International Journal of Clinical Nutrition & Dietetics

Research Article Open Access

Fast Estimation Method of Dietary Exposure to Carcinogenic Compounds in Cooked Meat for Individual Consumer Use

Wojciech Kolanowski[1]* and Joanna Trafialek[2]

[1]*Department of Dietetics and Food Assessment, Siedlce University of Natural Sciences and Humanities, Prusa str. 14, 08-110 Siedlce, Poland*
[2]*Department of Food Hygiene and Food Quality Management, Faculty of Human Nutrition and Consumer Sciences, Warsaw University of Life Sciences, Nowoursynowska str. 166, 02-787 Warsaw, Poland*

Abstract

High consumption of meat was shown to be positively associated with increased risk of some types of cancer. Meat could be involved in carcinogenesis via carcinogenic substances related to cooking and processing, e.g. nitrate, heterocyclic amines, polycyclic aromatic hydrocarbons. The evaluation of exposure to carcinogens from the diet is estimated using food frequency questionnaire and instrumental analysis. However, there is a lack of methods suitable to use by individual consumer enabling individual dietary hazards assessment. The purpose of this paper was to introduce the fast estimation method of dietary exposure risk to cooked meat-related carcinogenic substances, which could be easy to use by individual consumer. The method was based on special, formula which includes factors related to meat processing and cooking involved in carcinogens formation. It allows to calculate the hazard category and shows cooking procedures which lower the risk. The method may be helpful for every family to reduce intake of meat-related carcinogenic compounds, thus decreasing the risk of cancer.

Publication History:

Received: August 28, 2014
Accepted: November 24, 2015
Published: November 26, 2015

Keywords:

Carcinogens, Cooking methods, Heterocyclic amines, Meat, Polycyclic aromatic hydrocarbons

Introduction

Meat, especially red, is an important component of so called Western style diet [1]. In developed Western countries meat is consumed everyday. Average meat consumption is ca. 100 kg per person per year (in Europe ca. 80 kg, in USA ca. 125 kg). In contrast, average meat consumption in developing countries is ca. 30 kg per person per year or less [2]. Diet contains high meat level shows many nutritional benefits. Meat is very good source of protein, iron, zinc, B-vitamins and vitamin A. The bioavailability of iron and folate from meat is higher than from plant foods such as grains and vegetables. The weakness, however, is the high content of cholesterol and saturated fatty acids, and homocysteine which is formed during meat protein metabolism. These compounds are positively associated with plasma low density lipoprotein (LDL) concentrations and the risk of coronary heart disease [3,4]. Although iron is essential for prevention of anemia, a too high intake, especially of heme iron, is related to the endogenous formation of N-nitroso substances in the gastro-intestinal tract thus may be a risk factor for some types of cancer e.g. colon cancer [5-7]. Meat and meat-based products are almost always cooked before being eaten. The cooking process not only destroys pathogenic or spoilage microorganisms but also enable digestion and develops food taste, color, flavor and texture specific to the cooked product. However, despite high nutritional value, cooked meat may be also a source of carcinogenic substances related to cooking procedure and processing. The most important carcinogenic substances occurring in cooked meat are: nitrate, heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs).

Meat is usually divided into red meat (beef, pork, mutton/lamb, horse, goat), processed meat (all meat products, including ham, bacon, sausages, hot dogs, salami) and white meat (poultry, including chicken, hen, turkey, duck, goose, unclassified poultry, and rabbit (domestic). Red meat is usually cooked in higher temperature and longer than more delicate white meat. Moreover red meat contain higher amount of heme iron as well as carnitine and creatine which are the main source of HCAs formation. High consumption of meat, particularly red, processed and strongly heated has been related to an increased risk of some types of common cancers. Many epidemiological studies showed the link between meat consumption and colorectal, pancreatic, bladder, breast, and prostate cancers [8,9]. Meat could be involved in carcinogenesis via multiple carcinogenic compounds related to cooking and processing [10].

