British Journal of Nutrition (2017), **118**, 222–228
doi:10.1017/S0007114517002008

Processed red meat contribution to dietary patterns and the associated cardio-metabolic outcomes

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(Submitted 15 March 2017 – Final revision received 19 June 2017 – Accepted 12 July 2017)

Abstract

Evidence suggests that processed red meat consumption is a risk factor for CVD and type 2 diabetes (T2D). This analysis investigates the association between dietary patterns, their processed red meat contributions, and association with blood biomarkers of CVD and T2D, in 786 Irish adults (18–90 years) using cross-sectional data from a 2011 national food consumption survey. All meat-containing foods consumed were assigned to four food groups (*n* 502) on the basis of whether they contained red or white meat and whether they were processed or unprocessed. The remaining foods (*n* 2050) were assigned to twenty-nine food groups. Two-step and *k*-means cluster analyses were applied to derive dietary patterns. Nutrient intakes, plasma fatty acids and biomarkers of CVD and T2D were assessed. A total of four dietary patterns were derived. In comparison with the pattern with lower contributions from processed red meat, the dietary pattern with greater processed red meat intakes presented a poorer Alternate Healthy Eating Index (21·2 (SD 7·7)), a greater proportion of smokers (29 %) and lower plasma EPA (1·34 (SD 0·72) %) and DHA (2·21 (SD 0·84) %) levels ($P<0·001$). There were no differences in classical biomarkers of CVD and T2D, including serum cholesterol and insulin, across dietary patterns. This suggests that the consideration of processed red meat consumption as a risk factor for CVD and T2D may need to be re-assessed.

Key words: Processed red meat: CVD: Type 2 diabetes: Dietary pattern analysis

Consumption of red meat has been associated with the development of chronic diseases including the metabolic syndrome (MetS), CVD and type 2 diabetes (T2D)[1,2]. With the prevalence of these diseases increasing rapidly, the need for public health strategies to help improve dietary quality and reduce chronic disease risk is becoming more crucial[3].

Public health recommendations advise limiting red meat consumption to ≤500 g/week[4]. However, there is conflicting evidence regarding the type of red meat consumed. A number of reviews have highlighted the importance of discriminating processed from unprocessed red meat when examining the effect on health outcomes[2,5]. This suggests that preservation and processing, coupled with the confounding effects of diet and lifestyle factors, need to be considered in disease causality[6]. A meta-analysis by Micha *et al.* identified an association between a 50 g/d serving of processed meat and CVD risk (relative risk 1·42; 95 % CI 1·07, 1·89, $P=0·04$); however, no association was observed with a 100 g serving of unprocessed red meat (relative risk 1·00; 95 % CI 0·81, 1·23, $P=0·36$)[2]. Moreover, processed meat consumption has also been associated with a greater incidence of T2D (hazard ratio 1·32; 95 % CI 1·25, 1·40, $P<0·001$) than unprocessed red meat consumption (hazard ratio 1·12; 95 % CI 1·08, 1·16, $P<0·001$)[5].

However, because of their observational nature it is difficult to determine causality. A recent meta-analysis by O'Connor *et al.*, which included randomised controlled trials only, failed to find a causal relationship between daily total red meat intakes of ≥0·5 servings/d (≥35 g/d) and markers of CVD, cholesterol levels, TAG and blood pressure ($P>0·05$)[7]. Similarly, a meta-analysis by Fretts *et al.* reported that the association of red meat with markers of T2D was attenuated after controlling for BMI[8].

The above analyses were based on meat consumption; however, when reviewing the impact of food types on metabolic disease risk, it is important to consider the overall dietary pattern. Low red meat consumption (≤0·5servings/d) is typical of a Mediterranean dietary pattern, which has been associated with lesser risk for CVD and T2D[9,10]; however, randomised controlled trials have shown that Mediterranean diet patterns with ≥0·5

Abbreviations: %TE, percentage of total energy; T2D, type 2 diabetes.

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servings/d of red meat had a similar effect on CVD risk factors[11]. In contrast, higher red meat consumption is typically associated with a Western dietary pattern, which has been positively associated with increased risk of disease[9,10,12].

The aims of this analysis were to identify dietary patterns in a nationally representative cross-sectional cohort, to characterise the contribution of processed red meat to overall diet and, moreover, to investigate the association of biomarkers of CVD and T2D with nutrient intakes and plasma fatty acid levels within identified dietary patterns.

