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Review

# The World Cancer Research Fund report 2007: A challenge for the meat processing industry

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ABSTRACT

One of the 10 universal guidelines for healthy nutrition in a report of the World Cancer Research Fund released at the end of 2007 is to "limit intake of red meat and avoid processed meat", as a result of the "convincing evidence" for an association with an increased risk of colorectal cancer development. In the present paper, the scientific evidence for the association between processed meats intake and colorectal cancer development is explored and the most probable hypothesis on the mechanism underlying this relationship formulated.

It seems that the present state of knowledge is not well understood but relates to a combination of haem iron, oxidative stress, formation of *N*-nitroso compounds and related residues in the digestive tract as the causal factors. Although criticisms of the inaccurate definition of processed meats and the insufficient accounting for the large variability in composition of meat products have been expressed, it is clear that the report urges proper action by the meat and nutrition research community and the meat industry.

Research items that in our view should be addressed are discussed. They include: (1) evaluating the health risks associated with processed meats intake within the context of the supply of beneficial nutrients and other nutrition associated health risks; (2) definition of the role of nitrites and nitrates in meat processing; (3) investigating the role of red and processed meats on the endogenous formation of *N*-nitroso compounds in the digestive tract; and (4) developing improved processed meats using new ingredients.



**Contents**



## 1. Introduction

The widely recognized nutritional and gastronomic value of meat and meat products may be damaged in the future by several recent media reports relating, in particular, to processed meat consumption and the incidence of "civilization" diseases. The most prominent and recent example is the incidence of colorectal cancer, subject of media references to scientifically based evidence that recently resulted in "Limit intake of red meat and avoid processed meat" as one of the 10 universal guidelines for healthy nutrition (World Cancer Research Fund (WCRF), 2007). It is evident

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that, in addition to the fat and salt scares, such a statement represents 'a clear and present danger' for the meat industry. Perhaps the main reason for the maintenance and further development of the meat processing industry is its role in the production of pre-prepared convenience meat products, a social necessity becoming more visible every day. However, the consumption of these convenience products at a level justifying industrial activity without damaging public health requires the industry to meet that challenge rather than to ignore it.

The aim of this paper is to try and propose an evidence-based framework for action to be taken by the meat industry for that purpose, involving consecutively:

1. To recognize the current research evidence for the recommendation to avoid or reduce processed meats intake.
2. To situate the risks associated with processed meats intake within the context of its potential benefits and other health risks in order to better assess the overall impact on human health and well-being.
3. To formulate the most probable hypothesis on the mechanism(s) underlying the epidemiological relationship between processed meat intake and colorectal cancer incidence.
4. To propose a framework for research and development by the meat processing industry to solve or at least minimize the problem.

The approach will focus on processed meats rather than on red meat and is inspired by earlier work (Demeyer, 2007; Demeyer & Stahnke, 2002) as well as by reports from other EU member countries (see e.g. Palanca, Rodriguez, Senorans, & Reglero, 2006).

## 2. Recognition of the research evidence for the recommendation to avoid processed meat intake

The consumption of red meat and, in particular, processed meats, has been related to the incidence of colorectal cancer since 1975 in several epidemiological studies, mainly in the US and the UK. The problem was recognized by the international community of meat scientists, and was clearly stated at the 44th International Congress of Meat Science and Technology in Barcelona (Tarrant, 1998). A world wide recommendation for moderation in the consumption of preserved meat, such as sausages, salami, bacon and ham (meaning cured, smoked and nitrite treated meat products), was launched by the World Health Organisation in 2003 (WHO, 2003). The most recent international guidelines have now strengthened this recommendation to "avoid processed meat" (WCRF, 2007), based on a meta-analysis of cohort studies showing increased risk of colorectal cancer with increased intake of processed meats (summary estimate of relative risk 1.21 (95% CI 1.04–1.42) per 50 g/day) and supported by case-control studies. A dose–response relationship was also apparent. This is supported by a Swedish meta-analysis of 14 cohort studies reporting a summary hazard ratio estimate of 1.09 (95% CI 1.05–1.13) per 30 g/day (Larsson & Wolk, 2006). Assuming that the lifetime absolute risk of colorectal cancer is about 5% (1 in 20 persons), a relative risk of 1.2 then corresponds to a lifetime risk of 6% (1 in 18 persons). Referring to the WCRF summary estimate of relative risk, a decrease of 50 g/d in processed meats consumption may then lower the total number of colorectal cancer cases by approximately 20%. As Baghurst (2007) pointed out, a relative risk estimate of <1.3 would normally receive little attention in the epidemiological community, but 'exposure' to red meat is such a common event that even a modest association, if proven to be causal, may have considerable public health consequences.