Due to very high risk of cancer in the Western societies there is a need to decrease the exposure to carcinogenic substances also these from the diet, especially form meat. Lot of consumers prefer well done strongly heated meat and meat products like charcoal-grilled, fried or roasted at home, outdoor or in restaurants [11]. Such food is characterized by highly evaluated sensory properties, mainly taste and flavor and dark, flavorful crust formed during cooking [12,13]. However, most consumers do not realize the risk of such cooking procedures which can result in elevated formation of substances dangerous to human health [14]. The individual evaluation of exposure to carcinogenic substances from the diet may be based on food frequency questionnaire and instrumental analysis. Such evaluations can be performed by dietitians who consider individual person food intake data and formulate recommendations related to the whole diet. However, there is a lack of methods which could be easy to use by individual consumer enabling diet-related hazards assessment. The purpose of this paper was therefore to introduce the fast estimation method of dietary exposure to cooked meat-related carcinogenic substances, which could be easy to make by individual consumer. The method allows avoiding or diminishing potentially severe diet-related health hazards.

Meat-related carcinogenic substances

Carcinogenic substances are produced in a wide variety of cooked or processed meat and fish [15]. Substances found in cooked meat with the highest carcinogenic activity are: N-nitroso substances, heterocyclic aromatic amines (HACs) and polycyclic aromatic hydrocarbons (PHAs).

***Corresponding Author:** Dr. Wojciech Kolanowski, Department of Dietetics and Food Assessment, Siedlce University of Natural Sciences and Humanities, Prusa str. 14, 08-110 Siedlce, Poland; E-mail: wojciech.kolanowski@uph.edu.pl

Citation: Kolanowski W, Trafialek J (2015) Fast Estimation Method of Dietary Exposure to Carcinogenic Compounds in Cooked Meat for Individual Consumer Use. Int J Clin Nutr Diet 1: 102. doi: http://dx.doi.org/10.15344/ijcnd/2015/102

The N-nitroso substances fall into the category of dietary carcinogens and are known to be a risk factor for colon cancer. Ingestion of nitrate and nitrite can result in the endogenous formation of N-nitroso compounds. The main dietary sources of these substances are cured meat, pickled fish and vegetables. N-nitroso substances (N-nitrosodimethylamine) may be present in meat. They are used as component of curing salts which preserve the pleasant pink color of red meat [16]. Also a high intake of heme iron with meat is related to the endogenous formation of N-nitroso substances [9]. Iron can cause oxidative stress and DNA damage, and heme iron can catalyze endogenous formation of N-nitroso substances, which are potent carcinogens. The mechanism is not known, but heme iron has a catalytic effect on the endogenous formation of carcinogenic N-nitroso substances and the formation of cytotoxic and genotoxic aldehydes by lipoperoxidation. After eating meat, the large intestine is rich in nitrogenous residues which undergo endogenous N-nitrosation increasing the risk of carcinogens exposure [5,17]. It was shown that N-nitroso compounds may promote gastric, bladder and oesophageal cancers [10,18].

HCAs are a group of procancerogenic substances identified in meat, cooked to the well done stage, in pan drippings and in meat surface that shows a crispy brown crust. The higher is the cooking temperature the higher level of HCAs in meat. HCAs undergo metabolic activation by N-hydroxylation of the exocyclic amine group, to produce a common proposed intermediate, the arylnitrenium ion, which is the critical metabolite implicated in toxicity and DNA damage [19]. HCAs are formed in high-protein content food during heat-treatment in temperatures over 160°C. The higher temperature of meat treatment the more HCAs are formed [20]. Precursors of HCAs are free amino acids found in muscle tissue mainly creatine, creatinine as a result of Maillard reactions in conditions typically applied for preparation of meat dishes [15]. HCAs are formed when creatine, other amino acids and glucose are heated together at high temperatures range from 125 to 300°C (275-572°F) or cooked for long periods of time. HCAs form at lower end of this range when the cooking time is long, at the higher end of the range HCAs are formed within minutes [21]. The less creatine and creatinine content in meat, the fewer HCAs are formed [15]. Meat acidity (pH) also plays an important role during Maillard reactions which occur depending on the environment acidity. Hence the formation of HCAs depends also on meat acidity [20].

The highest level of HCAs is usually determined in meat juice exuded from meat during cooking. The juice is rich in free amino acids and has contact with high temperature of cooking which result in elevated HCAs formation. Due to rich, pleasant flavor, the meat juice together with meat fat, which also exudes from meat during cooking, are components of gravy commonly used as dressing for meat dishes? More than 20 HCAs have been identified. HCAs produce strong carcinogenic and mutagenic action [22]. Amounts of HCAs found in commercially processed foods or prepared in restaurant range from 0.1 to 14μg/kg. Laboratory fried samples may have much high levels of HCAs, over 300 μg/kg, such level was measured in grilled chicken breast [23].