Methods

Study population

The National Adult Nutrition Survey (NANS) is a cross-sectional food consumption survey of a demographically representative sample comprising 1500 free-living men (n 740) and women (n 760), aged 18–90 years, across the Republic of Ireland between 2008 and 2010[13]. Individuals who failed to provide a blood sample (n 364) were excluded from the current analysis, as were under-reporters (n 351) – those participants who presented an energy intake:BMR ≤1·10[14]. The final sample size was 786 (men: 399; women: 387). There were no differences in sex, age, current smoking status and supplement use between those included in the analysis and those excluded. However, there was a greater percentage of non-manual/skilled manual workers in the excluded population, who had a higher BMI than individuals in the current cohort ($P<0·05$). A detailed description of the NANS recruitment and methodology is reported elsewhere[13]. However, a concise overview of the data collection and laboratory techniques relevant to this analysis is outlined below. Ethical approval was granted by the University College Cork Clinical Research Ethics Committee of the Cork Teaching Hospitals and the Human Ethics Research Committee of University College Dublin (ECM 3 (p), 4 September 2008). Written consent was obtained from each participant, in accordance with the Declaration of Helsinki.

Dietary assessment and analysis

Food and beverage intakes were assessed over 4 consecutive days, including at least 1 weekend day, using a semi-weighed food diary. Brand names, recipes and cooking methods were also recorded. The food-composition database was updated to include recipes, nutritional supplements, commonly consumed generic Irish foods and new foods on the market. The database comprised 133 068 rows of data and 2552 food codes were assigned to all food, beverages and nutritional supplements consumed. Food and nutrient intakes were analysed using the Weighed Intake Software Program (WISP© version 3.0; Tinuviel Software)[13]. A total of 2048 food codes were aggregated into twenty-nine food groups, representative of the overall diet[15]. In all, 502 food codes contained meat and were characterised into four groups: unprocessed red, processed red, unprocessed white and processed white meat. Red meat included beef, lamb and pork, whereas poultry was classified as white meat. Processed meat had undergone salting, curing, fermentation, smoking, flavour enhancement or other preservation processes, examples of which included ham and sausages[16]. To calculate mean daily processed red meat intakes, each food code was updated for grams of meat per 100 g of product, using the online McCance and Widdowson Composition of Foods integrated data set and manufacturer's information[17]. Information on sociodemographic characteristics, health and lifestyle habits and anthropometric measurements were obtained[13]. Alternate Healthy Eating Index (AHEI) scores were assigned based on the criteria given by McCullough *et al.*[18] with a higher overall score representing a healthier diet pattern.

Biochemistry measurements

A clinical bioanalyser (RX Daytona; Randox Laboratories) was used to measure levels of glucose, TAG and total and HDL-cholesterol in serum samples[19]. LDL-cholesterol levels were calculated as (Total cholesterol/HDL-cholesterol) – (TAG/2·2). Insulin, leptin and TNFα levels were measured using a biochip array system (Evidence Investigator; Randox Laboratories). Adiponectin levels were measured using ELISA (ALPCO Diagnostics kit; ALPCO) and homocysteine levels using a flourescence polarisation immunoassay. A detailed description of the lipid extraction methodology and fatty acid analysis has been outlined elsewhere[20]. The National Cholesterol Education Programme's Adult Treatment Panel III criterion was applied to evaluate risk for the MetS[21].

Statistical analysis

Data were analysed using SPSS® for Windows™ statistical software package version 20.0 (SPSS Inc.). Descriptive statistics for continuous variables are presented as mean values and standard deviations, whereas categorical variables are reported as percentages.

The thirty-three food groups were converted to percentages of total daily energy (%TE) intakes, to derive dietary patterns proportional to energy intakes, and standardised as z scores. Preliminary two-step cluster analysis was applied to determine the optimal number of dietary clusters in the cohort. The first step involves the formation of preclusters, based on the distance criterion, whereas the second step applies the standard hierarchical clustering algorithm to these preclusters. This analysis identified four dominant dietary patterns. k-Means subsequently characterised these patterns by separating participants into non-overlapping groups derived from Euclidean distance. To validate the dietary patterns, the population was randomly divided into two parts and the analysis was repeated. In all, 69 % of individuals were re-classified in the cluster analysis validation.

Sociodemographic characteristics were analysed using the χ^2 statistic for categorical variables and one-way ANOVA with Bonferroni *post hoc* tests for continuous variables. Differences in nutrient intakes, dietary quality, anthropometric measurements and biochemical data were assessed using an adjusted general linear model. Bonferoni correction was applied by multiplying each P value by the number of traits in each table. $P\leq 0·05$ were considered significant and those that exceeded 1·0 were marked down to 1·000.