One of the major data reports included is European work emanating from the EPIC (European Prospective Investigation into Cancer and Nutrition) project, grouping 23 research institutes and following since 1992 about 500,000 people in 10 EU member states of whom 1 in 329 were diagnosed with colorectal cancer (Norat, Lukanova, Ferrari, & Riboli, 2002). Diet was measured by country-specific questionnaires. The method was validated using biomarker determination in 24-h urine and blood samples and measurements were calibrated across countries and corrected for systematic over- and underestimation in a separate experiment. After correction for age, sex, energy from fat and non-fat sources, height, weight, physical activity, smoking status, dietary fiber and alcohol intake the relationship was clear and significant for processed meats ($p = 0.02$) and approached significance for red meat ($p = 0.08$) (Norat et al., 2005). Norat, Bingham, and Riboli (2005) also rejected criticism suggesting socio-economical confounding of their results, as well as concluding that the positive association between colorectal cancer risk and red and processed meat intake is not affected by a high intake of vegetables. They also point out however that their results suggest a possible weakening of the association between red meat intake and colorectal cancer risk by higher fiber intake. As vegetables are sources of fiber (and folate) as well as of nitrate and nitrites, it is of course difficult to rationalize their effect, as reflected also in the authors' final statement that their results "do not demonstrate that high intake of red meat accompanied by high intake of vegetables is not associated with colorectal cancer risk".

The WCRF report as well as the individual reports, published in peer reviewed journals, are subject to some criticism because the large variability in composition and nature of meat and meat products is not sufficiently taken into account, nor are processed meats defined with sufficient precision. The WCRF report itself states that "There is no generally agreed definition of processed meat. The term is used inconsistently in epidemiological studies. Judgments and recommendations are therefore less clear than they could be". Doubts may also be expressed on the precise assessment of food intake by the questionnaire. Criticisms are therefore justified, mainly in relation to the difference between the associations of colorectal cancer risk with red meat and processed meat intake respectively. The definition used and the associated data do not permit evaluation of any differences between these respective relationships nor to establish relationships for different types of processed meats separately. The WCRF definition of processed meats as "meats preserved by smoking, curing or salting or by the addition of chemical preservatives" can nevertheless be accepted as sufficiently accurate to rely on the relationships for processed meats and/or red meat as a whole. Furthermore, additional support for the EPIC results is evident from recent independent epidemiological research from the National Cancer Institute in the US, involving 500,000 people aged 50–71 years of whom 5107 developed colorectal cancer over 8.2 years (Cross et al., 2007). Meat intake was again estimated from a food frequency questionnaire and similar hazard risk ratios of 1.20 ($p < 0.001$) for colorectal cancer incidence were found at the highest intake of both red and processed meats (63 and 23 g/1000 kcal, respectively). It should be mentioned however that in this study, bacon and hot dogs were included in estimates of both red and processed meats consumption, and processed meats also included poultry sausages, illustrating the variable definition of processed meats across studies.

Although colorectal cancer incidence was not found to be related to meat or processed meat intake in a Japanese limited population based case-control study (782 cases and 793 controls)(Kimura et al., 2007), some independent support can also be found in other recent Asian work (Sun-Il et al., 2008), showing that the increase in meat consumption in Japan and Korea as calcu-

lated from data of annual supply and demand of beef and pork was related in a similar manner to the increase of colorectal cancer incidence with a 20 year difference between the two countries. Finally, it should be mentioned that mechanisms underlying the association of processed meat intake with cancer may relate to more general effects beyond effects in the intestinal tract. Indeed similar epidemiological relationships between cancer incidence and intake of processed meats were recently shown to exist for stomach (Gonzalez et al., 2006; Larsson, Bergkvist, & Wolk, 2006), kidney (Faramawi, Johnson, Fry, Sall, & Yi, 2007) and lung cancer (Cross et al., 2007).

