RED AND PROCESSED MEAT AND CARDIOVASCULAR RISK FACTORS

BRUNO ATALIĆ, JURICA TOTH, VLASTA ATALIĆ, DANIJELA RADANOVIĆ, MAJA MIŠKULIN and ANA LUČIN

Department of Public Health and Primary Care, University of Cambridge, United Kingdom, [1]Department of Emergency Medicine, Zagreb, [2]Institute of Public Health for Osijek-Baranja County, Osijek, [3]Department of Internal Diseases, General Hospital Vukovar, and [4]Health Centre East, Zagreb, Croatia

Aims: The British National Diet and Nutrition 2000/1 Survey data set records on 1,724 respondents (766 males and 958 females) were analyzed in order to assess the potential influences of red and processed meat intakes on cardiovascular risk factors. *Methods:* Linear regression of the associations of the red, processed, combination of red and processed, and total meat intakes with body mass index (BMI), systolic blood pressure and plasma total cholesterol as cardiovascular risk factors was conducted, paying due attention to the subject age and sex as potential confounders. *Results:* Linear analyses showed the total meat intake and combined red and processed meat intake to cause a 1.03 kg/m^2 rise in BMI each, while the red and processed meat intakes analyzed as separate categories caused 1.02 kg/m^2 rise each. The greatest effects were observed on the systolic blood pressure with a 1.7 mm Hg rise for the total and the red and processed meat intakes, 1.5 mm Hg rise for the red meat intake, and 1.02 mm Hg rise for the processed meat intake. There were no associations between different meat intakes and plasma total cholesterol. *Discussion and Conclusion:* Study results revealed the interquartile ranges of the mentioned meat type intakes to increase BMI by around 1 kg/m^2 and systolic blood pressure by around 1.5 mm Hg, while they had no influence on plasma total cholesterol.

Key words: cardiovascular diseases; cardiovascular risk factors; body mass index; systolic blood pressure; plasma total cholesterol

Address for Correspondence: Bruno Atalić, MD, MSc (Oxon), PhD, MPhil (Cantab)
Hrvatskoga Proljeća 24
10000 Zagreb, Croatia
E-mail: bruno.atalic@cantab.net
Tel: 00 385 99 6 734 736

Research was conducted during the first author's education at the Department of Public Health and Primary Care of the University of Cambridge.

INTRODUCTION

The Committee on Medical Aspects of Food and Nutrition Policy (COMA) recommended in 1998 that high consumption of red and processed meat should be reduced due to the risk of colorectal cancer (1). Similarly, the 2005 US Dietary Guidelines for Americans recommend that the intake of red and processed meat be moderated due to their impact on plasma cholesterol (2). The latter recommendation is based on the 'diet-heart hypothesis' from the 1930s (3). It states that a diet rich in high saturated fats and cholesterol and poor in low polyunsaturated fats leads to high plasma cholesterol levels, which favor the development of atheromatous plaques, arterial narrowing and cardiovascular diseases (CVDs). Its major opposition is the fact that total cholesterol is present in serum not only as low-density lipoprotein (LDL) cholesterol, intermediate-density lipoprotein (IDL) cholesterol and very low-density lipoprotein (VLDL) cholesterol, which are positively correlated with the risk of CVDs, but also as high density lipoprotein (HDL) cholesterol, which is inversely related to the development of CVDs.

Ecological studies have discovered the association between low saturated fat intakes and low coronary death rates (CDRs) in less industrialized countries in comparison with high saturated fat intakes and high CDRs in highly industrialized countries (4-10). Migrant studies investigating CDRs in the populations of

Japanese living in Japan, Honolulu and San Francisco demonstrated the associations between their saturated fat intakes and their age adjusted incidence of CDRs (11,12). The Multiple Risk Factors Intervention Trial showed the relationship between the rise in serum cholesterol levels and rise in CDRs (13-18). Case-control studies have reported an increased risk of myocardial infarction (MI) in Italian women taking high levels of meat, butter and total fat and low levels of fish, vegetables and fruits (19) and in Greek women cooking on margarine (20). On the contrary, various cohort studies found men and women with higher total energy intakes to have lower CDRs, which they tended to explain by confounding with their higher levels of physical activity (21-25).