The most carcinogenic of the HCAs is MeIQ (2-Amino-3,4-dimethylimidazo(4,5-*f*-quinoline) [24]. Dietary exposure to HCAs contributes to the development of cancer by causing mutations in genes, causing new cells to grow in an uncontrolled manner and form a tumor. Epidemiological studies have linked consumption of well-done meat with an increased risk of colon or rectum cancers [25].

PAHs are formed by the incomplete combustion of organic matter. PAHs are present in the atmosphere, water, sediments, tobacco smoke, and food. In food PAHs are generated during meat smoking and grilling. About 660 different compounds belong to the PAHs group 7 of which show carcinogenic, mutagenic and teratogenic properties [26]. As a desired consequence of smoking, phenolic substances are generated, which are importance for the sensory properties of smoked meat products and antioxidative properties. In some countries high amount of smoked meat is consumed, e.g. in Germany about 60% of meat products are smoked [27].

The most well known carcinogenic PAHs is benzo(a)pyrene. Processing of food (such as drying and smoking) and cooking of foods at high temperatures (grilling, roasting, frying) are major sources generating PAHs [25]. Levels as high as 200 μg/kg food have been found for individual PAHs in smoked fish and meat. In developing countries where smoking is carried out in traditional way at very high temperature the mean total PAHs in the smoked sardines ranged from 510 μg/kg to 1460 μg/kg [28]. In barbecued meat, 130 μg/kg has been reported, whereas in uncooked foods the values range of 0.01-1 μg/kg [29]. There are several mechanisms of PAHs formation such as melted fat that undergoes pyrolysis when dripping onto the heat and pyrolysis of the meat due to the high temperature. PAHs amounts found in commercially processed or restaurant foods reach up to 1 μg/kg in a liquid smoke flavoring. Laboratory fried samples have greater amounts of PAHs, up to 38 μg/kg in hamburgers [23]. Charcoal grilling is an intense thermal process which can result in very high PAHs formation in meat. However, if dripping of melted fat onto the heat source is prevented, less PAHs will be formed in the grilled meat [30]. Understanding the processing conditions that form PAHs can lead to using cooking procedures greatly reducing their occurrence in meat.

Meat consumption and cancer

Much of the global variation in cancer incidence has been attributed to environmental influences, including dietary preferences. Diets high in meat, particularly red or processed meat may be associated with carcinogenesis. Most of epidemiological studies confirm that colorectal cancer risk is positively associated with high consumption of red and processed meat and in a less extend with poultry or fish meat [9,31]. Moreover, meat-cooking methods such as frying and doneness level increase risk of colorectal and other cancers [11,32]. Also high level of salt usually used in grilled or processed meat may additionally increase gastric cancer risk [9].

Despite colorectal or gastric cancer risk epidemiological studies report positive associations between intake of meat cooked in high-temperature and pancreatic cancer [8]. Findings provided also support for an increased risk of bladder cancer with total dietary nitrite and nitrate plus nitrite from processed meat [10]. Steck et al. showed a dose-response relationship between doneness levels of meat consumed and breast cancer risk [33]. Women who consumed hamburger, beef steak, and bacon meats consistently very well done had a 4.62 times higher risk than that of women who consumed the meat rare or medium done. Risk of breast cancer was also elevated with increasing intake of well-done to very well-done meat. Thus consumption of well-done meats and exposures to heterocyclic amines and other compounds formed during high-temperature cooking may play an important role also in the risk of breast cancer.

World Cancer Research Fund and American Institute for Cancer Research report based on an extensive review of the existing evidence

concluded that research supporting the association between red and processed meat intake and colon cancer risk was convincing [34]. The risk of colon cancer was estimated to increase by 29% for every 100 g/d increase in red meat consumption and by 21% for every 50 g/d increase in processed meat.

Hazard factors of carcinogens presence in cooked meat

There are many hazard factors which influence the carcinogenic substances level in cooked meat. Some of them may be estimated by each consumer. The most important hazard factors are meat type, cooking temperature (external), cooking method, turning over during cooking, dark, flavorful crust formation, meat doneness, seasonings and marinating, gravy consumption, serving size or vegetables and fruits intake with meat.