Results

Total red meat intake was 134 g/d (male) and 89 g/d (female), whereas processed red meat intakes were 52 g/d (male) and

29 g/d (female). Overall, four dietary patterns were identified (Table 1). Pattern 1 presented higher energy contributions from wholemeal breads, vegetables, fruit, fish and yogurts ($P<0·001$). The greatest contributors to energy in Pattern 2 were chips, processed potatoes, rice, pasta, fruit juices, smoothies and cheeses ($P<0·001$). Pattern 3 had higher contributions from alcoholic beverages, unprocessed red meat, ready-to-eat breakfast cereals, savouries and confectionery ($P<0·05$). White bread, processed red meat, butters, whole milk and potatoes were the greatest contributors towards energy intakes in Pattern 4 ($P<0·001$). Mean daily processed red meat intakes were lowest in Pattern 1 (1·3 %TE) and greatest in Pattern 4 (2·4 %TE) ($P<0·001$). There was a significantly greater contribution of processed red meat to Pattern 4, in comparison with all other patterns (online Supplementary Table S1).

Participants in Pattern 1 were predominantly older professional women, with better dietary quality and greater supplement usage ($P<0·001$). Pattern 2 was seen in younger female participants, who were of a lower social class and were more likely to smoke ($P<0·001$). Pattern 3 was observed in younger participants, with a slightly higher proportion of male participants than female, with the greatest energy intakes ($P<0·001$). Participants in Pattern 4 were typically older men and women, who presented the poorest AHEI scores ($P<0·05$). Participants in Patterns 3 and 4 were typically non-manual or skilled manual workers (Table 2).

Mean daily intakes, including contributions from supplements, are presented in Tables 3 and Table 4. Carbohydrate (%TE), sugar (%TE) and fibre (g/10 MJ) intakes were significantly greater in Pattern 1 ($P<0·001$). Total fat (%TE) intakes were greatest in Patterns 2 and 4, with Pattern 4 presenting significantly greater SFA (%TE) intakes than the other three patterns. MUFA (%TE), PUFA fat (%TE) and α-linolenic acid (%TE) intakes were greatest in Pattern 2 ($P<0·001$), with no significant differences in EPA (%TE) and DHA (%TE) levels between patterns. Na (mg/10 MJ) intakes were significantly lower, whereas intakes of other micronutrients were significantly greater in Pattern 1 (Table 3). In comparison with the other dietary patterns, plasma EPA (C20 : 5*n*-3) and DHA (C22 : 6*n*-3) levels were higher in Pattern 1 ($P<0·001$). No significant differences were observed in a suite of markers of CVD and T2D (Table 4).

Discussion

Overall, four dietary patterns were derived, which were distinguishable by both processed red meat consumption and other dominant food groups. The pattern with greater processed red

Table 1. Percentage of energy contribution of food groups across four dietary patterns in Irish adults (Mean values and standard deviations)