A meta-analysis conducted by Lewington *et al*., based on 61 prospective observational studies from Western Europe and North America and including 900,000 adults without previous disease has shown that age, sex, and blood pressure could modify the associations of total LDL and HDL cholesterol levels with vascular mortality (26). A prospective cohort study by Clarke *et al*. that included 5,360 men, mean age 77, followed for seven years, showed that vascular mortality was positively associated with logarithmic C-reactive protein, fibrinogen and total/HDL cholesterol, and inversely with albumin (27). Another study revealed that heart disease mortality was positively associated only in men without CVDs and inversely in men with CVDs (28). Another prospective cohort study conducted by Clarke *et al*. in 18,863 men employed in civil service in London, England, examined at entry in 1967-1970 and followed for 38 years, showed an association of baseline smoking, blood pressure and serum cholesterol levels with 10- to 15-year shorter life expectancy from age 50 (29). Numerous epidemiological surveys of systolic blood pressure (SBP) levels in different populations linked diets containing little meat and fish to low mean blood pressures (30).

Sacks *et al*. and Sacks and Kass have proposed a relation between SBP levels and intake of food from animal sources (31,32). Steffen *et al*. observed that plant food intake was inversely related, dairy intake was not related, while red and processed meat intakes were positively related to elevated blood pressure (33). Miura *et al*. examined relations of food intakes to SBP changes in the Chicago Western Electric Study and found that men with a higher intake of red and white meat had a significantly greater increase in blood pressure, while higher intakes of vegetables, fruits and fish were related to less of an increase in SBP and diastolic blood pressure (DBP) over time (34). Appel *et al*. designed a clinical trial called Dietary Approaches to Stop Hypertension (DASH) as a multicentre randomized feeding study and thus managed to demonstrate that certain dietary patterns independently of their sodium content could favorably affect blood pressure in adults with average SBP of less than 160 mm Hg and DBP of 80 to 95 mm Hg (35).

In their case-control study, Kontogianni *et al*. demonstrated the red meat intake to be strongly associated with 52% increased odds of acute coronary syndrome (ACS), while white meat intake seemed to be associated with only 18% likelihood of sustaining CVD events (36). Sinha *et al*. conducted a prospective cohort study called Diet and Health Study and showed that men and women in the highest *versus* lowest quintile of red and processed meat intakes had elevated risk of overall mortality and cause-specific mortality from cancer and CVDs (37). Micha *et al*. conducted a systematic review and meta-analysis of 17 prospective cohort and three case-control studies and showed that the intake of red meat was not associated with either chronic heart disease (CHD) or diabetes mellitus (DM), processed meat was associated with 42% higher risk of CHD and 19% higher risk of DM, and total meat intake showed intermediate associations, while neither meat type intake was associated with stroke (38).

The cited literature is mainly based on the assumption that the effect of meat on vascular risk is mediated only by the effect of saturated fats and dietary cholesterol on blood cholesterol levels. On this track, this paper will look at the associations between the intakes of different meat types (red, processed, red + processed, total) and CVD risk factors (BMI, SBP, and plasma total cholesterol (PTC)) according to the British National Diet and Nutrition Survey data set from 2000/1 (NDNS-DS) (39).

PATIENTS AND METHODS

The NDNS public access data set on adults aged 19-64 carried out in 2000/01 is available on request from the United Kingdom National Statistics Public Enquiry Service (39). It is comprised of 312,631 individual food item records from a total of 12,068 person-days of recording by 1,724 respondents (766 males and 958 females), recorded through weighted intakes for seven days. Records were coded using 4,612 different food codes from the NDNS food database of 7,374 codes (39). The main NDNS-DS reports on food intakes were not based on full disaggregation of composite dishes and they were derived by Aston *et al*. (40). Primary food constituents were identified according to a set of 45 project-specific food categories. Composite dishes like 'beef, veal and dishes', 'lamb and dishes' or 'meat, meat dishes and meat products' were fully disaggregated into primary ingredients (40).

