



## Review

## Red meat consumption: An overview of the risks and benefits

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## ABSTRACT

Red meat is long established as an important dietary source of protein and essential nutrients including iron, zinc and vitamin B12, yet recent reports that its consumption may increase the risk of cardiovascular disease (CVD) and colon cancer have led to a negative perception of the role of red meat in health. The aim of this paper is to review existing literature for both the risks and benefits of red meat consumption, focusing on case–control and prospective studies. Despite many studies reporting an association between red meat and the risk of CVD and colon cancer, several methodological limitations and inconsistencies were identified which may impact on the validity of their findings. Overall, there is no strong evidence to support the recent conclusion from the World Cancer Research Fund (WCRF) report that red meat has a convincing role to play in colon cancer. A substantial amount of evidence supports the role of lean red meat as a positive moderator of lipid profiles with recent studies identifying it as a dietary source of the anti-inflammatory long chain (LC) *n*–3 PUFAs and conjugated linoleic acid (CLA). In conclusion, moderate consumption of lean red meat as part of a balanced diet is unlikely to increase risk for CVD or colon cancer, but may positively influence nutrient intakes and fatty acid profiles, thereby impacting positively on long-term health.

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## 1. Introduction

Red meat forms part of the habitual balanced diet for many adults living in the UK and Ireland (Cosgrove, Flynn, & Kiely, 2004; Henderson & Gregory, 2002). It is recognised that over many years of evolution, humans have adapted to consuming large quantities of lean red meat (Mann, 2000). Recently, a number of epidemiological studies have associated red and processed meat consumption with the development of two of the major chronic diseases in the Western world; CVD and colon cancer (Cross et al., 2007; Giovannucci et al., 1994; Kelemen, Kushi, Jacobs, & Cerhan, 2005; Kontogianni, Panagiotakos, Pitsavos, Chrysohoou, & Stefanadis, 2008). Constituents of red meat that have been proposed to be responsible for these associations include the fat content, fatty acid composition and the possible formation of carcinogenic compounds, such as heterocyclic amines (HCAs), by cooking meat at high temperatures (Bingham, Hughes, & Cross, 2002). Although there are many studies documenting these associations, results are not always consistent and there are several methodological issues which could limit their findings. In the same way as the risks to health of red meat consumption must be evaluated, there are many health benefits which are equally as important in establishing public health messages in relation to red meat consumption. This review will examine current literature on both the risks and benefits associated with red meat consumption, with a particular focus given to the fatty acid composition of red meat as it plays a role in both arguments for risks and benefits.

## 2. Meat intakes in the UK and Ireland

Meat continues to be an important food group in the diet for many consumers, particularly in the developed world (Delgado, 2003; Rosegrant, Leach, & Gerpacio, 1999; Speedy, 2003). Many factors such as wealth, volume of livestock production and socio-economic status of consumers could explain the higher consumption pattern of meat by Western populations (Mann, 2000; Speedy, 2003). Other factors influencing meat consumption include sex, age, religion, body mass index (BMI) and total energy intake, as reported by the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort (Linseisen et al., 2002).

In the UK and Ireland, men and women's average daily intakes of total meat are 108 g and 72 g and 168 g and 107 g, respectively (Linseisen et al., 2002; Cosgrove, Flynn, & Kiely, 2005). Total meat can be broken down into red meat (including beef, lamb, veal and pork), white meat (including chicken, game and turkey) and processed meat (including cured and smoked meats; ham, bacon, sau-

sages, hamburgers, salami and tinned meat) (Linseisen et al., 2002). For the purpose of this paper, the mention of red meat from here on will refer only to red meat which is unprocessed. Data from the North South Ireland Food Consumption Survey (NSIFCS) show that red meat is consumed by 88% of the Irish population, who have slightly higher intakes of beef (39.1 g/d) than of lamb (22.8 g/d) (Cosgrove et al., 2004). According to this survey, men tend to eat more beef (46.8 g/d vs. 30.5 g/d) and lamb (28.1 g/d vs. 16.9 g/d) than women. This is a trend also observed within many European countries as measured by the EPIC study and within Australia, as measured by the Australian National Dietary Survey, which found adult men to consume 88 g/d red meat compared to 45 g/d by women (Baghurst, 1999). Table 1 shows mean daily intakes of total, red and processed meats among consumers of Ireland, the UK and several European countries as measured by NSIFCS and EPIC, respectively. The EPIC study presents total, red and processed meat consumption data for 10 European countries which, owing to its standard method of dietary assessment by the 24 h recall, makes it a useful dataset for comparing intakes by country (Riboli et al., 2002). It can be seen that the UK has the lowest mean daily intake of red meat compared to other European countries, which is of interest considering that Mediterranean countries have long been recognised for their lower incidence of colon cancer and CVD than northern countries (Helsing, 1995). Consumers in Ireland have greater daily intakes of red meat than the UK, which are still lower than of Denmark and Spain.

Earlier UK guidelines set by the Committee on Medical Aspects of Food Policy (COMA) recommended that intakes of red and processed meat should not rise and that individuals with higher intakes (140 g/d) ought to consider a reduction (DoH, 1991). The World Cancer Research Fund (WCRF) 1997 report recommended that red meat intakes should be no more than 80 g/d, of which very little should be processed (WCRF, 1997). After 10 years however, the most recent report lowered this limit to 71 g/d or 500 g red meat per week and further emphasised that intakes of processed meat should be avoided completely (WCRF, 2007).

There is difficulty in accurately measuring meat intakes, since in the modern world meat is typically consumed as part of a composite meal, containing various non-meat components such as vegetables, pasta, legumes or potatoes (Cosgrove et al., 2004). Recently, it was found that earlier assessment of total meat intakes failed to account for the weight of non-meat components of meat dishes and products resulting in a 43% overestimation of total meat intakes by the NSIFCS and a 32% overestimation by the National Survey of Health and Development (NSHD) (Cosgrove et al., 2004; Prynne, Wagemakers, Stephen, & Wadsworth, 2009). Furthermore, the National Food Survey (NFS) will also have over estimated total meat intakes, owing to an assessment of meat that is purchased and not consumed (DEFRA, 2005). Disaggregating composite meat containing meals showed that a large majority of the Irish (88%) and UK (80–90%) meat-eating population consume less than 71 g/d red meat, which could suggest that there are fewer people at risk from poor health associated with over consumption than previously recognised by the WCRF (Cosgrove et al., 2004; Henderson & Gregory, 2002).