There can be no question that the problem summarized above should be recognized in addition and perhaps related to the separate issue of processed meats contributing to salt (or sodium) intake. Fresh meat is low in sodium but processed meats contain 2% added salt, a value that may increase to 6% in dried products. Processed meats contribute for 20–30% to the daily salt (NaCl) intake in industrialized countries amounting to between 9 and 12 g/d, a much larger value than the recommended value of <5 g/d (Jiménez-Colmenero, Carballo, & Cofrades, 2001; WHO, 2003). The clear association between consumption of processed meats and the incidence of hypertension (Paik, Wendel, & Freeman, 2005) confirms the importance of meat technology in relation to salt intake. The problems discussed here require an urgent solution also because of the much greater mortality associated with the nature rather than with the contamination of foods (Krebs, 2001).

## 3. Positioning of the health risks associated with processed meats intake within the context of nutrient supply and other consumption associated health risks

In a summary of the EPIC study, Gonzalez and Riboli (2006) report that a consumption of 100 g/d of red meat or 25 g/d of processed meats results in a risk increase for colorectal cancer of between 12% and 17% and 49%, respectively. These findings are in line with the data referred to earlier (Larsson & Wolk, 2006; WCRF, 2007) and with other recent results indicating that also outside Europe and the US, increased meat consumption is associated with an increase of general mortality (see e.g. Cai et al., 2007). The levels of intake corresponding to these relative risk estimates should be positioned against a zero risk red meat intake of 70 g/week (Norat et al., 2002). In comparison, the mean consumption of red meat and meat products was estimated at approximately 55 and 35 g per person per day respectively in Flanders (De Geeter, 2007), and 60 and 93 g per person per day respectively in Austria and Germany (Bauer & Honikel, 2007).

Although the epidemiological evidence for the relationship between colorectal cancer risk (at least!) and processed meats intake cannot be denied, it is also clear that this relationship should be further investigated and evaluated, both in a qualitative and quantitative way. Research should be initiated to:

1. Contrast the levels of intake associated with an increased risk for developing cancer with the levels necessary to supply micro-nutrients such as zinc, iron and vitamins (Bauer & Honikel, 2007; Grillenberger et al., 2007; Hambidge & Krebs, 2007) and with the contribution of meat intake to the supply of functional food components such as antihypertensive peptides (Sentandreu & Toldra, 2007), carnitine (Rigault, Mazue, Bernard, Demarquoy, & Le Borgne, 2008), creatine and carnosine (Bauchart et al., 2006) as well as in the maintenance of mental health (Baines, Powers, & Brown, 2007).
2. Compare the impact of the risk associated with processed meats intake with other risk factors associated with colorectal cancer. As an example, hazard ratio's (highest vs. lowest intake or level) of similar magnitude (1.20–1.50) as for processed meats were calculated for the risk of colorectal cancer due to alcohol consumption (Ferrari et al., 2007), lack of physical activity (Friedenreich et al., 2006), a 5 point increase in body mass index (Larsson & Wolk, 2007) and for cigarette smoking (Huang et al., 2006).
3. Evaluate the magnitude of the risk involved against that of analogous risk increasing effects for other civilization diseases. Bjerregaard et al. (2006) e.g. reported that the incidence of lung and other cancers increases about twofold when smoking frequency increases from <2 cigarettes/day to between 10 and 16 cigarettes/day. He, Nowson, Lucas, and MacGregor (2007) found that increasing fruit and vegetable intake from <3 to >5 portions/day lowers cardiovascular incidence rate by 17%. Such values can be compared to an estimated 10% prevention of colorectal cancer cases by lowering the highest to the lowest level of processed meats intake studied by Cross et al. (2007).
4. Investigate the effect of genetic differences on the strength of the relationship. It is clear that the presence of well known polymorphisms of DNA repair and detoxifying enzymes (such as cytochrome P450, acetylases and sulphatases) may affect the strength of the relationship (Ahmed, 2006). Such effects were recently discussed (see e.g. Hansen et al., 2007; Huang et al., 2007; Kiss et al., 2007; Skjelbred et al., 2007) and reported for red meat (Kury et al., 2007) as well as for heterocyclic amines (HCA) (Butler et al., 2008; Shin et al., 2008) and polyaromatic hydrocarbons (PAH) (Pavanello, Pulliero, & Clonfero, 2008) associated cancers. Similarly, it was recently found that an association between breast cancer and red and processed meat consumption was limited to genetically susceptible women (Egeberg et al., 2008).
5. Further evaluate and investigate the relationship with separate types of better specified processed meats (Linseisen et al., 2006). In detailed Swedish research (Larsson et al., 2006) similar hazard risk ratio estimates of 1.55, 1.50 and 1.48 were found for bacon, hot dogs and salami, respectively.