Statistical analysis, which included 766 male and 958 female respondents aged 19 to 64 was conducted by the PASW version 18. Due to their observed adverse influence in the development of CVDs, red (meat from mammals) and processed (meat products) meat intakes were chosen for analysis rather than white (poultry) meat intakes. In order to asses their possible additive or multiplicative effects, the already created red + processed meat intake variable was also included. Finally, in order to evaluate the effects of meat by itself, total meat intakes were chosen as well. Sex and age were chosen as confounding factors due to their known effects on both meat intake and CVDs morbidity and mortality rates. Considering cardiovascular risk factors, BMI was chosen because of both its relevance and robustness. SBP was chosen rather than DBP due to its greater importance in the development of CVDs. PTC was chosen rather than adverse LDL cholesterol or protective HDL cholesterol. On the one hand, LDL cholesterol could not have been chosen because it was not directly measured during data collection, but was only later calculated from PTC by using Friedewald's formula, which has obviously affected its robustness. On the other hand, HDL cholesterol was dismissed because it is currently perceived as a protective factor against CVDs, while the purpose of this paper was to assess adverse factors for the development of CVDs and in this respect, it was not so relevant.

In order to assess the normality of distribution of the seven-day mean total, red + processed, red and processed meat intakes, Smirnov-Kolmogorov tests were performed and histograms of distributions produced. Only, after logarithm (ln) transformation of the cases that remained after dropping the mentioned zero values cases, it was possible to obtain normal distributions. Linear regression was performed in order to assess the potential impact of the red, processed, red + processed and total meat intakes on BMI (weight in kg/height in m^2), SBP (mean of two systolic measurements in mm Hg) and PTC (plasma total cholesterol in mm/L) as cardiovascular risk factors, while paying attention to subject age and sex as potential confounders. The mentioned potential confounders were analyzed both independently and in combination and a new age by sex variable was created in order to asses their possible additive or multiplicative effects, while in order to reduce confounding by body size and energy turnover, the energy adjusted meat intakes were used, expressed as g/MJ (grams/mega joule) of total energy recorded over seven days of dietary recording; in order to respond to their lack of normal distribution, their interquartile ranges (IQRs) were used in calculations instead of their standard deviations (SDs). Results were described as significant if their p values were less than 0.05 and as highly significant if their p values were less than 0.01; all p values reported in this paper were obtained by two-tailed tests. Bearing in mind the general robustness of linear regression, it was decided to use the total, red + processed meat, red and processed meat intakes as recorded in the NDNS-DS in all cases, and ln transformations of the mentioned variables after dropping the cases with zero values in linear regression. Linear regression models were built for the mentioned cardiovascular risk factors as a dependent variable and each of the mentioned meat type intakes combined with sex, age and age by sex as independent variables. The preferred model was the one that had the greatest adjusted R^2 value for the least number of the confounding variables (sex, age, and age by sex). Descriptive statistical analysis showed that BMI had abnormal distribution, so it was ln transformed into lnBMI, while SBP and PTC had normal distributions. As mentioned above, the total, red + processed meat, red and processed meat intakes, and ln transformations of the mentioned variables after dropping the cases with zero values were used in linear regression.

RESULTS

Distributions and correlations of the intakes of different meat types are shown in Tables 1 and 2. After linear regression, the preferred models for lnBMI and PTC with particular meat type intakes were those that also included subject age, while for SBP they included both age and sex of respondent. Histograms and scatter plots of the preferred models were produced and showed normality of distribution and independence of residuals. Nonstandardized coefficients (B) for constant, meat type intakes and age and sex were extracted together with their p values. They were used to calculate the effects of IQRs of the meat type intakes on the CVD risk factors analyzed according to the formulas:

$$\text{lnBMI} = B0 + B1 \times \text{age of respondent} + B2 \times \text{IQR meat type intakes}$$

$$\text{SBP} = B0 + B1 \times \text{sex of respondent} + B2 \times \text{age of respondent} + B3 \times \text{IQR meat type intakes}$$

$$\text{PTC} = B0 + B1 \times \text{age of respondent} + B2 \times \text{IQR meat type intakes}$$

Results are shown in Table 3.