Moreover, a trend for declining intakes of red meat in the UK over the last 20 years has been reported (Robinson, 2002). Public opinion of the potential adverse effects of red meat on the risk of CVD and colon cancer, together with public concerns over beef safety stemming from the bovine spongiform encephalopathy (BSE) outbreak might have resulted in a lack of consumer trust in red meat (Verbeke, Frewer, Scholderer, & De Brabander, 2007). Interestingly, since meat intakes have been declining, the incidence of colon cancer in the UK has been increasing significantly, contradicting major reports that red meat consumption is a significant cause of colon cancer (Hill, 2002).

**Table 1**  
Mean daily intakes of total, red and processed meat (g/d) by men and women as measured in several European countries.

Country	Total meat <sup>a</sup>		Red meat <sup>b</sup>		Processed meat <sup>c</sup>	
	Men	Women	Men	Women	Men	Women
UK <sup>d</sup>	108.1	72.3	40	24.6	38.4	22.3
Ireland <sup>e</sup>	167.9	106.6	63.9	37.5	30.9	19.9
Greece <sup>d</sup>	78.8	47.1	45.3	25.5	10	5.8
Spain <sup>d</sup>	170.4	99.2	74	37.8	52.8	29.6
Germany <sup>d</sup>	154.6	84.3	52.2	28.6	83.2	40.9
Italy <sup>d</sup>	140.1	86.1	57.8	40.8	33.5	19.6
Denmark <sup>d</sup>	141.1	88.3	69.6	44.1	51.9	25.3
Netherlands <sup>d</sup>	155.6	92.7	63.8	41.1	72.4	37.9

<sup>a</sup> Total meat: pork, beef, veal, lamb/mutton, poultry, game, rabbit, horse, goat, offal and processed meat.

<sup>b</sup> Red meat: beef, veal, pork and lamb/mutton.

<sup>c</sup> Processed meat: ham, bacon, processed meat cuts, minced meat and sausages.

<sup>d</sup> Source: Linseisen et al. (2002).

<sup>e</sup> Source: Cosgrove et al. (2005).

**Table 2**

Summary of prospective, cohort and case-control studies investigating the associations between meat (red &amp; processed) and risk of CVD and colon cancer.

Author (year)	Study, country	Subjects (n)	Sex (age range)	Type of meat studied	Cases vs. non-cases (n)	Significance (high vs. low quintiles)	Outcome examined	Relative risk/hazard/odds ratios (C.I.)	Variables controlled for	Potential limitations of study
<i>Cardiovascular disease, non-significant findings for red meat</i>										
Hu et al. (1999a)	Nurses Health Study (NHS), prospective study, USA	121,700	f (30–55)	Red meat	~	NS	CHD risk	1.09 (0.91–1.3)	Age, BMI, SM, menopausal status, FH, vitamin E use, AL, aspirin, vigorous PA	Red and processed meats grouped together
Key et al. (1998)	Five prospective studies from USA, UK, Germany	76,172	m & f (16–89)	Non-vegetarians vs. vegetarians	625 vs. 1530	NS	Ischaemic heart disease mortality	0.76 (0.62–0.94)	BMI, AL, education, PA, SM	Not possible to analyse food/nutrient consumption between studies, so findings cannot be related to any particular food group
<i>Cardiovascular disease, significant findings for red meat</i>										
Azadbakht and Esmailzadeh (2008)	Cross-sectional study, Iran	482	f (40–60)	Red meat	39 vs. 22	Sig	Metabolic syndrome risk	2.15 (1.18–4.01)	Age, PA, EI, HRT, menopausal status, FH, intakes of fibre, F&V, white meats, fish, dairy, vegetable oils, whole & refined grains	Red and processed meats grouped together; potential bias with dietary recall
Kontogianni et al. (2008)	CARDIO2000 case-control study, Greece	848 <sup>a</sup>	m & f	Red meat	~	Sig	Acute coronary syndrome risk	4.79 (~)	BMI, SM, PA, education, FH, medication	Case-control study prone to misreporting of dietary data; no definition of red meat
Heidemann et al. (2008)	NHS, USA	121,700	f (34–59)	Western dietary pattern	254 vs. 208	Sig	CVD mortality	1.22 (1.01–1.48)	Age, BMI, PA, EI, SM, HRT, history of hypertension, supplement use	Finding is for dietary pattern only, does not isolate red meat
Keleman et al. (2005)	Iowa Women's Health Study, cohort study, USA	29,017	f	Red meat	739	Sig	CHD mortality	1.44 (1.06–1.94)	Age, EI, PA, BMI, HRT use, supplement use, education, FH	Red and processed meats grouped together; potential error in dietary assessment
Steffen et al. (2005)	Coronary Artery Risk Development in Young Adults (CARDIA) Study, prospective study, USA	5115	m & f (18–30)	Red & processed meat	246 vs. 139	Sig	Elevated blood pressure	1.39 (1.05–1.82)	Age, sex, race, EI, education, PA, AL, SM, supplement use	Red & processed meat grouped together; dietary data collected only twice over 15 y
Fraser (1999)	California Seventh day Adventists study, USA	34,192	m & f (>25)	Beef	~	Sig	Ischaemic heart disease risk	2.31 (1.11–4.28)	Age, sex	Significant finding for men only
<i>Colon cancer, non-significant findings for red meat</i>										
Kimura et al. (2007)	Fukoka Colorectal Cancer case-control Study, Japan	782 <sup>a</sup>	m & f (20–74)	Red Processed	166 vs. 154 170 vs. 152	NS NS	Colorectal cancer risk	1.14 (0.81–1.61) 1.15 (0.83–1.6)	Age, sex, residential area, BMI, FH, SM, AL, occupation, PA, intake of calcium & fibre	Red and processed meats grouped together
Shin et al. (2007)	Tennessee Colorectal Polyp case-control Study	1028 <sup>a</sup>	m & f (40–75)	Red Processed	159 vs. 129 167 vs. 139	NS NS	Adenomatous & hyperplastic polyp risk	1.5 (0.9–2.6) 1.1 (0.7–0.8)	Age, sex, cancer subsites, education, SM, AL, BMI, PA, EI, NSAID use	Red and processed meats grouped together
Norat et al. (2005)	European Prospective Investigation into Cancer and Nutrition (EPIC), 10 European countries	478,040	m & f (35–70)	Red Processed	250 vs. 132 232 vs. 121	NS Sig	Colorectal cancer risk	1.49 (0.91–2.43) 1.70 (1.05–2.76)	Sex, age, Ht, Wt, EI, SM, AL, fibre, fat intake, PA, country	Age ranges differ by country, potential error associated with meat intake measurement & calibration method
Robertson et al. (2005)	The Antioxidant & the Calcium Polyp Prevention clinical trials, USA	1794	m & f (< 80)	Red Processed	373 vs. 133 363 vs. 146	NS NS	Adenoma recurrence risk	0.97 (0.78–1.21) 1.15 (0.92–1.43)	Age, sex, clinical centre, treatment category, study & duration of observational period	Did not control for all variables in results presented; 4 years could be considered as too short a follow-up period; no definition of red or processed meat

