kestin M, Rouse IL, Correll RA *et al.* (1989) Cardiovascular disease risk factors in free-living men: comparison of two prudent diets, one based on lactoovovegetarianism and the other allowing lean meat. *American Journal of Clinical Nutrition* **50**: 280–7.

Key TJ, Allen NE, Spencer EA *et al.* (2002) The effect of diet on risk of cancer. *The Lancet* **360**: 861–8.

Key TJ, Fraser GE, Thorogood M *et al.* (1998) 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* **1**: 33–41.

Key TJ, Fraser GE, Thorogood M *et al.* (1999) Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *American Journal of Clinical Nutrition* **70** (Suppl. 3): 516S–24S.

Kirk JK, Graves DE, Graven TE *et al.* (2008) Restricted-carbohydrate diets in patients with type 2 diabetes: a meta-analysis. *Journal of the American Dietetic Association* **108**: 91–100.

Kontogianni MD, Panagiotakos DB, Pitsavos C *et al.* (2008) Relationship between meat intake and the development of acute coronary syndromes: the CARDIO2000 case-control study. *European Journal of Clinical Nutrition* **62**: 171–7.

Lam TK, Cross AJ, Consonni D *et al.* (2009) Intakes of red meat, processed meat, and meat mutagens increase lung cancer risk. *Cancer Research* **69**: 932–9.

Larsson SC, Rafter J, Holmberg L *et al.* (2005) Red meat consumption and risk of cancers of the proximal colon, distal colon and rectum: the Swedish mammography cohort. *International Journal of Cancer* **113**: 829–34.

Larsson SC, Orsini N & Wolk A (2006) Processed meat consumption and stomach cancer risk: a meta-analysis. *Journal of the National Cancer Institute* **98**: 1078–87.

Larsson SC & Wolk A (2006) Meat consumption and risk of colorectal cancer: a meta-analysis of prospective studies. *International Journal of Cancer* **119**: 2657–64.

Larsson SC, Bergkvist L & Wolk A (2009) Long-term meat intake and risk of breast cancer by oestrogen and progesterone receptor status in a cohort of Swedish women. *European Journal of Cancer* **45**: 3042–6.

Lee DH, Folson AR & Jacobs DR (2004) Dietary iron intake and type 2 diabetes incidence in postmenopausal women: the Iowa Women's Health study. *Diabetologia* **47**: 185–94.

Lee DH, Folsom AR & Jacobs DR (2005) Iron, zinc, and alcohol consumption and mortality from cardiovascular diseases: the Iowa Women's Health Study. *American Journal of Clinical Nutrition* **81**: 787–91.

Li D, Siriamornpun S, Wahlqvist ML *et al.* (2005) Lean meat and heart health. *Asia Pacific Journal of Clinical Nutrition* **14**: 113–9.

Linseisen J, Kesse E, Slimani N *et al.* (2002) Meat consumption in the European prospective investigation into cancer and nutrition (EPIC) cohorts: results from 24-hour dietary recalls. *Public Health Nutrition* **5**: 1243–58.

Lunn J & Theobald HE (2006) The health effects of dietary unsaturated fatty acids. *Nutrition Bulletin* **31**: 178–224.

Malaviarachchi D, Veugelers PJ, Yip AM *et al.* (2002) Dietary iron as a risk factor for myocardial infarction. Public health considerations for Nova Scotia. *Canadian Journal of Public Health* **93**: 267–70.

Manger MS, Strand E, Ebbing M *et al.* (2010) Dietary intake of n-3 long-chain polyunsaturated fatty acids and coronary events in Norwegian patients with coronary artery disease. *The American Journal of Clinical Nutrition* **92**: 244–51.

Mannisto S, Kontto J, Kataja-Tuomola M *et al.* (2010) High processed meat consumption is a risk factor of type 2 diabetes in the alpha-tocopherol, beta-carotene cancer prevention study. *British Journal of Nutrition* **103**: 1817–22.