Table 1

Interquartile range (IQR) of different meat type intakes

	All consumers (N=1724) (g/MJ)	ln excluding zero consumers (ln g/MJ)
Total meat	8.23	0.61 (n=1640)
Red plus processed meat	7.12	0.82 (n=1582)
White meat	5.08	0.93 (n=1339)
Red meat	5.28	0.94 (n=1390)
Processed meat	4.31	0.93 (n=1338)

Table 2

Bivariate Pearson's correlation coefficients (r) between different meat type intakes

	All consumers r (p)	Excluding zero consumers r (p)
Red meat and processed meat	0.114 (<0.001)	0.121 (<0.001)
Red meat and white meat	-0.018 (0.467)	-0.061 (0.015)
Processed meat and white meat	-0.19 (0.428)	-0.010 (687)
Red + processed meat and white meat	-0.24 (0.314)	-0.082 (0.001)

Table 3

Effect sizes for increase in each meat variable by its interquartile range (IQR)

Type of meat	IQR	BMI (kg/m^2)	SBP (mm Hg)	PTC (mmol/L)*
	All cases (ln without zero cases)	All cases (ln without zero cases)	All cases (ln without zero cases)	All cases (ln without zero cases)
Total	8.23 (0.61)	1.033 (1.029)	1.679 (1.409)	-0.00876 (0.01952)
Red and Processed	7.12 (0.82)	1.029 (1.026)	1.744 (1.460)	-0.00712 (-0.00164)
Red	5.28 (0.94)	1.021 (1.024)	1.536 (1.320)	0.04224 (0.07144)
Processed	4.31 (0.93)	1.022 (1.011)	1.022 (0.365)	-0.06465 (-0.11625)

*While all the estimates for body mass index (BMI) and standard blood pressure (SBP) were based on the statistically significant regression coefficients (p<0.05), no regression coefficients used in the plasma total cholesterol (PTC) estimates were statistically significant (p>0.05).

DISCUSSION

Considering BMI, it should be noted that the effects of different meat type intakes were almost the same for their IQRs with all cases included and for their IQRs after ln transformation of the remaining cases after excluding zero meat intake cases. The mentioned effects were greatest for total meat intake with 1.03 rise in kg/m^2 of the BMI and 1.03 rise in kg/m^2 for the ln transformed values. They were closely followed by the combined red and processed meat intakes with 1.03 and 1.03 rise in kg/m^2 of the BMI, respectively, but one should bear in mind that the combined red and processed meat intakes were entirely included in the total meat intakes, of which they formed the greatest part, which explains the observed similarity in their effects. The red and processed meat intakes analyzed as separated categories had almost the same effects of around 1.02 or 1.02 and around 1.02 or 1.01 rises in kg/m^2 of the BMI, respectively, which could be interpreted as their influence just *per se*, regardless of the methods of their preparation and preservation. By looking at all meat intake effects together, there was no adding or multiplication of the effects between the red and processed meat intakes. As sex of respondents exerted no significant effect and subject age was included in the preferred model in order to address its potential confounding, the observed rise in BMI values by approximately 1 kg/m^2 *per* IQR of meat intakes could be ascribed to the effects of the mentioned meat intakes. Although the mentioned effects were not striking by their values, they all were statistically highly significant. The only nonsignificant result (p=0.125) was the one for the ln processed meat intake effects, which was also the only result that was slightly lower (1.01) than the others.