	Pattern 1 (*n* 131)		Pattern 2 (*n* 70)		Pattern 3 (*n* 405)		Pattern 4 (*n* 180)		
Food groups	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**
Processed red meat	2·65^{a}	3·55	4·21^{a,b}	3·94	4·46^{b}	4·20	7·86^{c}	7·13	<0·001
Unprocessed red meat	5·34^{a}	4·67	4·64^{a}	4·30	7·12^{b}	5·93	7·08^{b}	5·31	<0·001
Processed white meat	0·91	2·84	1·82	2·22	1·86	3·19	1·16	2·52	0·066
Unprocessed white meat	3·07	3·57	4·23	4·78	3·57	4·14	2·58	3·10	0·198
Alcoholic beverages	3·56^{a}	4·91	7·94^{b}	8·72	8·16^{b}	8·88	3·54^{a}	5·18	<0·001
Biscuits, cakes and pastries	6·76^{a}	5·57	3·95^{b}	4·67	6·74^{a}	6·46	5·61^{a,b}	5·55	0·033
Butters, fat spreads and cooking fats	2·40^{a}	2·87	2·38^{a}	2·25	2·54^{a}	2·54	6·83^{b}	6·79	<0·001
Cheeses	1·98^{a,c}	2·20	3·33^{b}	2·87	2·45^{a,b}	2·91	1·82^{c}	2·37	<0·001
Chips and processed potatoes	2·71^{a}	3·06	5·09^{b}	5·22	4·84^{b}	4·82	4·71^{b}	5·37	<0·001
Confectionery	2·56^{a}	3·26	3·51^{a,b}	4·19	3·61^{b}	4·30	2·20^{a}	2·92	0·033
Creams, ice creams and desserts	3·15^{a}	4·36	2·79^{a,b}	4·21	1·99^{b}	2·96	1·69^{b}	2·71	<0·001
Egg and egg dishes	1·92	2·59	1·60	2·65	1·35	1·92	1·26	1·60	0·759
Fish, fish dishes and products	4·25^{a}	4·55	1·84^{b}	2·70	1·94^{b}	2·86	1·68^{b}	2·59	<0·001
Fruit	6·42^{a}	4·51	1·73^{b}	2·42	2·06^{b}	2·04	1·91^{b}	2·52	<0·001
Fruit juices and smoothies	1·40^{a,b}	2·42	1·63^{a,c}	1·88	0·96^{b}	1·60	0·64^{c}	1·45	<0·001
High-energy beverages	0·85	2·13	1·95	3·19	1·85	2·96	1·51	2·81	0·198
Low-energy beverages	0·31^{a}	0·82	0·11^{a,b}	0·46	0·10^{b}	0·34	0·15^{a,b}	0·59	0·033
Low-fat and skimmed milk	2·25	2·73	1·33	1·78	2·31	3·54	1·86	3·46	0·264
Low-fat spreads and oils	1·20^{a,c}	1·91	0·47^{a,b}	1·15	0·69^{b}	1·53	1·33^{c}	2·70	<0·001
Other breakfast cereals	2·23	3·74	1·53	3·15	1·15	2·83	1·92	4·33	0·165
Other milk products and milk-based beverages	0·62	1·57	0·46	1·13	0·42	1·45	0·23	1·22	1·000
Potatoes	2·97^{a}	2·65	1·61^{b}	2·15	2·21^{b}	2·14	4·13^{c}	3·68	<0·001
Rice, pasta, flours and starches	2·80^{a,b}	4·11	3·24^{a}	3·88	2·11^{b}	2·84	1·03^{c}	1·97	<0·001
Ready-to-eat breakfast cereals	3·67^{a}	4·59	2·40^{a}	2·85	5·07^{b}	5·05	2·64^{a}	3·41	<0·001
Savouries	1·78^{a}	3·16	3·02^{a,b}	4·56	4·31^{b}	6·16	1·95^{a}	4·15	<0·001
Savoury snacks	3·78^{a}	5·74	2·02^{b,c}	2·22	2·67^{b}	3·83	1·41^{c}	2·56	<0·001
Soups, sauces and condiments	2·55^{a}	2·42	1·91^{b}	1·59	1·96^{c}	1·60	1·73^{c}	1·91	<0·001
Sugars, syrups, preserves and sweeteners	1·57^{a}	2·25	1·24^{a}	1·67	1·37^{a}	1·89	3·11^{b}	4·04	<0·001
Vegetables and vegetable dishes	7·06^{a}	4·71	3·25^{b}	2·67	2·55^{b}	1·78	2·51^{b}	2·03	<0·001
White bread, rolls, scones and croissants	4·75^{a}	4·35	7·83^{b}	5·84	6·00^{a,b}	4·48	13·78^{c}	7·81	<0·001
Wholemeal, brown bread and rolls	7·98^{a}	6·06	5·03^{b,c}	4·86	6·46^{a,b}	6·12	4·22^{c}	5·47	<0·001
Whole milk	1·52^{a}	3·01	2·47^{a,b}	3·41	3·42^{b}	4·62	4·65^{c}	6·12	<0·001
Yogurts	2·04^{a}	2·63	1·03^{b}	1·83	1·23^{b}	2·00	0·89^{b}	1·76	<0·001

^{a,b,c} Mean values with unlike superscript letters are significantly different between groups ($P<0·05$).

* Differences across dietary patterns were assessed using one-way ANOVA. Bonferroni correction was applied by multiplying the *P* values by the number of traits in the table. *P* values that exceeded 1·0 have been marked down to 1·000.

Table 2. Participant characteristics across the four dietary patterns (Mean values and standard deviations)