(continued on next page)

Table 2 (continued)

Author (year)	Study, country	Subjects (n)	Sex (age range)	Type of meat studied	Cases vs. non-cases (n)	Significance (high vs. low quintiles)	Outcome examined	Relative risk/hazard/odds ratios (C.I.)	Variables controlled for	Potential limitations of study
Sinha et al. (2005)	Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer screening trial case-control study, USA	3696 <sup>a</sup>	m & f (55–74)	Red Processed	~	NS NS	Colorectal cancer risk	1.07 (0.92–1.24) 1.04 (0.9–1.19)	Age, sex, screening centre, EI, ethnicity, education, SM, AL, aspirin, PA, BMI, fibre, calcium and folate	Large imbalance in No. of controls (34,817) and cases (3,696); large number of multiple comparisons may affect the reported findings
Chao et al. (2005)	Cancer Prevention II Nutrition cohort, USA	148,610	m & f (50–74)	Red Processed	210 vs. 164 26 vs. 153	NS NS	Colon cancer risk	1.15 (0.9–1.46) 1.13 (0.91–1.41)	Age, sex, EI, SM, PA, education, BMI, HRT, AL, F&V, fibre, supplements	Red and processed meats grouped together; short-term meat consumption a crude measure of cancer risk; potential error associated with FFQ and measurement of long-term intakes owing to major differences in questionnaires at two timepoints
English et al. (2004)	Melbourne Collaborative Cohort Study, Australia	37,112	m (27–75)	Red Processed	Unknown	NS Sig	Colorectal cancer risk	1.4 (1.0–1.9) 1.5 (1.1–2.0)	Sex, country of birth, EI, intake of fat & cereal products	Did not control for all variables in analysis, including BMI or alcohol consumption
Wei et al. (2004)	NHS & Health Professionals follow-up study (HPFS), prospective cohort studies, USA	134,365	m & f (30–75)	Red Processed	155 vs. 31 81 vs. 15	NS Sig	Colon cancer risk	1.43 (1.00–2.05) 1.33 (1.04–1.7)	Age, sex, Ht, BMI, PA, FH, AL, calcium, folate, SM, history of endoscopy, total meat intake	Used baseline dietary data collected at one timepoint that was previously associated with colon cancer; potential error associated with pooling results of several studies
Flood et al. (2003)	Breast Cancer Detection and Demonstration Project (BCDDP) cohort study, USA	45,496	f (35–80)	Red Processed	~	NS NS	Colorectal cancer risk	1.04 (0.77–1.41) 0.97 (0.73–1.28)	EI, total meat intake	Did not control for other covariates in analysis; red and processed meats grouped together
Cross et al. (2007)	NIH-AARP, USA	567,169	m & f (50–71)	Red Processed	935 vs. 255 932 vs. 251	Sig Sig	Colorectal cancer risk	1.24 (1.12–1.36) 1.2 (1.09–1.32)	Age, sex, education, marital status, FH, race, BMI, SM, PA, EI, AL, F&V	Red and processed meats were grouped together

Larsson et al. (2005)	Swedish Mammography Cohort prospective study, Sweden	66,651	f (40–75)	Red Processed	~	Sig NS	Distal colon cancer risk	1.32 (1.03–1.68) 1.07 (0.85–1.33)	Age, BMI, education, EI, AL, intake of SFA, calcium, folate, F& V, whole-grains	13 years could be considered a long follow-up period between dietary assessment & case identification; some processed meats were grouped with red meats; unable to control for PA
Sinha et al. (2005)	PLCO cancer screening trial case-control study, USA	3696 <sup>a</sup>	m & f (55–74)	Red meat cooked: medium well-done		NS Sig	Colorectal cancer risk	1.12 (0.99–1.28) 1.21 (1.06–1.37)	Age, sex, screening centre, EI, ethnicity, education, SM, AL, aspirin, PA, BMI, fibre, calcium and folate	Large imbalance in No. of controls (34,817) + cases (3696); large number of multiple comparisons may affect the reported findings; potential error associated with meat-specific questionnaire used
Chiu et al. (2003)	Case-control study, Shanghai, China	931 <sup>a</sup>	m & f (30–74)	Red	~	Sig for m only	Colon cancer risk	1.5 (1.0–2.1)	Age, BMI, EI, education, income, PA	Potential error associated with dietary assessment-subjects were asked to recall diet up to 5 years before diagnosis, using one standard portion size
Giovannucci et al. (1994)	HPFS cohort study, USA	47,949	m (40–75)	Beef, pork or lamb	~	Sig	Colon cancer risk	3.57 (1.58–8.06)	Age, BMI, EI, SM, AL, PA, history of previous polyp, FH, aspirin use, intake of fibre	6 years could be considered a long follow-up period between meat intake assessment and case identification; model used for total red meat included some processed meats

Abbreviations: NS, non-significant; Sig, significant; EI, energy intake; PA, physical activity, BMI, body mass index; SM, smoking; AL, alcohol; FH, family history, NSAID, non-steroidal anti-inflammatory drug; F&V, fruit & vegetables; SFA, saturated fatty acids; HRT, hormone-replacement therapy.

~ Data not measured or reported.

<sup>a</sup> Number of subjects in case group.

### 3. Risks to health

#### 3.1. Cardiovascular disease

Diet is one of the modifiable risk factors for CVD, which includes coronary heart disease (CHD), stroke and myocardial infarction (MI) (Williamson, Foster, Stanner, & Buttriss, 2005). Red meat has been associated with an increased risk of CVD by several studies (Fraser, 1999; Kelemen et al., 2005; Kontogianni, Panagiotakos, Pitsavos, Chrysohoou, & Stefanadis, 2008) Table 2 shows the results of some of these studies as well as their potential methodological limitations. Generally there has been no consistent use of any type of study design in the investigation of the relationship between meat consumption and CVD risk. Studies to date have used case-control, cross-sectional and cohort studies and the outcomes examined are not consistent, making it difficult to compare their findings. In one instance, Hu et al. (1999a) found a significant positive association between servings of red meat and the risk of CHD when age was adjusted for, but this effect became non-significant after controlling for age, BMI, smoking, alcohol, physical activity, energy intake and family history of CHD in the multivariate analysis (RR 1.09, (C.I. 0.9–1.3),  $p=0.35$ ). Another study reported a significant association with beef consumption ( $\geq 3$  servings/wk) and the risk of fatal CHD; however this association was observed only in men (Fraser, 1999). Kelemen et al. (2005) found that red meat was associated with an increased risk of mortality from CHD, but their classification of red meat included some processed meats. Similar inconsistencies have been observed in other studies (Hu et al., 1999a; Steffen et al., 2005), as there is no universal agreement of which meats can be classed as processed or red (Chao et al., 2005; WCRF, 2007).