Meat is usually divided on red meat, processed and white. Red meats are usually cooked in higher temperature and longer than more delicate white meats which may results in higher HCAs concentration. Additionally due to higher level of heme iron also the risk of exposure to N-nitroso compounds is higher, especially when processed meat is cooked and consumed (e.g. hamburgers, sausages). So meat type influence the potetntial level of health hazardous compounds [9].

High temperature of cooking is strongly associated with formation of carcinogenic substances. It was shown that temperature increase by 400C (from 1600C to 2000C), as well as lengthening the frying time can cause even fourfold increase in HCAs content [21]. The most hazardous is cooking in direct open flame contact with meat, e.g. traditional charcoal-grilling or roasting. In Western societies charcoal-grilling is preferably chosen by consumers during summer time picnics and open air parties, which results in higher dietary exposure risk to HCAs and PHAs. In many developing countries meat cooking in contact with open flame to is a traditional cooking technique. Thus consumer dietary habits and preferences also strongly influence the exposure risk to meat-related carcinogenic substances.

Cooking methods using high or very high temperatures like grilling/barbecuing, pan frying, broiling, traditional roasting increase HCAs formation. When cooking in direct meat contact with open flame the cooking temperature is high and fat burning results in PHAs formation which penetrate to meat with smoke. Boiling, oven roasting in bags, as well as deep-frying cause the formation of a smaller amount of HCAs [20].

Long-time grilling, broiling or frying of a portion of meat on one side, without frequent turning over causes a longer contact of the meat with the heat source and contributes to an increase in amine level in the more browned outer part. It was demonstrated that frequent turning over of meat during the frying process can be an important factor lowering the content of HCAs [35]. It was shown that when meat was turned over just once at 5 min there was a great effect of pan temperature on the formation of the HCAs. If the meat was turned every minute during cooking, much less of the HCAs was detected. So cooking at a lower surface temperature (160-180°C) and turning the meat over every minute greatly reduces the formation of HCAs when meat frying [36].

Dark flavorful crust is formed when cooking in high temperature. The highest temperature the crust if more dark, crispy and flavorful until it starts to burn. Color and flavor of the crust is an effect of Maillard reactions of free amino acids with sugars which also result in HCAs formation. The darker crust the more HCAs is formed. Moreover, well done or very well done meat consumption is positively correlated with cancer due to high HCAs formation during cooking in high temperature. Meat doneness level depends on cooking temperature and time. Very well done outer meat surface can contain even 20 - 300 times higher level of HCAs than in the case of microwave cooking. This is why it is recommended to remove the burnt fragments of meat [37]. Dietary exposure risk to harmful compounds influence also serving size of cooked meat. The highest serving size the longer time of cooking is needed. Long time of cooking in high temperature results in elevated risk of carcinogenic substances formation. Unprocessed meat serving sizes is ca. 85 g, or a portion which is about the size of a deck of playing cards or bar of soap. Processed red meat serving size is usually lower, e.g. bacon - 2 slices (13g), hot dogs - one (45g) [4].

Many consumers enjoy meat consuming with gravy, which consist of meat juice and fat run naturally from meat during cooking. Meat juice contains lot of free amino acids which are easily transforming to HCAs in high temperature of cooking. The higher temperature the more dark meat juice and the highest HCAs level. However, spices used in meat can effectively reduce HCAs formation. The most effective are rosemary, sage and garlic [38]. Onion added to minced meat, e.g. hamburger, was found to be a beneficial way to reduce mutagenicity. Antioxidant substances present in species and virgin olive oil e.g. phenolic compounds also reduce formation of HCAs during cooking [15]. Also, marinating of meat before cooking with acid, oily marinades containing virgin olive oil, lemon juice and natural spices like onion, garlic, rosemary can reduce the formation of some carcinogen compounds such HCA and PAH during the cooking process [30,39]. Nevertheless, some authors suggest that flavor enhancing monosodium glutamate (MSG) which occurs in commercially available meat seasoning blends may increase the content HCAs during cooking in high temperature [40].

Vegetables and fruit consumption together with meat may diminish the dietary exposure to meat-related carcinogenic substances due to fiber and natural antioxidants content. An inverse association between fruit and vegetables and colon cancer risk was observed among individuals with elevated intakes of red and processed meat [41,42]. Fiber, in the form of vegetables, bran or resistant starch, does not reduce the level of N-nitroso substances, HCAs and PHAs level, although carcinogens contact time to intestinal mucosa is reduced by acceleration of peristaltic [5].