	Pattern 1 (*n* 131)		Pattern 2 (*n* 70)		Pattern 3 (*n* 405)		Pattern 4 (*n* 180)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P*§
Processed red meat (g/d)*	25·8^{a}	22·9	39·2^{b}	35·0	34·3^{a,b}	22·7	57·1^{c}	46·6	<0·001
Unprocessed red meat (g/d)*	66·9^{a}	43·0	95·9^{b}	63·5	75·7^{a,b}	56·9	86·2^{a,b}	57·9	<0·001
Processed white meat (g/d)*	26·7	27·8	32·8	29·4	22·7	23·5	29·2	23·4	1·000
Unprocessed white meat (g/d)*	69·6	50·3	76·1	61·8	80·0	60·2	62·4	40·8	1·000
Sex (male/female, %)†	34/66		36/64		56/44		57/43		<0·001
Age (years)*	48·4^{a}	15·7	33·4^{b}	13·0	38·9^{c}	15·9	48·6^{a}	17·2	<0·001
Social class (%)†									
Professional	65·4		50·7		44·4		41·7		<0·001
Non-manual	15		16·4		16·2		14·3		
Skilled manual	10·2		3·0		13·4		21·7		
Unskilled	9·4		29·9		26·0		22·3		
Smoker (%)†	13		32·9		16·6		28·9		<0·001
Supplement user (%)†	48·9		31·4		29·7		21·7		<0·001
Physical activity (h/week)*	84·3	58·1	114·3	104·8	103·8	84·2	90·0	85·7	0·400
Energy (kJ/d)	8636^{a}	2381	9728^{b,c}	2356	10 134^{b}	2636	9360^{a,c}	2360	<0·001
Energy (kcal/d)*	2064^{a}	569	2325^{b,c}	563	2422^{b}	630	2237^{a,c}	564	<0·001
BMI (kg/m^{2})‡	25·4	3·9	25·7	4·5	26·0	4·0	26·8	4·4	1·000
Body fat (%)‡	29·3	8·0	28·1	9·6	26·6	9·3	28	8·5	1·000
Muscle mass (kg)‡	47·6	10·2	50·9	10·7	53	11·1	52·1	10·1	1·000
Waist:hip ratio (cm)‡	0·86	0·08	0·83	0·07	0·87	0·08	0·9	0·09	0·920
Systolic BP (mmHg)‡	121·8	17·7	118·1	15·5	124·3	17·1	126·8	20·1	1·000
Diastolic BP (mmHg)‡	77·6	11·2	75·9	10·3	77·6	11	79	11·5	1·000
Metabolic syndrome (%)†	11·5		7·1		17·5		20·6		0·240
Alternate Healthy Eating Index‡	34·4^{a}	9·9	25·7^{b}	7·4	24·9^{b}	8·5	21·2^{c}	7·7	<0·001

BP, blood pressure.
^{a,b,c} Mean values with unlike superscript letters are significantly different between groups ($P<0·05$).
* Differences across meat consumption and demographics were assessed by one-way ANOVA.
† Differences across sex, social class, smoking and supplement use were assessed using the Pearson χ^2 Statistic.
‡ Differences across anthropometric measurements and dietary quality were assessed using a general linear model adjusted for age, sex, energy, social class, smoking status and supplement usage.
§ Bonferroni correction was applied by multiplying the *P* values by the number of traits in the table. *P* values that exceeded 1·0 have been marked down to 1·000.

Table 3. Nutrient composition across four dietary patterns (Mean values and standard deviations)

	Pattern 1 (*n* 131)		Pattern 2 (*n* 70)		Pattern 3 (*n* 405)		Pattern 4 (*n* 180)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**
Carbohydrate (%TE)	44·6^{a}	6·4	39·6^{b}	5·7	42·3^{a}	6·8	43·0^{a}	7·0	<0·001
Sugars (%TE)	19·9^{a}	5·6	15·7^{b}	4·4	16·9^{b}	5·2	16·4^{b}	6·2	<0·001
Protein (%TE)	16·8	3·4	15·2	3·0	16·2	3·2	16·0	3·1	1·000
Total fat (%TE)	34·2^{a,b}	6·2	36·0^{b,c}	6·7	33·1^{a}	5·7	36·0^{c}	6·1	<0·001
SFA (%TE)	12·6^{a}	3·3	12·7^{a}	3·2	13·1^{a}	3·1	14·7^{b}	3·7	<0·001
MUFA (%TE)	12·2^{a,c}	2·5	13·9^{a}	3·2	12·1^{b}	2·3	13·1^{b,c}	2·7	<0·001
PUFA (%TE)	7·2^{a}	2·7	7·2^{a}	2·4	5·8^{b}	2·0	5·9^{b}	2·3	<0·001
ALA (%TE)	0·7^{a}	0·4	0·8^{a}	0·3	0·5^{b}	0·2	0·6^{a}	0·6	<0·001
EPA (%TE)	0·12	0·67	0·01	0·04	0·02	0·17	0·03	0·23	1·000
DHA (%TE)	0·17	0·66	0·01	0·04	0·02	0·16	0·03	0·23	1·000
Fibre (g/10 MJ)	30·6^{a}	8·4	20·0^{b,c}	6·2	21·4^{b}	6·7	19·9^{c}	6·6	<0·001
Vitamin A (μg/10 MJ)	1830·5^{a}	1146·2	1095·6^{a,b}	766·1	1179·5^{b}	988·2	1208·2^{b}	809·5	<0·001
Vitamin B_6 (mg/10 MJ)	6·6	13·1	3·7	4·8	4·6	9·1	3·9	7·3	1·000
Vitamin B_{12} (μg/10 MJ)	21·3	113·4	5·4	4·5	6·6	10·0	6·1	7·8	1·000
Biotin (μg/10 MJ)	91·2	207·0	45·0	28·2	54·7	43·1	47·1	30·6	0·572
Riboflavin (mg/10 MJ)	5·4	12·7	3·1	5·9	3·9	9·5	3·0	9·8	1·000
Vitamin C (mg/10 MJ)	269·1^{a}	446·2	134·8^{b}	110·6	125·9^{b}	180·4	100·0^{b}	122·9	<0·001
Vitamin D (μg/10MJ)	9·7^{a}	14·6	3·4^{b}	4·1	4·6^{b}	5·0	5·1^{b}	5·7	<0·001
Ca (mg/10 MJ)	1191·0	534·3	1008·7	394·8	1099·5	390·5	1112·3	431·7	1·000
Cu (mg/10 MJ)	1·8	1·1	1·2	0·4	1·4	1·8	1·2	0·8	1·000
Fe (mg/10 MJ)	17·9	10·6	14·5	8·5	16·3	17·1	16·8	20·9	1·000
Mg (mg/10 MJ)	417·5^{a}	152·2	312·7^{b,c}	64·2	340·2^{b}	79·1	307·4^{c}	73·0	<0·001
K (mg/10 MJ)	4115·3^{a}	705·8	3419·6^{b,c}	564·5	398·0^{b}	771·3	3389·0^{c}	747·2	<0·001
P (mg/10 MJ)	1735·3^{a}	301·1	1495·5^{b}	286·7	1599·2^{a,b}	277·5	1561·4^{b}	339·5	<0·001
Na (mg/10 MJ)	2787·8^{a}	638·2	2902·1^{a,b}	454·8	2838·3^{a}	601·5	3106·9^{b}	610·9	<0·001
Zn (mg/10 MJ)	15·8	17·4	9·6	2·9	11·6	4·9	11·7	7·1	0·130