Matthews K & Strong M (2005) Salt – its role in meat products and the industry's action plan to reduce it. *Nutrition Bulletin* **30**: 55–61.

Micha R, Wallace SK, Mozaffarian D *et al.* (2010) Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. *Circulation* **121**: 2271–83.

Miles L & Caswell H (2008) Advancing beef safety and quality: ProSafeBeef. *Nutrition Bulletin* **33**: 140–4.

Millward DJ & Garnett T (2010) Food and the planet: nutritional dilemmas of greenhouse gas emission reductions through reduced intakes of meat and dairy foods. *Proceedings of the Nutrition Society* **69**: 103–18.

Ministry of Agriculture Fisheries and Food (MAFF) (1995) *Manual of Dietetic Nutrition*. HMSO: London.

Ministry of Agriculture Fisheries and Food (MAFF) (1997) *1997 total diet study – aluminium, arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, tin and zinc*. Food Surveillance Information Sheet no. 191.

Ministry of Agriculture Fisheries and Food (MAFF) (1998) *Fatty acids. Supplement to McCance and Wiidowson's the comparison of foods*. The Royal Society of Chemistry: Cambridge and the Ministry of Agriculture, Fisheries and Food: London.

Moloney AP, Scollan ND & Miles L (2008) Enrichment of n-3 fatty acids and conjugated linoleic acid in beef: ProSafeBeef. *Nutrition Bulletin* **33**: 374–81.

Montonen J, Jarvinen R, Heliovaara M *et al.* (2005) Food consumption and the incidence of type II diabetes mellitus. *European Journal of Clinical Nutrition* **59**: 441–8.

Mozaffarian D & Rimm EB (2006) Fish intake, contaminants, and human health: evaluating the risks and the benefits. *JAMA* **296**: 1885–99.

Müller WJ, Bruce HL, Tume RK *et al.* (2009) National sampling and gross composition of Australian retail pork cuts 2005/2006. *Meat Science* **81**: 626–31.

Mulvihill B (2001) Ruminant meat as a source of conjugated linoleic acid (CLA). *Nutrition Bulletin* **26**: 295–9.

Muñoz MVI, Vilar I & García-Erce JA (2009) An update on iron physiology. *World Journal of Gastroenterology* **15**: 4617–26.

National Health Service (2010) *NHS bowel cancer screening programme*. Available at: http://www.cancerscreening.nhs.uk/bowel/about-bowel-cancer-screening.html (accessed 2 December 2010).

Navas-Acien A, Bleys J & Guallar E (2008) Selenium intake and cardiovascular risk: what is new? *Current Opinion in Lipidology* **19**: 43–9.

Nelson M, Beresford SAA & Kearney JM (2004) Nutritional epidemiology. In: *Public Health Nutrition*, (MJ Gibney, BM Margetts, JM Kearney eds), pp. 26–65. Blackwell Science: Oxford.

Norat T, Bingham S, Ferrari P *et al.* (2005) Meat, fish, and colorectal cancer risk: the European Prospective Investigation into cancer and nutrition. *Journal of the National Cancer Institute* **97**: 906–16.

Nordmann AJ, Nordmann A, Briel M *et al.* (2006) Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors. A meta-analysis of randomized controlled trials. *Archives of Internal Medicine* **166**: 285–93.

Nowson CA, Wattanapenpaiboon N & Pachett A (2009) Low-sodium dietary approaches to stop hypertension-type diet including lean red meat lowers blood pressure in postmenopausal women. *Nutrition Research* **29**: 8–18.

O'Dea K, Traianedes K, Chisholm K *et al.* (1990) Cholesterol-lowering effect of a low-fat diet containing lean beef is reversed by the addition of beef fat. *The American Journal of Clinical Nutrition* **52**: 491–4.

Olmedilla-Alonso B, Granado-Lorencio F, Herrero-Barbudo C *et al.* (2008) Consumption of restructured meat products with added walnuts has a cholesterol-lowering effect in subjects at high cardiovascular risk: a randomised, crossover, placebo-controlled study. *Journal of the American College of Nutrition* **27**: 342–8.