The majority of these studies do not give absolute figures for quantities of meat associated with CVD and there is inconsistency in their use of servings or portions. It is also important to consider that most prospective studies have calculated the degree of risk by comparing the lowest to highest quintiles of meat intake, thereby excluding the majority of consumers who consume moderate amounts of meat. Kontogianni et al. (2008) found that high intakes of red meat (classified as greater than eight portions per month) were associated with an increased risk of acute coronary syndrome (ACS), but that low intakes of red meat (less than four portions per month) showed no association. Moreover, their use of a standard portion size of 60 g also presents limitations considering the variability of consumer interpretation of a typical portion size.

Much evidence is based on studies that have investigated dietary patterns rather than meat consumption in relation to risk of CVD (Iqbal et al., 2008; Panagiotakos et al., 2005; van Dam, Grievink, Ocke, & Feskens, 2003; Zyriax et al., 2008). A typical Western dietary pattern has been identified that is high in red meat and meat products, low in fruit and vegetables and coupled with a lifestyle of smoking, high alcohol intake and low levels of physical activity (Kontogianni et al., 2008). Heidemann et al. (2008), found this type of diet to be associated with a 22% greater risk of mortality from CVD than a prudent dietary pattern that is high in fruit and vegetables, legumes, poultry and whole grains. In the past, it has also been a common theme for prospective studies to compare mortality of CVD between vegetarians and non-vegetarians (Szeto, Kwok, & Benzie, 2004; Teixeira, Molina, Zandonade, & Mill, 2007). One such study found a reduced CVD mortality in vegetarians (compared to omnivores) who had been vegetarian for longer than 5 years, however not in those who had been vegetarian for less than 5 years (Key et al., 1998). Although these studies are useful in assessing multiple risk factors, analysing dietary patterns makes it impossible to isolate the effects of red meat alone. As a consequence, it has been difficult for studies to provide a convincing

mechanism for red meat in CVD. Past research has, however, predominantly assumed that the fat and fatty acid composition of red meat are responsible for its implication in CVD.

##### 3.1.1. Fat, saturated and trans fatty acids

Dietary recommendations to reduce the risk of CVD are to lower the contribution to daily energy intakes of total fat, saturated fatty acids (SFA) and trans fatty acids, in order to avoid their cholesterol-raising effects (Gidding et al., 2005). The major SFA within beef (myristic acid C14:0, palmitic acid C16:0 and stearic acid C18:0) have each been found to be significantly associated with CHD risk in the Nurses Health Study (Hu et al., 1999a), although others argue that a distinction should be made for stearic acid (C18:0) which has been found to have little cholesterol-raising effects in humans (Kelly et al., 2002; Mensink, Zock, Kester, & Katan, 2003). High consumers of meat ( $\geq 285$  g/d) have been found to possess both higher intakes of cholesterol and higher plasma concentrations of total cholesterol and low-density lipoprotein (LDL) cholesterol and triglycerides (TG) than vegetarians, vegans or moderate and low consumers of meat (Li et al., 1999). However, no distinction was made with unprocessed red meat in this study and it must be noted that the daily amount consumed by this group of 285 g is exceptionally high. When investigating the contribution of fat intake from meat consumption, it is important to consider the overall fatty acid composition of the diet, rather than studying the fat content of meat alone. With consumption of a low-fat diet, the addition of up to 180 g/d lean beef did not negate the total and LDL cholesterol lowering effects of the diet in hypercholesterolemic subjects (Beauchesne-Rondeau, Gascon, Bergeron, & Jacques, 2003; Watts et al., 1988; Wolmarans et al., 1999). In a cross-sectional study, Wagemakers, Prynne, Stephen and Wadsworth (2009) also observed no relationship between moderate red meat consumption (18–61 g/d) and blood concentrations of cholesterol. Moderate red meat consumption (24–72 g/d) in men and women has been found to contribute to 14.4% and 14.3% of total SFA intakes among Irish consumers respectively, but interestingly, this figure did not differ significantly from SFA intakes in non-consumers of red meat (Cosgrove et al., 2005).

Red meat produced today is leaner and lower in fat content than that produced ten years ago (Higgs, 2000). This is thought to be owing to a combined effect of changes in animal production, diets and butchery techniques (Williamson et al., 2005). Recently, lean red meat has been described as low in both SFA and total fat (Li, Siriamornpun, Wahlqvist, Mann, & Sinclair, 2005; Williams, 2007). When trimmed of excess fat, commonly consumed cuts of beef and lamb were found to contain less than 5% total fat content (Enser et al., 1998). For beef, the total fat content is equal to or lower than the SFA content of some white meats (Chan, Brown, Church, & Buss, 1996). Several studies have found no benefits of consuming poultry and fish instead of lean red meat in relation to effects on blood lipoprotein concentrations (Beauchesne-Rondeau et al., 2003; Watts et al., 1988; Wolmarans et al., 1999). When lean red meat consumption has been investigated in human intervention studies, all have failed to show any negative effects on blood concentrations of cholesterol, thrombotic factors, markers of oxidative stress or blood pressure in both healthy and hypertensive subjects (Hodgson, Burke, Beilin, & Puddey, 2006; Hodgson, Wards, Burke, Beilin, & Puddey, 2007; Li et al., 1999; O'Dea, Traianedes, Chisholm, Leyden, & Sinclair, 1990).

Trans unsaturated fatty acids have been deemed particularly potent in their ability to increase blood concentrations of cholesterol; knowledge which has led to recommendations advising their full or partial removal from the manufacturing process of food products involving hydrogenation of vegetable oils (Hulshof et al., 1999; USFDA, 2003; World Health Organisation., 1990). Trans unsaturated fatty acid isomers are often grouped together, with

man-made *trans* fats not being distinguished from those found naturally within milk and meat from ruminants (Palmquist, Lock, Shingfield, & Bauman, 2005). Intakes of elaidic acid (C18:1 *trans*-9), the major industrially produced *trans* fat, can negatively affect cholesterol metabolism in humans (Sundram, Ismail, Hayes, Jeyamalar, & Pathmanathan, 1997). In contrast, there is emerging evidence that *trans*-vaccenic acid (TVA, C18:1 *trans*-11), the major *trans* fatty acid found within red meat, has no effect on either total cholesterol or LDL cholesterol concentrations (Chardigny et al., 2008). TVA is also an intermediate in the production of the conjugated linoleic acid (CLA) isomer *cis*-9, *trans*-11, which may have potential health benefits (Palmquist et al., 2005). Therefore, the *trans* fatty acid content of red meat is unlikely to be a contributing factor to risk of CVD. Furthermore, minor concentrations of total *trans* fatty acids consumed from European diets are not thought to be a cause for concern (Hulshof et al., 1999).