%TE, percentage of total energy; ALA, *α*-linolenic acid.
^{a,b,c} Mean values with unlike superscript letters are significantly different between groups ($P<0·05$)
* Differences in nutrient intakes across dietary patterns were assessed using a General Linear Model adjusted for age, sex, social class, smoking status and supplement usage. Bonferroni correction was applied by multiplying the *P* values by the number of traits in the table. *P* values that exceeded 1·0 have been marked down to 1·000.

Table 4. Plasma fatty acid levels and markers of metabolic health across the four dietary patterns (Mean values and standard deviations)

	Pattern 1 (*n* 100–130)		Pattern 2 (*n* 57–70)		Pattern 3 (*n* 291–403)		Pattern 4 (*n* 130–179)			
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**	*P*†
% Total plasma fatty acids										
C15 : 0	0·33	0·09	0·32	0·10	0·38	1·04	0·35	0·10	0·900	1·000
C16 : 0	22·60	2·92	22·50	1·64	23·00	2·22	22·91	1·95	0·146	1·000
C16 : 1	2·23[a]	0·74	2·34[a,b]	6·70	2·56[b]	0·88	2·62[b]	0·89	<0·001	<0·001
C18 : 1*n*-9	17·90	3·21	18·50	4·47	18·20	3·42	19·03	4·15	0·035	1·000
C18 : 3*n*-3	0·81	0·28	0·88	0·30	0·83	0·25	0·93	0·36	0·001	0·456
C18 : 3*n*-6	0·51	0·19	0·58	0·30	0·56	0·17	0·56	0·20	0·058	0·144
C20 : 1*n*-9	0·24	0·07	0·23	0·09	0·22	0·96	0·23	0·07	0·030	0·816
C20 : 3*n*-6	1·87[a]	0·50	1·95[a,b]	0·43	2·08[b,c]	0·48	2·07[c]	0·42	<0·001	<0·001
C20 : 4*n*-6	7·69	2·06	7·73	1·98	7·57	1·86	7·44	1·93	0·611	1·000
C20 : 5*n*-3	1·83[a]	1·42	1·27[a,b]	0·61	1·26[b]	0·70	1·34[b]	0·72	<0·001	<0·001
C22 : 4*n*-6	0·23	0·08	0·22	0·08	0·24	0·09	0·25	0·10	0·070	1·000
C22 : 6*n*-3	3·03[a]	1·20	2·30[b]	0·81	2·30[b]	0·81	2·21[b]	0·84	<0·001	<0·001
C23 : 0	0·60	4·33	0·16	0·07	0·18	0·09	0·17	0·09	0·123	1·000
C24 : 0	0·25	0·15	0·24	0·10	0·26	0·14	0·24	0·10	0·223	1·000
Metabolic health										
Glucose (mmol/l)	5·19	0·88	5·09	0·73	5·20	0·81	4·58	1·31	0·004	1·000
Insulin (μIU/ml)	9·52	10·97	10·30	9·48	12·10	13·73	15·82	25·79	0·006	1·000
TAG (mmol/l)	1·11	0·59	1·09	0·65	1·32	0·83	1·41	0·82	0·001	1·000
Total cholesterol (mmol/l)	5·01	0·92	4·72	0·90	4·94	1·06	4·97	0·99	0·255	1·000
HDL-cholesterol(mmol/l)	1·70	0·48	1·66	0·39	1·55	0·40	1·54	0·41	0·001	1·000
LDL-cholesterol (mmol/l)	2·78	0·78	2·57	0·77	2·80	0·88	2·80	0·93	0·245	1·000
Adiponectin (μg/ml)	7·08	4·51	6·59	3·01	5·89	2·93	5·81	2·68	0·001	1·000
Leptin (ng/ml)	5·29	6·03	6·14	5·85	5·29	6·85	5·10	6·85	0·798	1·000
Homocysteine (mmol/l)	11·5	3·92	11·9	2·88	12·00	3·35	13·49	5·01	<0·001	0·672
TNF*α* (pg/ml)	6·66	3·05	6·17	1·62	6·72	1·96	7·52	2·88	<0·001	1·000