Meat intakes had greatest effects on SBP, with approximately 1.5 mm Hg rise *per* IQR of different meat type intakes. They were slightly bigger for IQRs of the combined red and processed meat intakes with around 1.7 and 1.5 mm Hg than for IQRs of the total meat intakes with around 1.7 and 1.4 mm Hg, respectively. One possible explanation for this discrepancy that a part had bigger effect than the whole could be the potential protective effect of white meat intakes, which were not analyzed separately, but were forming a part of the total meat intakes together with the other two meat types. One cannot continue without commenting that the IQRs of the red meat intakes had bigger effects of around 1.5 and 1.3 mm Hg than the IQRs of the processed meat intakes of around 1.02 and 0.37 mm Hg, respectively. It could be interpreted as a good evidence of the adverse effects of meat intakes on SBP just themselves as such and regardless of the contents of salt present in them. On this track, it should be noted that both age and sex of the respondents as potential confounders were included in the preferred models for SBP, which could be used as yet another evidence for the above statement. In this respect, it should be repeated that all the observed effects were statistically highly significant. The only nonsignificant result (p=0.548) was again the one for the ln processed meat intakes, which was now strikingly smaller (0.37) than the other results.

The observed influence of the red meat intake on SBP is in accordance with the cross-sectional epidemiological study conducted by Tzoulaki *et al*., which included 4680 adults aged 40-59 from 17 population samples from Japan, China, the United Kingdom, and the United States participating in the international collaborative study on macro-/micronutrients and blood pressure (INTERMAP). Their multiple linear regression analyses showed that 102.6 g/24 h (2 SD) higher red meat intake was associated with 1.25 mm Hg higher SBP (41). On this track, one should mention a prospective study conducted by Pan *et al*. on 37 698 men from the Health Professionals Follow-up Study (1986-2008) and 83 644 women from the Nurses' Health Study (1980-2008), which proved that red meat consumption was associated with an increased risk of total, CVDs, and cancer mortality, while substitution of other healthy protein sources for red meat was associated with a lower mortality risk (42). A possible explanation for the mentioned connection was suggested by a cross-sectional study conducted by Azadbakht *et al*., which included 482 Tehrani female teachers aged 40-60 (43). Their statistical analyses showed that the increased red meat consumption was cross-sectionally associated with a greater risk of metabolic syndrome and inflammation.

Although dyslipidemias are recognized as a risk factor for CVD development and the guidelines for their prevention and treatment propose reduction of red and processed meat intakes (44-46), our linear regression models revealed no association between the mentioned meat intakes and PTC level rise. Half of them even had negative signs, thus suggesting a possible protective influence of the mentioned meat intakes against PTC level rise. However, it should be highlighted that none of the results was statistically significant, except for the one for the ln processed meat intakes (p=0.027), which in this case was much bigger (-0.12) than the other results, and thus different as usual.

One should also take into account the robustness of the cardiovascular risk factors evaluated. BMI was most robust due to its calculation from weight and height, which were easily obtained from the study participants. SBP was intermediate due to its frailty towards regression dilution bias, which was addressed by performing two measurements on a single occasion and calculating their mean value. PTC was the least robust factor due to its estimation from the casual blood sample, which relied on the study participants' willingness to follow dietary instructions. Just described relevant differences in the robustness of the cardiovascular risk factors analyzed could also be used as a possible explanation for the differences in the size and significance of the effects of different meat type intakes on the mentioned cardiovascular risk factors.

It should be stated that the above results are in accordance with earlier studies. The results showing the meat intakes to increase SBP are in accordance with a prospective study conducted by Sacks *et al*., which established a relation between the increased SBP levels and the intakes of food from animal sources (32); then, the CARDIA study conducted by Steffen *et al*., which proved that the risk of increased SBP was positively associated with the red and processed meat intakes (33); and finally The Chicago Western Electric Study conducted by Miura *et al*., which showed that men with higher intakes of red and white meat had a significantly greater SBP increase in comparison with those with higher intakes of vegetables, fruits and fish (34). The finding that the red meat intakes have greater effects on SBP rise than the processed meat intakes, despite their opposite salt contents, is supported by the DASH trial constructed by Appel, which proved that meat intakes increased SBP independently of their salt contents (35). The results showing that the combined red and processed meat intakes increased SBP more than the total meat intakes, of which they formed a greater part, were interpreted through the possible protective effect of the white meat intakes as its smaller component, and are in accordance with the case-control study conducted by Kontogianni *et al*., where the increased red meat intakes showed a strong positive association with CVD risk, whereas white meat intakes showed less prominent results (36). The findings of the adverse effects of the red and processed meat intakes on BMI and SBP are in accordance with the prospective cohort Diet and Health Study conducted by Sinha *et al*., which revealed the connection between the CVD risk and the red and processed meat intakes (37).