In conclusion several studies have hypothesised that the fat content of red meat might be a risk factor for CVD, however there is a lack of evidence to suggest that consuming lean red meat trimmed of excess fat, which is lower in both total fat and SFA, can increase risk of CVD (Li et al., 2005). There is a need for future studies to investigate lean red meat in relation to risk of CVD, to avoid making the assumption that all red meats have an equal fat content.

### 3.2. Cancer

#### 3.2.1. Colon cancer

Meat intake has been significantly associated with an increased risk of colon cancer by several epidemiological studies (Cross et al., 2007; Giovannucci et al., 1994; Wei et al., 2004). Recently, the WCRF report summarised extensive evidence in this field and concluded that research supporting the association between red and processed meat intake and colon cancer risk was convincing (WCRF, 2007). This came a decade after the previous report stated the evidence for red meat was probable and possible for processed meat (WCRF, 1997). However, Table 2 shows that some studies in this area have reported findings which are not consistently significant. Indeed, some studies have shown no associations between red meat intake and the incidence of colon cancer (Goldbohm et al., 1994; Robertson et al., 2005). Hill (2002) proposed that several additional studies showing no relationship between red meat intake and cancer risk were omitted from the WCRF, 1997 report. Prospective studies carried out over a long time period are the most commonly cited study design in reports of the investigation of colon cancer risk, which are generally accepted as being more robust in their design. However, it is worth noting that Truswell (2002) reviewed 30 case–control studies and 20 of these found no significant association between red meat and colorectal cancer.

It is difficult to compare results across studies owing to the many differences in study design which exist; from differences in sample size or method of dietary assessment, to variation of the endpoint measurement; whether it is cancer incidence, or occurrence of adenoma (Robertson et al., 2005). Several studies have grouped colon and rectum cancers together as colorectal cancer when investigating risk in relation to meat intake (Cross et al., 2007; Kimura et al., 2007; Norat et al., 2005). It could be argued that these cancers differ in aetiology and as such, should be investigated separately. Larsson, Rafter, Holmberg, Bergkvist, and Wolk (2005) reported an elevated risk of colorectal cancer with high red meat intakes, but in a separate analysis of individual sites, this risk was significant at the distal colon only. In another study where colorectal cancer sub sites were distinguished, there was a non-significant association at both the colon and rectum with high red meat intakes (English et al., 2004). Probably the greatest limitations of these studies are the discrepancies in how they define

red and processed meat (Flood et al., 2003; Goldbohm et al., 1994). The reported association between processed meat consumption and colon cancer risk is known to be stronger than for unprocessed red meat (Norat et al., 2005). However, studies have consistently failed to analyse these meats separately. Larsson et al. (2005), found a significantly elevated risk of colorectal cancer with red meat consumption, when bacon, hot-dogs, luncheon meat and ham were grouped as both red and processed meats. Kimura et al. (2007) also combined red and processed meats in their analysis, making it impossible to ascertain the effect of red meat alone.

How studies actually measure meat intake also presents a potential source of error, with self-reported intakes being commonly used in case–control and cross-sectional studies (Williamson et al., 2005). There is often a period of several years between dietary data collection and when the measurement of the outcome, during which time subjects' diets could have changed considerably (Giovannucci et al., 1994; Larsson et al., 2005).

Generally, the proposed carcinogenicity of meat has been assigned to the type of meat consumed (red or processed), the method of cooking, the quantity consumed and the individual genetic risk (Larsson & Wolk, 2006). The most supported mechanism is that formation of mutagenic compounds, including HCAs and polycyclic aromatic hydrocarbons (PAHs), formed within meat cooked at high temperatures to a well-done state, are responsible for carcinogenesis (Alaejos, Gonzalez, & Afonso, 2008; Bingham et al., 2002). However, very few studies have assessed the method of cooking when measuring meat consumption. Indeed the effects of red meat *per se* have not been isolated from the effects of processing or cooking temperatures. Moreover, of the few studies which have considered these factors some found an increased risk of colon cancer (Butler et al., 2003) or adenoma recurrence (Martinez et al., 2007) with high intakes of well or very well done red meat compared to low intakes, whilst others found no increase in risk (Shin et al., 2007; Wu et al., 2006). It is only possible to speculate on the role of HCAs in carcinogenesis, as exact quantities within cooked red meat are likely to be very small and many interactions will exist with other dietary carcinogens and possible protective components of the diet such as fruit and vegetables and fibre. There is also evidence that there is a genetic influence on HCA metabolism, which gives some individuals a predisposed risk, before the quantity of meat consumed is considered (Kampman et al., 1999; Skog, 2002).

Approximately 80% of colon cancer cases are thought to be caused by modifiable diet and lifestyle factors (Willet, 1995). However, it is unlikely that red meat consumption is an independent risk factor for colon cancer development owing to the complex nature of this disease and the large number of interacting risk factors, including smoking and physical activity level, which will contribute to cancer aetiology even when these factors are adjusted for in the analysis (Williamson et al., 2005). Put in context, it is unlikely that reducing meat consumption alone is sufficient to reduce risk, unless the complete dietary balance has been addressed. Despite a wealth of studies in this area, there is still conflicting and inconsistent evidence that red meat contributes to colon cancer risk, which contradicts the WCRF findings. In a recent letter challenging the most recent WCRF report, Truswell (2009) lists several flaws of the meta-analysis within the red meat section, most specifically the decision to re label red meat as a convincing cause of colorectal cancer. Leading cancer researchers also criticised the media confusion created by the report and recommend that in future, changes be made as to how cancer risk is measured to avoid placing too much blame on any one food (Boyle, Boffetta, & Autier, 2008). However, since there is currently a lack of evidence to show that red meat plays no role in colon cancer, the current advice still stands, which is to consume less than 500 g of red

meat per week, minimise intakes of processed meat and avoid cooking meat at very high temperatures (WCRF, 2007).