[a,b,c] Mean values with unlike superscript letters are significantly different between groups ($P < 0·05$).
* Differences in fatty acids and markers of metabolic health across dietary patterns were assessed using a one-way ANOVA.
† Differences in fatty acids and markers of metabolic health across dietary patterns were assessed using general linear model adjusted for age, sex, energy (kJ (kcal)), social class, smoking status, supplement usage and fasting status. Bonferroni correction was applied by multiplying the *P* values by the number of traits in the table. *P* values that exceeded 1·0 have been marked down to 1·000.

meat intakes presented a poorer AHEI score and a lower *n*-3 PUFA status compared with the pattern with significantly lower contributions from processed red meat; however, there were no significant differences in traditional biomarkers of CVD and T2D between the patterns.

Total red meat intake in the current analysis was 134 g/d in men and 89 g/d in women, with 46 % adhering to the recommendation of ≤500 g/week[4]. In all, 85 % of the cohort consumed processed red meat; intakes of which are recommended to be limited[4]. Men (52 g/d) presented greater mean daily processed red meat intakes than women (29 g/d). Irish intakes are slightly higher than intakes in the UK, similar to those in Spain, Sweden and Denmark, and lower than those in Germany[22–24]. However, it must be noted that much of the dietary intake data in the aforementioned studies were collected over a decade ago and the applied definitions differ slightly. Data exist for processed red meat intakes in the USA (male 29 g/d, female 18 g/d); again this is not directly comparable due to the definitions applied[25]. This lack of a stringent, global definition for processed meat is one of the major limitations when investigating processed red meat consumption as a risk factor for disease. Further research is required to ascertain whether there is greater risk associated with specific products as opposed to total processed red meat.

The current analysis included red meat that underwent smoking, salting, curing, fermentation or other processing to enhance flavour or improve preservation[16]. It is important to consider the effects of these processing techniques, and the added ingredients, including salt, which may be contributing to the observed association between processed red meat and risk for incident CVD and T2D[2,6]. In a review by Micha *et al.* which included twenty-seven observational studies (CVD; *n* 10; T2D; *n* 17) from ten countries, studies presented varying quantities of mean daily red meat intakes, with differing processed meat definitions and differing levels of confounder adjustment outlining the difficulties in reviewing this area[2]. Furthermore, high processed meat consumers were characterised by less-favourable dietary and lifestyle factors[2]. However, recent studies are inconsistent with the aforementioned results, with no causal association observed between total red meat intakes and biomarkers of CVD using randomised controlled trials, with similar biomarker levels to the current study[7]. Further, it has been noted that the observed association between both processed and unprocessed red meat and biomarkers of T2D were attenuated following adjustment for confounding factors, particularly BMI, and multiple comparisons[8]. There were significant differences between processed red meat consumption and biomarkers of health in the current cohort; however, this was before adjustment for confounding factors and Bonferroni correction (online Supplementary Table S2).