Paddon-Jones D, Westman E, Mattes RD *et al.* (2008) Protein, weight management, and satiety. *American Journal of Clinical Nutrition* **87**: 1558S–61S.

Pala V, Krogh V, Berrino F *et al.* (2009) Meat, eggs, dairy products, and risk of breast cancer in the European prospective investigation into cancer and nutrition (EPIC) cohort. *American Journal of Clinical Nutrition* **90**: 602–12.

Panagiotakos D, Pitsavos C, Chrysohoou C *et al.* (2009) Dietary patterns and 5-year incidence of cardiovascular disease: a multivariate analysis of the ATTICA study. *Nutrition, Metabolism, and Cardiovascular Diseases* **19**: 253–63.

Parkin DM (2002) The global health burden of infection-associated cancers in the year 2002. *International Journal of Cancer* **118**: 3030–44.

Phillips F (2005) Vegetarian nutrition. *Nutrition Bulletin* **30**: 132–67.

Pietinen P, Malila N, Virtanen M *et al.* (1999) Diet and risk of colorectal cancer in a cohort of Finnish men. *Cancer Causes and Control* **10**: 387–96.

Preis SR, Stampfer MJ, Spiegelman D *et al.* (2010) Dietary protein and risk of ischemic heart disease in middle-aged men. *The American Journal of Clinical Nutrition* **92**: 1265–72.

Promislow JH, Goodman-Gruen D, Slymen DJ *et al.* (2002) Protein consumption and bone mineral density in the elderly: the Rancho Bernardo Study. *American Journal of Epidemiology* **155**: 636–44.

Prynne C, Wagemakers J, Stephen A *et al.* (2009) Meat consumption after disaggregation of meat dishes in a cohort of British adults in 1989 and 1999 in relation to dietary quality. *European Journal of Clinical Nutrition* **63**: 660–6.

Raben A, Agerholm-Larsen L, Flint A *et al.* (2003) Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism, but not on appetite and energy intake. *American Journal of Clinical Nutrition* **77**: 91–100.

Red Meat and Health Expenditure Advisory Committee (2001) *The role of red meat in healthy Australian diets*. Report prepared by Red Meat and Health Expenditure Advisory Committee.

Riley H (2010) NDNS rolling programme – what do the year 1 results show? *Nutrition Bulletin* **35**: 235–9.

Roughead ZK, Johnson LK & Lykken GI (2003) Controlled high meat diets do not affect calcium retention or indices of bone status in healthy postmenopausal women. *Journal of Nutrition* **133**: 1020–6.

Sauvaget C, Nagano J, Hayashi M *et al.* (2004) Animal protein, animal fat, and cholesterol intakes and risk of cerebral infarction mortality in the adult health study. *Stroke* **35**: 1531–7.

Schulze MB, Mason JE, Wilet WC *et al.* (2003) Processed meat intake and incidence of type 2 diabetes in younger and middle-aged women. *Diabetologia* **46**: 1465–73.

Schulze MB, Hoffman K & Boeing H (2007) An accurate risk score based on anthropometric, dietary, and lifestyle factors to predict the development of type 2 diabetes. *Diabetes Care* **30**: 510–5.

Scientific Advisory Committee on Nutrition (SACN) (2003) *Salt and Health*. The Stationary Office: London.

Scientific Advisory Committee on Nutrition (SACN) (2004) *Advice on fish consumption: benefits and risks*. Available at: http://www.food.gov.uk/multimedia/pdfs/fishreport2004full.pdf (accessed 2 November 2010).

Scientific Advisory Committee on Nutrition (SACN) (2007a) *Update on trans fatty acids and health. Position statement by the scientific advisory committee on nutrition*. Available at: http://www.sacn.gov.uk/pdfs/sacn_trans_fatty_acids_report.pdf (accessed 2 November 2010).

Scientific Advisory Committee on Nutrition (SACN) (2007b) *Update on vitamin D: position statement by the scientific advisory committee on nutrition*. Available at: http://www.sacn.gov.uk/pdfs/sacn_position_vitamin_d_2007_05_07.pdf (accessed 15 July 2010).