### 3.2.2. Other cancers

In relation to other cancers, evidence associating either red or processed meat consumption with cancers of the prostate, lung, bladder, oesophagus or pancreas is limited and not thought to be convincing (WCRF, 2007). Recently, the UK Women's Cohort Study found a significant association between red meat consumption and breast cancer risk in post-menopausal women, with a stronger effect for processed meat (Taylor, Burley, Greenwood, & Cade, 2007). However, a pooled analysis of several cohort studies measuring meat intake in relation to breast cancer risk found no significant association and revealed considerable differences between studies in their use of methods for assessing meat intake (Missimer et al., 2002). Taking account of cooking methods, Kabat et al. (2009) found no significant associations between red meat consumption and breast cancer risk. There is a need for further studies before conclusions can be made on the effect of red meat consumption on risk of other cancers.

## 4. Benefits to health

### 4.1. Fatty acid composition

Approximately 50% of the intra-muscular fat of beef and lamb is made up of unsaturated fatty acids; monounsaturated fatty acids (MUFA), primarily oleic acid (C18:1 *c*-9) and polyunsaturated fatty acids (PUFA), predominantly the essential *n*-6 and *n*-3 PUFA linoleic acid (LA, C18:2) and alpha-linolenic acid (ALA, C18:3), respectively. The ratio of PUFA to SFA (P:S) is approximately 0.11 in beef and 0.15 in lamb and much lower than the desired dietary ratio of 0.4, owing to the degree of biohydrogenation of unsaturated fatty acids in the rumen (Scollan et al., 2006). A meta-analysis has shown that increasing the dietary ratio of P:S can lead to a reduction in plasma total cholesterol and as a result, there is much research focusing on ways to improve this ratio within meat (Howell, McNamara, Tosca, Smith, & Gaines, 1997; Scollan et al., 2001). The fatty acid composition of meat will vary by animal age, sex, breed, diet and within the cut of meat (Wood & Enser, 1997); variations which food composition tables do not account for. There is clearly a need for an update of fatty acid compositional data and for future epidemiological research to take account of this variation in fat content within meat tissue. Table 3 documents studies that have reported several benefits of red meat consumption, with particular emphasis on the positive effects on plasma lipoproteins in consumers. Most notably, moderate consumption of lean red meat was found to lower total cholesterol, LDL cholesterol and TG (Beauchesne-Rondeau et al., 2003) and to have no effect on markers of platelet aggregation (Li et al., 1999) or oxidative stress markers (Hodgson et al., 2006), in comparison to a control group.

#### 4.1.1. *n*-3 polyunsaturated fatty acids (*n*-3 PUFA)

Lean tissue of red meat contains ALA and the long chain *n*-3 PUFA (LC $n$ -3 PUFA) eicosapentaenoic acid (EPA, C20:5), docosapentaenoic acid (DPA, C22:5) and docosahexaenoic acid (DHA, C22:6). ALA, derived mainly from plant sources, has been associated with a reduced risk of CVD by epidemiological studies (Ascherio et al., 1996; Hu et al., 1999b). Its elongation products, the LC $n$ -3 PUFA, are widely recognised for their numerous effects on heart health; improving platelet aggregation, vasodilation and thrombotic tendency (Mann et al., 2006; Siddiqui, Harvey, & Zaloga, 2008). Beneficial effects to the central nervous system, retinal function and the inflammatory response have also been ascribed

to LC $n$ -3 PUFA (Ruxton, Reed, Simpson, & Millington, 2004). Yet their synthesis from ALA is small and somewhat inefficient which requires them to be present in the diet in their elongated form (Burdge & Calder, 2006).

Concentrations of LC $n$ -3 PUFA found in beef and lamb are lower than those within oily fish (0.28 mg and 0.52 mg vs. 19.9 mg/g), but may be more important than previously realised since red meat intakes are greater than those of oily fish in the UK and Ireland (Enser, Hallett, Hewitt, Fursery, & Wood, 1996; Cosgrove et al., 2004; SACN/COT, 2007). Red meat is the main dietary source of DPA, which accumulates in mammals but not in oily fish (Givens & Gibbs, 2006). Little research exists on the clinical significance of DPA, but it has been suggested to be inversely related to atherosclerotic risk and risk of acute coronary events in middle-aged men from Finland (Hino et al., 2004; Rissanen, Voutilainen, Nyyssönen, Lakka, & Salonen, 2000). Despite the fact that DPA is not considered in dietary recommendations for LC $n$ -3 PUFA, it is has comparable health benefits to those of EPA and DHA in reducing CVD risk (Howe, Buckley, & Meyer, 2007).

Studies have shown that meat consumers have greater plasma concentrations of LC $n$ -3 PUFA than vegetarians (Li et al., 1999; Mann et al., 2006; Rosell et al., 2005), but there is a lack of data to show whether consuming modest amounts of red meat can provide concentrations sufficient to produce biological effects. Concentrations of LC $n$ -3 PUFA are recognisably higher within meat from animals fed a grass diet (Arouseau, Bauchart, Calichon, Micol, & Priolo, 2004; Enser et al., 1998; French et al., 2000; Ponnampalam, Mann, & Sinclair, 2006). In Australia, where animals are grass-fed for most of the year, an updated analysis of fatty acid composition showed that total meat and meat products contribute 43% of total dietary intakes of LC $n$ -3 PUFA compared to 48% from oily fish in Australian adults, owing to meat intakes being 6 times higher than of fish (Howe, Meyer, Record, & Baghurst, 2006; Howe et al., 2007). Givens and Gibbs (2006) estimated that in the UK, beef and sheep meat provide 9.85 mg/d and 3.82 mg/d LC $n$ -3 PUFA compared with 142.1 mg/d from oily fish based on meat intakes measured in the NDNS (Henderson & Gregory, 2002) and fatty acid data measured by Enser, Hallett, Hewitt, Fursery, and Wood (1996). However, since existing UK food composition tables indicate negligible amounts of many fatty acids within meat, it is likely that an update in this information would provide more precise data which in turn may show that the contribution of beef and lamb to LC $n$ -3 PUFA intakes is currently underestimated (Chan et al., 1996). As with Australia, total meat intakes within Ireland are known to be greater than of oily fish, so red meat could be contributing more LC $n$ -3 PUFA than hitherto recognised (NSIFCS, 2001).