Although a meta-analysis of the relevant cohort and case-control studies conducted by Micha *et al*. (38) managed to find an association between the processed meat intakes and CHD risk, it failed to find it for the red meat intakes and CHD risk, and in this respect our study made a step further by revealing the connection between both meat types mentioned and the increase in BMI and SBP. The findings that the IQRs of different meat type intakes increased BMI by around 1 kg/

m^2 and SBP by around 1.5 mm Hg, although low, cannot be perceived as trivial effects at the population level. Thus, linear regression proved the existence of the mentioned effects on the individual level as well.

ACKNOWLEDGMENTS

First author would like to thank Professor John Powles, Doctor Louis Aston and Doctor Rajna Golubić from the Department of Public Health and Primary Care, University of Cambridge, for their guidance and help.

REFERENCES

1. Willet W. Foods and nutrients. Oxford: Oxford University Press, 1998.

2. Sacks FM, Campos H. Dietary therapy in hypertension. N Engl J Med 2010; 362: 2102-12.

3. Gordon MS. Ecological physiology. Science 1988; 242: 16.

4. McGill HC Jr. Introduction to the geographic pathology of atherosclerosis. Lab Invest 1968; 18: 465-7.

5. McGill HC Jr, Strong JP. The geographic pathology of atherosclerosis. Ann N Y Acad Sci 1968; 149: 923-7.

6. Keys A. Alpha lipoprotein (HDL) cholesterol in the serum and the risk of coronary heart disease and death. Lancet 1980; 2: 603-6.

7. Keys A. W. O. Atwater memorial lecture: overweight, obesity, coronary heart disease and mortality. Nutr Rev 1980; 38: 297-307.

8. Keys A. Wine, garlic, and CHD in seven countries. Lancet 1980; 1: 145-6.

9. Keys A. Coronary heart disease, serum cholesterol, and the diet. Acta Med Scand 1980; 207: 153-60.

10. Tillotson JL, Kato H, Nichaman MZ *et al*. Epidemiology of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: methodology for comparison of diet. Am J Clin Nutr 1973; 26: 177-84.

11. Robertson TL, Kato H, Rhoads GG *et al*. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California. Incidence of myocardial infarction and death from coronary heart disease. Am J Cardiol 1977; 39: 239-43.

12. Robertson TL, Kato H, Gordon T *et al*. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California. Coronary heart disease risk factors in Japan and Hawaii. Am J Cardiol 1977; 39: 244-9.

13. Benfari RC, Sherwin R. The multiple risk factor intervention trial after 4 years: a summing-up. Prev Med 1981; 1: 544-6.

14. Multiple Risk Factor Intervention Trial. Risk factor changes and mortality results. Multiple Risk Factor Intervention Trial Research Group. JAMA 1982; 248: 1465-77.

15. Michel D. Multiple Risk Factor Intervention Trial. Fortschr Med 1983; 101: 3-4.

16. The Multiple Risk Factor Intervention Trial. JAMA 1983; 249: 1435-7.

17. Mishkel MA. Multiple Risk Factor Intervention Trial. Can Med Assoc J 1983; 128: 1140-1.

18. Grimm RH Jr. The Multiple Risk Factor Intervention Trial in the U.S. A summary of results at four years in special intervention and usual care men. Prev Med 1983; 12: 185-90.

19. Gramenzi A, Gentile A, Fasoli M, Negri E, Parazzini F, La Vecchia C. Association between certain foods and risk of acute myocardial infarction in women. BMJ 1990; 300: 771-3.

20. Tzonou A, Kalandidi A, Trichopoulou A *et al*. Diet and coronary heart disease: a case-control study in Athens, Greece. Epidemiology 1993; 4: 511-6.