Scientific Advisory Committee on Nutrition (SACN) (2008) *The Nutritional wellbeing of the British population*. The Stationary Office: London.

Scientific Advisory Committee on Nutrition (SACN) (2009) *Draft SACN report on iron and health*. Available at: http://www.sacn.gov.uk/reports_position_statements/reports/draft_iron_and_health_report_scientific_consultation_-_june_2009.html (accessed 1 November 2010).

Scientific Advisory Committee on Nutrition (SACN) (2010) *Iron report, draft February 2010*. Available at: http://www.sacn.gov.uk/meetings/committee/main_sacn_meetings/24022010.html (accessed 11 January 2011).

Sellmeyer DE, Stone KL, Sebastain A *et al.* (2001) A high ratio of dietary animal to vegetable protein increases the rate of bone loss and the risk of fracture in postmenopausal women. *American Journal of Clinical Nutrition* **72**: 118–22.

Sinclair AJ, Barone S, Stoaus T *et al.* (2010) Lipid composition of Australian pork cuts 2005/2006. *Food Chemistry* **121**: 672–81.

Singh PN & Fraser GE (1998) Dietary risk factors for colon cancer in a low-risk population. *American Journal of Epidemiology* **148**: 761–74.

Sinha R, Cross AJ, Graubard BI *et al.* (2009) Meat intake and mortality: a prospective study of over half a million people. *Archives of Internal Medicine* **169**: 562–71.

Sjogren P, Becker W, Waresjo E *et al.* (2010) Mediterranean and carbohydrate-restricted diets and mortality among elderly men: a cohort study in Sweden. *American Journal of Clinical Nutrition* **92**: 967–74.

Song Y, Manson JE, Burint JE *et al.* (2004) A prospective study of red meat consumption and type 2 diabetes in middle-aged and elderly women: the women's health study. *Diabetes Care* **27**: 2108–15.

Souberbielle J-C, Body J-J, Lappe JM *et al.* (2010) Vitamin D and musculoskeletal health, cardiovascular disease, autoimmunity and cancer: recommendations for clinical practice. *Autoimmunity Reviews* **9**: 709–15.

Spencer EA, Key TJ, Appleby PN *et al.* (2010) Meat, poultry and fish and risk of colorectal cancer: pooled analysis of data from the UK dietary cohort consortium. *Cancer Causes and Control* **21**: 1417–25.

Stanner S, ed. (2005) *British Nutrition Task Force Report on Cardiovascular Disease: Diet, Nutrition and Emerging Risk Factors*. Blackwell Science: Oxford.

Stanner S, Thompson R & Buttriss JL (2009) *Healthy Ageing: The Role of Nutrition and Lifestyle*. British Nutrition Foundation: London.

Steffen LM, Kroenke CH, Yu X *et al.* (2005) Associations of plant food, dairy product, and meat intakes with 15-y incidence of elevated blood pressure in young black and white adults: the coronary artery risk development in young adults (CARDIA) study. *The American Journal of Clinical Nutrition* **82**: 1169–77. Quiz 1363–1164.

Steinbrecher A, Erber E, Grandinetti A *et al.* (2010) Meat consumption and risk of type 2 diabetes: the multiethnic cohort. *Public Health Nutrition* **13**: 1–7.

Szeto YT, Kwok TC & Benzie IF (2004) Effects of a long-term vegetarian diet on biomarkers of antioxidant status and cardiovascular disease risk. *Nutrition* **20**: 863–6.

Tabatabaei SM, Heyworth JS, Knuiman MW *et al.* (2010) Dietary benzo[a]pyrene intake from meat and the risk of colorectal cancer. *Cancer Epidemiology, Biomarkers & Prevention* **14**: 2030–4.

Talbot G (2006) *Independent Advice on Possible Reductions of Saturated Fat in Products That Contribute to Consumer Intakes. Summary Report*. UK Food Standards Agency: London.