Studies which have discussed strategies to increase beneficial quantities of LC $n$ -3 PUFA within meat through grass feeding or otherwise, have often referred to a lowering of the *n*-6:*n*-3 ratio of meat and overall diet as desirable for lowering CVD risk in adults (Scollan et al., 2006; Wood & Enser, 1997). However, the usefulness of this ratio has recently been questioned, with concern that it detracts from actual amounts of both *n*-3 and *n*-6 PUFA that are essential for human health (Givens & Gibbs, 2008). In the UK recommendations to prevent deficiency exist for both ALA (1.1 g/d for women; 1.6 g/d for men) and LC $n$ -3 PUFA (450 mg/d EPA + DHA) (Institute of Medicine., 2002; SACN/COT, 2004), with no universally agreed amount required for the prevention and treatment of CVD (Anil, 2007; Gebauer, Psota, Harris, & Kris-Etherton, 2006). One problem of the *n*-6:*n*-3 ratio is its assumption that ALA and LC $n$ -3 PUFA are equal with regard to their effects on health, which is unlikely to be the case given that LC $n$ -3 PUFA are precursors for eicosanoids (Gorjao et al., 2009).

No specific dietary recommendations exist for *n*-6 PUFA, perhaps owing to their abundance in Western diets as a constituent



**Table 3**  
Summary of cross-sectional and intervention studies showing some benefits of meat and red meat consumption.

Author (year)	Study, country	Subject status (n)	Sex (age range)	Length of study (wks)	Type of meat	Groups	Markers examined	Main outcomes (↑, ↓, -) <sup>a</sup>	Significance (vs. control/ between groups)
Mann et al. (1999)	Cross-sectional intervention, Australia	Healthy (147)	m (20–55)	~	~	Vegans & vegetarians vs. moderate meat-eaters (<285 g/d) & high meat-eaters (>285 g/d)	Dietary data, plasma & serum folate, vitamin B12 & homocysteine	↑ Plasma B12 ↓ Homocysteine	Sig Sig
Li et al. (1999)	Cross-sectional analysis, Australia	Healthy (147)	m (20–50)	~	~	Vegans & vegetarians vs. moderate meat-eaters (<285 g/d) & high meat-eaters (>285 g/d)	Markers of platelet aggregation, inflammatory markers	- Ex vivo platelet aggregation - TXB <sub>2</sub>	NS NS
Mann et al. (2006)	Cross-sectional analysis, Australia	Healthy (47)	m (20–55)	~	~	Vegans & vegetarians vs. moderate meat-eaters (<285 g/d) & high meat-eaters (>285 g/d)	Nutrient intakes	↑ LCn-3 PUFA - SFA	Sig NS
Beauchesne-Rondeau et al. (2003)	Cross over intervention, France	Healthy (18)	m (50.1) <sup>b</sup>	12	Lean beef	Lean beef diet vs. poultry vs. lean fish diet	Plasma lipids	↓ TC ↓ LDL ↓ VLDL ↓ TG	NS NS NS NS
Cosgrove et al. (2005)	Cross-sectional analysis, Ireland	Healthy (958)	m & f (18–64)	~	~	Red meat consumers vs. vegetarians	Nutrient deficiency	↓ Zinc ↓ Riboflavin ↓ Vitamin C ↓ Vitamin B12 ↓ Iron ↓ Folate	Sig Sig Sig NS NS NS
Hodgson et al. (2006)	Parallel intervention, Australia	Hypertensive (60)	m & f (58.6) <sup>b</sup>	8	Lean red meat	~215 g/d lean meat diet vs. control	Blood pressure, serum lipids	↓ Systolic blood pressure	Sig
Hodgson, Wards, Burke, Beilin, and Puddey (2007)	Parallel intervention, Australia	Hyperlipidaemic (60)	m & f (58.6) <sup>b</sup>	8	Lean red meat	~215 g/d lean meat diet vs. control	Markers of iron status, inflammatory markers (SAA, CRP, plasma fibrinogen), markers of oxidative stress (GGT, plasma & urinary isoprostanes), dietary data	- Iron status - Plasma F <sub>2</sub> isoprostanes - Serum GGT - Serum SAA - Plasma fibrinogen	NS NS NS Sig NS Sig
Wagemakers et al. (2009)	Cross-sectional analysis, UK	Healthy (2256) <sup>c</sup>	m & f (43, 53) <sup>b</sup>	~	Red meat	High consumers (38–127 g/d) vs. low/moderate consumers (0–15 g/d)	Dietary data, blood pressure, serum lipids, waist circumference	- TC - LDL - HDL	NS NS NS
Prynne et al. (2009)	Cross-sectional analysis, UK	Healthy (2256) <sup>c</sup>	m & f (43, 53) <sup>b</sup>	~	Red meat	High consumers (38–127 g/d) vs. low/moderate consumers (0–15 g/d)	Nutrient intakes	↑ Vitamin B12 ↑ Haem iron ↑ Zinc	Sig Sig Sig

Abbreviations: NS, non-significant; Sig, significant; TC, total cholesterol; LDL, low-density lipoprotein cholesterol; VLDL, very low-density lipoprotein; HDL, high-density lipoprotein cholesterol; TG, triglycerides; TXB<sub>2</sub>, thromboxane B<sub>2</sub>; SAA, serum amyloid; CRP, C-reactive protein; GGT, Gamma-glutamyl transferase; SFA, saturated fatty acids.

~ Data not measured or reported.

<sup>a</sup> ↑ increase observed; ↓ decrease observed; - no change observed.

<sup>b</sup> Mean age reported only.

<sup>c</sup> Number of subjects in 1989 only.

of vegetable oils (Harbige, 2003). An additional assumption of the  $n-6:n-3$  ratio is that lowering the amount of  $n-6$  PUFA in the diet is always beneficial to health, yet there are reports that not all  $n-6$  PUFA are proinflammatory and their essentiality in infant development is often overlooked (Harbige, 2003).

In consideration of these factors and owing to a lack of evidence that lowering the  $n-6:n-3$  ratio continuously improves physiological function, it was concluded by a UK Food Standards Agency (FSA) workshop that this ratio should not be used in future as an indice of CVD health (Stanley et al., 2007). Instead, Stanley et al. (2007) suggest moving focus away from the ratio to absolute amounts of  $n-3$  and  $n-6$  PUFA, giving individual consideration to ALA and LC $n-3$  PUFA. Similarly it may also be a misleading ratio to use when describing the fatty acid content of meat in relation to nutritional value.

#### 4.1.2. Conjugated linoleic acid

Conjugated linoleic acid (CLA) is a term used to describe a group of positional and geometric isomers of octadecadienoic acid, of which ruminant meat and milk are the major dietary sources (Turpeinen et al., 2002). Within beef muscle concentrations of CLA have been found to range from 0.37 to 1.08 g/100 g (French et al., 2000). Beef and lamb consumption in Portugal contribute to 4.12% and 11% of total CLA intakes (Martins et al., 2007). CLA is formed both through the ruminal biohydrogenation of dietary LA and also through an endogenous synthesis pathway from TVA. The isomer, *cis-9, trans-11* (also known as rumenic acid), is the major and most important CLA isomer found in red meat (Chardigny et al., 2008). Its concentration within meat tissue is, like LC $n-3$  PUFA, higher when animals have been grass-fed (Beam, Jenkins, Moate, Kohn, & Palmquist, 2000).