21. Elwood PC, Yarnell JW, Pickering J, Fehily AM, O'Brien JR. Exercise, fibrinogen, and other risk factors for ischemic heart disease. Caerphilly Prospective Heart Disease Study. Br Heart J 1993; 69: 183-7.

22. Fraser GE, Strahan TM, Sabaté J, Beeson WL, Kissinger D. Effects of traditional coronary risk factors on rates of incident coronary events in a low-risk population. The Adventist Health Study. Circulation 1993; 86: 406-13.

23. Goldbourt U, Yaari S, Medalie JH. Factors predictive of long-term coronary heart disease mortality among 10,059 male Israeli civil servants and municipal employees. A 23-year mortality follow-up in the Israeli Ischemic Heart Disease Study. Cardiology 1993; 82: 100-21.

24. Goldbourt U. Coronary heart disease prevention, nutrition, physical exercise and genetics: rationale for aiming at the identification of subgroups at differing genetic risks. World Rev Nutr Diet 1993; 72: 23-37.

25. Lapidus L, Bengtsson C. Socioeconomic factors and physical activity in relation to cardiovascular disease and death. A 12-year follow up of participants in a population study of women in Gothenburg, Sweden. Br Heart J 1986; 55: 295-301.

26. Lewington S, Whitlock G, Clarke R *et al*. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths – Prospective Studies Collaboration. Lancet 2007; 370: 1829-39.

27. Clarke R, Emberson JR, Parish S *et al*. Cholesterol fractions and apolipoproteins as risk factors for heart disease mortality in older men. Arch Intern Med 2007; 167: 1373-8.

28. Clarke R, Emberson JR, Breeze E *et al*. Biomarkers of inflammation predict both vascular and non-vascular mortality in older men. Eur Heart J 2008; 29: 800-9.

29. Clarke R, Emberson J, Fletcher A, Breeze E, Marmot M, Shipley MJ. Life expectancy in relation to cardiovascular risk factors: 38-year follow-up of 19,000 men in the Whitehall study. BMJ 2009; 339: 3513.

30. Kimuw T, Ota M. Epidemiologic study of hypertension: comparative results of hypertensive surveys in two areas in northern Japan. Am J Clin Nutr 1965; 17: 381-90.

31. Sacks FM, Rosner B, Kass EH. Blood pressure in vegetarians. Am J Epidemiol 1974; 100: 390-8.

32. Sacks FM, Kass EH. Low blood pressure in vegetarians: effects of specific foods and nutrients. Am J Clin Nutr 1988; 48: 795-800.

33. Steffen LM, Kroenke CH, Yu X *et al*. 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. Am J Clin Nutr 2005; 82: 1169-77.

34. Miura K, Greenland P, Stamler J, Liu K, Daviglus ML, Nakagawa H. Relation of vegetable, fruit, and meat intake to 7-year blood pressure change in middle-aged men: the Chicago western electric study. Am J Epidemiol 2005; 159: 572-80.

35. Appel LJ, Moore TJ, Obarzanek E *et al*. A clinical trial of the effects of dietary patterns on blood pressure. N Engl J Med 1997; 336: 1117-24.

36. Kontogianni MD, Panagiotakos DB, Pitsavos C, Chrysohoou C, Stefanadis C. Relationship between meat intake and the development of acute coronary syndromes: the CARDIO2000 case-control study. Eur J Clin Nutr 2008; 62: 171-7.

37. Sinha R, Cross AJ, Graubard BI, Leitzmann MF, Schatzkin A. Meat intake and mortality: a prospective study of over half a million people. Arch Intern Med 2009; 169: 562-71.

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

39. Hoare J, Henderson L, Bates CJ *et al*. The national diet and nutrition survey: adults aged 19 to 64 years – Appendix G – Food types, main and subsidiary food groups. Norwich: Her Majesty's Stationery Office, 2004. Available at: http://food.gov.uk/multimedia/pdfs/ndnsprintedreport.pdf; Accessed on: September 11, 2013.