Taylor EF, Burley VJ, Greenwood DC *et al.* (2007) Meat consumption and risk of breast cancer in the UK Women's Cohort study. *British Journal of Cancer* **96**: 1139–46.

Teixeira Rde C, Molina Mdel C, Zandonade E *et al.* (2007) Cardiovascular risk in vegetarians and omnivores: a comparative study. *Arquivos Brasileiros de Cardiologia* **89**: 237–44.

The European Parliament and the Council of the European Union (2007) Regulation (EC) No 1924/2006 of the European parliament and of the Council of 20 December 2006 on nutrition and health claims made on food. *Official Journal of the European Union* **L12**: 3–18.

Truswell AS (2009) Problems with red meat in the WCRF2. *American Journal Clinical Nutrition* **89**: 1274–9.

Uhe AM, Collier GR & O'Dea K (1992) A comparison of the effects of beef, chicken and fish protein on satiety and amino acid profiles in lean male subjects. *Journal of Nutrition* **122**: 467–72.

Vang A, Singh PN, Lee JW *et al.* (2008) Meats, processed meats, obesity, weight gain and occurrence of diabetes among adults: findings from Adventist health studies. *Annals of Nutrition and Metabolism* **52**: 96–104.

Villegas R, Shu XO, Gao Y-T *et al.* (2006) The association of meat intake and the risk of type 2 diabetes may be modified by body weight. *International Journal of Medical Sciences* **3**: 152–9.

Wagemakers JJ, Prynne CJ, Stephen AM *et al.* (2009) Consumption of red or processed meat does not predict risk factors for coronary heart disease; results from a cohort of British adults in 1989 and 1999. *European Journal of Clinical Nutrition* **63**: 303–11.

Wang Y, Beydoun M, Caballero B *et al.* (2010) Trends and correlates in meat consumption patterns in the US adult population. *Public Health Nutrition* **13**: 1333–45.

Wei EK, Giovannucci E, Wu K *et al.* (2004) Comparison of risk factors for colon and rectal cancer. *International Journal of Cancer* **108**: 433–42.

Westerterp-Plantenga MS, Smeets A & Nieuwenhuizen A (2007) Sustained protein intake for bodyweight management. *Nutrition Bulletin* **32** (Suppl. 1): 22–31.

Whiteman D, Muir J, Jones L*et al.* (1999) Dietary questions as determinants of mortality: the OXCHECK experience. *Public Health Nutrition* **2**: 477–87.

Williamson CS, Foster RK, Stanner SA *et al.* (2005) Red meat in the diet. *Nutrition Bulletin* **30**: 323–55.

Wood JD, Enser M, Fisher A *et al.* (1999) Manipulating meat quality and composition. *Proceedings of the Nutrition Society* **58**: 363–70.

Wood JD, Richardson RI, Nute GR *et al.* (2003) Effects of fatty acids on meat quality: a review. *Meat Science* **66**: 21–32.

World Cancer Research Fund and American Institute for Cancer Research (2007) *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*. American Institute for Cancer Research: Washington, DC.

World Health Organization (2000) *Obesity: Preventing and Managing the Global Epidemic*. Report of a WHO consultation. World Health Organization: Geneva.

Wyness L, Buttriss J & Stanner S (2011) Reducing the population's sodium intake: the food standard agency's salt reduction programme. *Public Health Nutrition* (in press).

Yokoyama M, Origasa H, Matsuzaki M *et al.* (2007) Effects of eicosapentaenoic acid on major coronary events in hypercholesterolaemic patients (JELIS): a randomised open-label, blinded endpoint analysis. *Lancet* **369**: 1090–8.

Zemel MB (1988) Calcium utilization: effect of varying level and source of dietary protein. *American Journal of Clinical Nutrition* **48**: 880–3.

Zittermann A, Schleithoff SS & Koerfer R (2005) Putting cardiovascular disease and vitamin D insufficiency into perspective. *British Journal of Nutrition* **94**: 483–92.