CLA is receiving increasing attention for its observed anti-carcinogenic and anti-atherogenic properties in animal studies (Lock, Corl, Barbano, Bauman, & Clement, 2004; Lock, Horne, Bauman, & Salter, 2005; Hargrave-Barnes, Azain, & Miner, 2008). It has also been found to favourably modulate immune function in humans (Tricon et al., 2005). Although the consumption of ruminant products contributes to blood concentrations of CLA in humans (Burdge et al., 2005; Martins et al., 2007), it is largely unknown whether these physiological doses might have biological effects in humans (Turpeinen et al., 2002).

#### 4.2. Other nutrients in red meat

Red meat consumption contributes many vitamins and minerals to the diet that are essential for health. It is a major source of protein, providing about 20 g/100 g of beef or lamb consumed (Chan et al., 1996). In comparison to vegetarians, omnivores have greater intakes of protein (Davey, Spencer, & Appleby, 2003). Consuming a high protein (from lean red meat as an example) and low carbohydrate diet whilst controlling energy intake has recently been found to facilitate weight loss and weight maintenance when compared with consuming a diet of similar energy intake that is low in protein (Layman, Clifton, Gannon, Krauss, & Nuttall, 2008; Paddon-Jones, Westman, Mattes, Wolfe, & Astrup, 2008). This effect is owing to the satiety inducing effect of protein and its positive effects on lean muscle mass in humans. In the past meat consumption has been associated with higher BMI (Rosell, Appleby, Spencer, & Key, 2006; Spencer, Appleby, Davey, & Key, 2003), but the result of these studies suggest that consuming lean red meat as part of an energy controlled diet may not result in increased body weight, when controlling for other dietary factors. Modest increases in protein intakes from red meat have also been shown to lower blood pressure without increasing blood lipids (Hodgson et al., 2006).

Iron-deficiency anaemia (IDA) is a major nutritional deficiency, affecting populations of both high and low socioeconomic status worldwide, being particularly prevalent among children and young women (Gibson & Ashwell, 2002). Iron is vital for many cellular processes in the body and, as a component of haemoglobin, is essential to maintaining adequate transport of oxygen in the blood. Therefore, even mild suboptimal status before the onset of anaemia can impact negatively on health (Gibson & Ashwell, 2002). Haem iron found in meat is more bioavailable than non-haem iron found in plant sources and, for this reason, meat consumers maintain better iron status than vegetarians and vegans (Cosgrove, Flynn, & Kiely, 2005; Gibson & Ashwell, 2002). Red meat in particular is recognised as a significant source of haem iron compared to poultry and fish (Johnston, Prynne, Stephen, & Wadsworth, 2007). Intakes of red meat classified as low ( $\leq 41$  g/d) were found to supply 13.1 mg and 15.8 mg/d iron among Irish men and women, respectively (Cosgrove et al., 2005). These figures show that even low consumption of red meat positions Irish men and women favourably above the UK recommended nutrient intake (RNI) for iron of 8.7 mg and 14.8 mg/d for males and females. However, Gibson and Ashwell (2002) report that consuming less than 90 g/d red meat may put men and women at three times higher risk of having low iron status (Gibson & Ashwell, 2002). In which case, cutting out or lowering meat intakes to the current advised limit of 71 g/d red meat, could seriously impact on iron status.

Moderate red meat consumption by Irish men and women has been estimated to contribute to 5.3  $\mu$ g and 6.5  $\mu$ g/d of vitamin B12, respectively (Cosgrove et al., 2005). Red meat is indeed the major dietary source of B12 in the diet, providing over two thirds of the daily requirement in one 100 g serving (Cosgrove et al., 2005). Since B12 is required by active enzymes within the methylation cycle, low intakes of B12 as well as folate and vitamin B6 have been associated with elevated homocysteine, which is a risk factor for CVD and stroke (Scott, 1999; Wagemakers, Prynne, Stephen, & Wadsworth, 2009). Mann et al. (1999) have confirmed in a cross-sectional study that consumers with high intakes of total meat compared to vegetarians have lower homocysteine levels. It has also been shown that regular consumption of moderate amounts of red meat (40–72 g/d) can help to lower the risk of inadequate B12 intakes compared to low consumers (Cosgrove et al., 2005; Prynne et al., 2009).

In relation to contribution of zinc, beef and lamb contain 4.1 mg and 3.3 mg/100 g tissue (Chan et al., 1996) and, as a result, have been classified as rich sources (British Nutrition Foundation, 2002). Low consumption of red meat ( $\leq 41$  g/d) is estimated to contribute 10.2 mg and 10.6 mg/d of zinc to Irish men and women respectively (Cosgrove et al., 2005). Considering that the RNI for zinc is set at 9.5 mg and 7.0 mg for males and females, intakes of red meat at such levels are capable of providing adequate zinc for optimum health.

It is clear that red meat is a nutrient rich food, supplying valuable amounts of protein, haem iron, zinc, B vitamins, selenium and retinol, with increased bioavailability than found in other dietary sources (Cosgrove et al., 2005; Davey et al., 2003).

## 5. Conclusions

In the present review, studies investigating associations between red meat consumption and outcomes of health and disease were reviewed. Within studies which implicate red meat in the development of CVD and colon cancer, a number of methodological limitations were found; they do not assess the degree of fat-trimming or method of cooking used and their method of assessing meat intake is potentially prone to error or bias. Most notably, not all studies were consistent in how they measured meat con-

sumption, with many including processed meat as red meat in their analysis and fewer studies examining lean red meat *per se*. Questions have recently been raised over the scientific basis for red meat being labelled a convincing cause of colon cancer in the recent WCRF report, as well as the basis for the daily recommended intake being lowered to 71 g/d. Nevertheless, it is likely that maintaining intakes at or below the current advised level, whilst reducing intakes of processed meat and meat cooked at very high temperatures will ensure a balance is reached where the potential risk of colon cancer is reduced and the beneficial effects of consuming red meat are achieved.

This review of the risks and benefits associated with red meat consumption has shown that consuming moderate amounts of lean red meat, as part of a balanced diet, valuably contributes to intakes of essential nutrients and possibly to intakes of LCn–3 PUFA and CLA, but its contribution to risk of either CVD or colon cancer warrants further research through larger controlled prospective studies before it can be definitively implicated as having a causative role in these diseases.

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