There are also other hazard factors of meat-related carcinogenic substances formation during cooking like creatine and creatinine content, glucose content and meat pH. However, these factors can not be estimated by the consumer.

Estimation of dietary exposure to meat-related carcinogens

Many epidemiologic studies showed that a diet with high level of meat and caloric intake increases risk of some cancers [43]. Exposure to carcinogenic substances present in the diet depends on both food consumption patterns and the concentration of the particular substance in foods consumed. Exposures to carcinogenic substances present in food are usually evaluated by food consumption data collected using food frequency questionnaire method. This method indicates how often specific foods are consumed. Collected data are elaborated by dietitians who, based on known carcinogenic potency of some food substances, can formulate recommendations related to the one's or whole population diet. Rodrigues-Hernandez et al. evaluated carcinogens intake by chemical analysis of typically consumed meat

products and elaboration of sophisticated formula [44]. However, there is a lack of methods which could be easy to use by individual consumer enabling diet-related hazards assessment.

To estimate individual's probability of meat-related carcinogenic substances intake a special formula called Meat Carcinogenicity Index (MCI) was elaborated (see below). The formula includes hazard factors related to cooking procedures. The formula summarized evaluations of particular hazard factors of carcinogenic compounds occurrence in cooked meats which are: meat type (MT), cooking temperature (external) (CT), cooking method (CM), turning over during cooking (TC), dark, flavorful crust formation (CF), meat doneness (MD), seasonings and marinating (SM), gravy consumption (meat juice) (GC), serving size (SS), vegetables and fruits intake with meat (VF). Hazard factors and hazard factors variants are specified in Table 1. The proposed MCI formula is as follow:

MCI = MT + CT + CM + TC + CF + MD + SM + GC + SS + VF

MCI formula is intended for particular meat dish and can be calculated by each consumer. Summarized value of all hazard factors estimated grades shows the probability of carcinogenic substances intake i.e. the probable risk of dietary exposure to these substances. The proposed formula includes particular hazard factors which reflect possible level of carcinogenic substances in cooked meat. Factors included meat type, cooking procedures including cooking temperature, cooking method, turning during cooking, brown crust formation, meat doneness level, meat seasonings, meat juice (gravy) consumption, serving size and vegetables and fruits intake [45]. Calculating the probability grade for each hazard factor only one, of mentioned in Table 1, hazard factors variants can be selected. Depending on hazard factor variant chosen during estimation the following hazard grades were assigned for each hazard factor: 0 – no risk; 1 - medium risk, 2 - high risk; 3 - very high risk; 4 - extremely high.

The higher MCI value the higher dietary exposure risk to meat-related carcinogenic substances. According to the summarized values of MCI five levels of hazard categories, reflecting dietary exposure risk to meat carcinogens, were specified. These are: very low – with total value from 4 to 7; low – with value from 8 to 11; medium – with value from 12 to 15; high – with value from 16 to 20 and very high - with value from 21 to 25 (Table 2).

MCI total value	Hazard category
4-7	very low
8-11	low
12-15	medium
16-20	high
21-25	very high

Table 2: Suggested interpretation of MCI index value indicating hazard categories of dietary exposure to carcinogenic substances in particular meat dish.

It is well established that high meat consumption elevare exposure risk to meat-related carcinogens. Average meat consumption in developed Western societies is ca. 100 kg per person per year i.e. 275 g/d. Some consumers can eat more than 200 kg per year and other, very small quantities. Nevertheless, based on meat consumption data per individual consumer during specified period of time average meat consumption per day or per year may be established. Additionally we estimate average structure of consumed meat. About ½ of the totally consumed meat is bought as raw meat and cooked at home usually by pan frying, roasting or grilling. These allow estimating dietary exposure risk to meat carcinogens in relation to meat consumption which is shown in Table 3.