40. Aston LM, Smith J, Powles JW. Impact of a reduced red and processed meat dietary pattern on disease risks and greenhouse gas emissions in the UK: a modeling study. BMJ 2012; 2: 1-9.

41. Tzoulaki I, Brown IJ, Chan Q *et al*. Relation of iron and red meat intake to blood pressure: cross sectional epidemiological study. BMJ 2008; 337: 258.

42. Pan A, Sun Q, Bernstein AM *et al*. Red meat consumption and mortality: results from 2 prospective cohort studies. Arch Intern Med 2012; 172: 555-63.

43. Azadbakht L, Esmaillzadeh A. Red meat intake is associated with metabolic syndrome and the plasma C-reactive protein concentration in women. J Nutr 2009; 139: 335-9.

44. Chapman MJ, Ginsberg HN, Amarenco P *et al*. Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. Eur Heart J 2011; 32: 1345-61.

45. Reiner Z, Catapano AL, De Backer G *et al*. ESC/EAS Guidelines for the management of dyslipidaemias: the Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). Eur Heart J 2011; 32: 1769-818.

46. Perk J, De Backer G, Gohlke H *et al*. European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). Eur Heart J 2012; 33: 1635-701.

S A Ž E T A K

CRVENO I OBRAĐENO MESO KAO ČIMBENICI RIZIKA SRČANO-KRVOŽILNIH BOLESTI

B. ATALIĆ, J. TOTH[1], V. ATALIĆ[2], D. RADANOVIĆ[3], M. MIŠKULIN i A. LUČIN[4]

Zavod za javno zdravstvo i primarnu zaštitu, Sveučilište u Cambridgeu, Cambridge, Velika Britanija, [1]Zavod za hitnu medicinu, Zagreb, [2]Zavod za javno zdravstvo Osječko-baranjske županije, Osijek, [3]Odjel za internu medicinu, Opća bolnica Vukovar i [4]Dom zdravlja Zagreb Istok, Zagreb, Hrvatska

Cilj: Utvrđivanje mogućih utjecaja konzumacije crvenoga i prerađenoga mesa na srčano-krvožilne rizične čimbenike na temelju podataka sadržanih u *British National Diet and Nutrition 2000/1 Survey* podatkovnome setu za 1724 ispitanika (766 muškaraca i 958 žena). *Metode:* Provedena je statistička linearna regresija povezanosti između konzumacije crvenoga, prerađenoga, kombinacije crvenoga i prerađenoga, te ukupnoga mesa s jedne strane i indeksa tjelesne mase (BMI), sistoličkoga krvnog tlaka (SBP), te ukupnoga plazmatskog kolesterola (PTC) kao srčano-krvožilnih rizičnih čimbenika s druge strane, uz obraćanje pozornosti na dob i spol ispitanika kao moguće utjecajne čimbenike. *Rezultati:* Linearna analiza je pokazala da konzumacija ukupnoga mesa, te kombinacije crvenoga i prerađenoga mesa uzrokuju porast BMI za oko 1,03 kg/m^2, dok odvojena konzumacija crvenoga i prerađenoga mesa uzrokuje porast za oko 1,02 kg/m^2. Najveći utjecaj je opažen kod porasta SBP koji je iznosio 1,7 mm Hg kod konzumacije ukupnoga mesa i kombinacije crvenoga i prerađenoga mesa, 1,5 mm Hg kod konzumacije crvenoga mesa, te 1,02 mm Hg kod konzumacije prerađenoga mesa. Između konzumacije različitih vrsta mesa i razine PTC nije utvrđena nikakva povezanost. *Rasprava i zaključak:* Dobiveni rezultati pokazuju da interkvartalni rasponi (IQR) konzumacije navedenih vrsta mesa povisuju BMI za oko 1 kg/m^2, a SBP za oko 1,5 mm Hg, dok na PTC nemaju nikakav utjecaj.

Ključne riječi: srčano-krvožilne bolesti; srčano-krvožilni rizični čimbenici; indeks tjelesne mase; sistolički krvni tlak; ukupni plazmatski kolesterol