Dietary patterns have been associated with predicting risk of disease; processed red meat is typical of the Western-style diet,

which has been associated with an increased risk for CVD and T2D[9]. However, a recent study found no association between a 'meat and fish' pattern and 10-year CVD risk, whereas 'refined foods' including soft drinks and alcohol were associated with a predicted 10-year CVD risk in a Mexican cohort using factor analysis (relative risk 2·98; 95 % CI 1·46, 6·10; $P_{trend} = 0{\cdot}020$)[26]. As the majority of studies to date have focused on high processed red meat intakes, not the overall diet, and on the incidence of disease, the aim of this analysis was to characterise the contributions of red meat to dietary patterns in a European cohort. With four dominant dietary patterns derived, it was observed that Pattern 1 was similar to the Mediterranean pattern with Patterns 2 and 3 comprising components of the Western pattern[9]. Pattern 4 had a significantly greater contribution from processed red meat than the other dietary patterns. Other dominant food groups in this pattern included butters and whole milk, and lower contributions from fruit, vegetables and fish, consistent with significantly lower plasma EPA and DHA levels; however, only plasma DHA remained significant after exclusion of fish-oil supplement users (*n* 94). Mean EPA and DHA intake in the total population (*n* 786) was 120 mg/d, lower than the European Food Safety Authority (EFSA) recommendation of 250 mg/d[27]. Pattern 1 was the sole achiever of the EPA and DHA recommendation (342 g/d), potentially due to greater dietary intakes of fish, which was reflected in their plasma fatty acid levels.

Similar to previous studies, participants in the high processed red meat pattern were typically older and of a lower social class; 29 % were current smokers with a significantly lower AHEI score and dietary fibre intake and greater SFA intakes. This is consistent with the findings of the study by Li *et al.* in which older participants presented greater SFA intakes[28]. However, unlike other analyses investigating processed red meat and disease risk, the current study had access to a suite of blood biomarkers of CVD and T2D, to complement the dietary intake data. Significant differences were observed between dietary patterns and biomarkers of CVD and T2D; however, this was attenuated when confounding factors were included in the model (Table 4). The possibility that red meat may be associated with increased non-traditional biomarkers should also be investigated. It is evident that further research is required to confirm the degree of association between processed red meat consumption and development of cardio-metabolic diseases, with careful consideration of the definition applied, the processing procedures and the residual confounding factors. A Mendelian randomisation approach as suggested by Rohrmann & Linseisen may be a potential strategy[6].

The large, nationally representative cohort, the 4-d semi-weighed food diaries, and product information at brand level, coupled with metabolic biomarkers, strengthened the current analysis, whereas the inclusion of plasma fatty acid data provided a novel aspect. Dietary intakes were self-reported; thus, the removal of under-reporters eliminated potential reporting bias, and the Bonferoni correction for multiple comparisons strengthened the statistical analysis. However, the cross-sectional nature of the NANS is a limitation, as we cannot comment on the causal relationship between processed red meat and these diseases but merely state that we failed to observe an association between the dietary patterns and traditional blood biomarkers. This analysis was also limited to processed red meat contributions to dietary patterns. The application of cluster analysis to derive dietary patterns may have potentially resulted in a loss of statistical power, as it classified participants into an individual pattern, in comparison with factor analysis in which individuals receive a factor score for all derived dietary patterns. Further, the NANS was typical of a healthy cohort; findings may differ in an at-risk or diseased cohort.

In conclusion, no association was observed between high consumption of processed red meat and biomarkers of CVD and T2D in the current cohort. This finding is similar to those from the meta-analyses by O'Connor *et al.* and Fretts *et al.*, who failed to find an association between red meat consumption and CVD[7] and T2D[8]. Similar to other cohorts, high consumers of processed red meat presented a more unfavourable diet and lifestyle, which needs to be considered when investigating the association between processed red meat consumption and incidence of CVD and T2D. This analysis supports previous findings that emphasise overall dietary quality as a measure of health, rather than intakes of single foods and nutrients. Therefore, future public health recommendations should consider focusing on the total diet, based on the conflicting evidence for the role of processed red meat in disease risk. Furthermore, a global definition of processed meat should also to be developed, and modification of ingredients, similar to salt reductions, may be an effective public health strategy to improve the quality of processed red meat.

Acknowledgements

The authors are grateful for the assistance of Ricardo Segurado (Statistician at CStar, www.cstar.ie) for advice on statistical components of the study, and the NANS participants for providing detailed dietary intake data and blood samples.

This research was supported by the Irish Department of Agriculture, Food and the Marine under the National Development Plan (2007–2013) (grant no. 13/F/514) and the Health Research Board under their joint Food for Health Research Initiative (2007–2012) (grant no. FHRIUCC2).

The authors' contribution are as follows: B. A. M., A. P. N., H. M. R., L. B., A. F. and J. W. designed the research, Y. M. L., B. A. M., A. P. N. and K. F. L. conducted the research, Y. M. L. and B. A. M. analysed the data and prepared the manuscript. All authors read and approved the final manuscript.

None of the authors has any conflicts of interest to declare.

Supplementary material

For supplementary material/s referred to in this article, please visit https://doi.org/10.1017/S0007114517002008

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