Hazard factor	Hazard factor variants	Assigned hazard grade
Meat type (MT)	Processed meat* (MT_1)	3
	Red meat (raw) ($MT_{2)}$	2
	White meat (raw) (MT_3)	1
Cooking temp. (external) (CT)	Very high temp. (>200°C) (CT1)	4
	High temp. (160-190°C) (CT2)	3
	Medium temp. (130-155°C) (CT_3)	1
	Low temp. (100-125°C) (CT_4)	0
Cooking method (CM)	Grilling/barbecue, pan frying, broiling, traditional roasting (CM_1)	3
	Roasting in bags (CM_2)	2
	Boiling, stewing or microwaving (CM_3)	0
Turning over during cooking** (TC)	Often – every minute (TC_1)	1
	Rarely (TC_2)	2
Dark, flavorful crust formation (CF)	Very high – partly burnt surface (CF_1)	4
	High – brown and crispy crust (CF_2)	3
	Low – light brown and soft crust (CF_3)	1
	Very low – pale and soft crust (CF_4)	0
Meat doneness (MD)	Well done (MD_1)	3
	Medium done (MD_2)	2
	Rare done (MD_3)	1
Seasonings and marinating (SM)	Natural spices or acid oily marinates (SM_1)	0
	Only salt or none (SM_2)	1
Gravy consumption (meat juice) (GC)	Yes (GC_1)	2
	No (GC_2)	0
Serving size (SS)	Large - 150-200 g (SS_1)	2
	Small - 50-100 g (SS_2)	1
Vegetables and fruits intake with meat (VF)	Yes (VF_1)	0
	No (VF_2)	1

Table 1: Hazard factors of carcinogenic compounds occurrence in cooked meat.

* meat and meat products red and white, cured with nitrate (bacon, sausages) or/and smoked

** refers to meat grilled/barbecued, pan fried or traditionally roasted.

Meat daily consumption, g	Meat servings per day*	Meat consumption per year, kg	Estimated exposure risk to meat-related carcinogens
< 50	<1	<18	Very low
50-100	1-2	19-36	
101-150	2-3	37-55	Low
151-200	3-4	56-73	
201-250	4-5	74-91	Medium
251-300	5-6	92-110	
301-350	6-7	111-127	High
351-400	7-8	128-146	
401-450	8-9	147-164	Very high
>450	>9	>165	

Table 3: Dietary exposure risk to meat carcinogens in relation to meat consumption.
* standard meat serving is 50-75 g.

Regarding the association between meat intake and cancer the World Cancer Research Fund and American Institute for Cancer Research advices to consume less than 500 g of red meat per week, i.e. less than 71 g/d or 24 kg/year, minimize intake of processed meat and avoid cooking meat at very high temperatures [34]. Regarding the cardiovascular health the American Heart Association recommend consumption of no more than 180 g of meat per day. Due to fatty acids composition and cholesterol content fish, poultry without the skin, and trimmed lean meats should be chosen [46]. However, it was shown that the cooking procedure may influence the level of carcinogenic compounds in meat, also in fish, poultry and lean meat. Nevertheless, such consumption level allows classifying the dietary exposure risk to meat-related carcinogenic substances as low. In many countries average meat consumption level is high e.g. in USA, Spain, Australia which is ca. 120 kg per capita per year [2]. Such consumption level allows classifying the dietary exposure risk to meat-related carcinogenic substances as high. However, some consumers eat much more meat which may elevate the exposure risk to very high.

Conclusion

Exposures to meat-related carcinogenic substances among individuals, depending on dietary preferences and methods of meat preparation, vary greatly. Raw meats should usually not contain high amounts of N-nitroso compounds and HCAs or PAHs which level increase during processing and cooking. Reducing the cooking temperature seems to be the most practical way to reduce formation of HCAs, but avoiding the conditions where the temperatures are below those needed to kill harmful bacteria is also important. The formation of PAHs can be reduced by not exposing the food directly to the heat source and resulting smoke when grilling foods. The cooking process is responsible for the formation of HCAs and PAHs from natural constituents in foods, with cooking time and temperature being important determinants in both the qualitative and the quantitative formation of these compounds. Also marinating meat with acid, oily marinades containing natural spices like onion, garlic, rosemary, virgin olive oil and lemon juice can reduce the formation of HCAs and PAHs during the cooking process. Additionally avoiding consumption of meat juice or gravy and vegetable and fruit consumption together wit meat limits dietary exposure to meat-related carcinogenic substances. Even health-conscious consumers find it hard to maintain a healthy diet. However, food quality and safety is of paramount concern to modern consumers. MCI calculation method may be a suitable tool for each consumer to estimate the dietary exposure risk to meat-related harmful compounds in the diet. It allows to calculate the hazard category and shows cooking procedures which lower the risk. This method may be helpful for every family, as well as for quality control personnel within restaurants and catering companies, to reduce dietary exposure to meat-related carcinogenic substances, thus decreasing the risk of cancer.

Competing Interests

The author declare that she has no competing interests.

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