A more accurate assessment of the overall impact of reducing the intake of meat products should allow both government and health care institutes and the meat industry to evaluate both the nature and the urgency of actions to take, i.e. whether or not to set-up information campaigns for the large public and to adapt meat processing technologies respectively.

## 4. Hypotheses on the causal mechanism(s) underlying the relationship between colorectal cancer risk and processed meats intake

The stronger association with cancer risk for processed meats than for red meat suggests that the underlying mechanism relates to the processing treatments or additives used. Apart from salt, nitrites and nitrates are often used in meat processing. In box 4.3.2 on page 118 of the WCRF report, it is suggested that dietary nitrites (from processed meats for example) and nitrates (from vegetables) are to be considered as human carcinogens because they may be converted in the body to *N*-nitroso compounds that are known carcinogens. It is highly unlikely however that these additives are the responsible agents as such. Indeed, it is well known that processed meats contain very little residual nitrates and nitrites, in amounts far less than many vegetables (Honikel, 2008). Also, both nitrate and nitrite are formed endogenously and recycle with the saliva as recognized years ago, whereas NO (formed from nitrite) is a well known metabolic regulator and dietary nitrite supplementation has been reported for protection of cardiovascular injury (Bryan et al., 2007). Finally, the epidemiological and other studies used

in the report do not show positive relations between intake of vegetables (rich in nitrates) and cancer incidence (indeed negative relations are often reported!). Because also of the lack of uniformity and precision in defining processed meats, not all processed meats recorded contained added nitrites and so far, a specific type of processed meat type that is more strongly associated with colorectal cancer risk than others could not be identified (Norat, Bingham, & Riboli, 2005).

In addition to the suggestion for an involvement of dietary nitrites and nitrates as disputed above, it is argued on page 37 of the report that a combination of haem iron, oxidative stress, formation of *N*-nitroso compounds and related residues in the digestive tract is associated with the formation of carcinogenic DNA adducts in colon cells. The formation of *N*-nitroso compounds (NOC) under influence of haem as a possible explanation was further substantiated by Kuhnle and Bingham (2007) and Kuhnle et al. (2007). Nevertheless, in animal studies haem seems to be more important in the induction of precancerous lesions than NOC (Pierre, Tache, Petit, Van der Meer, & Corpet, 2003). Lee, Kim, Kang, and Kim (2006) showed that the haem group accelerates the growth of colon cancer cells through production of reactive oxygen radicals, pointing to the involvement of oxidative stress in this complex mechanism. Further evidence for the implications of iron overload and red meat on colorectal cancer risk can be found in Huang (2003) and Lewin et al. (2006). An important report emanating from the EPIC project (Jakszyn et al., 2007) associated stomach cancer risk with estimates of both nitroso compounds formed endogenously in the digestive tract as well as with the intake of nitrosamines present in food. A significant increase of the risk was found only for nitroso compounds formed endogenously and responsible for the formation of specific DNA adducts.

Other possible causative agents referred to in the literature are HCA, PAH and nitrosamines formed during the preparation and/or processing of meats. For processed meats, these compounds are to be considered of less importance for a number of reasons. Indeed, poultry for example is an important source of HCA but its intake is not associated with cancer (Norat et al., 2005). A clear association of processed meats prepared with nitrate/nitrite with cancer incidence was recently demonstrated independent from HCA intake and in the absence of detectable amounts of nitrosamines (Ward et al., 2007). The formation of nitrosamines in meat products manufactured under GMP is small (Deierling, Hemmrich, Grosch, & Taschau, 1997; Honikel 2008; Yurchenko & Mölder, 2007). Other work showed a clearly greater risk for colorectal adenome formation with intake of processed meats than with fried meats (Martinez et al., 2007).

In conclusion, both the presence of haem iron and the endogenous formation of *N*-nitroso compounds seem to be the most likely potential factors in the contribution of processed meats to colorectal cancer.

## 5. A framework for problem solving research and development

The arguments presented above clearly indicate the urgent need for an adaptation of meat processing in relation to the health value of processed meats. It is also clear that the mechanisms underlying the association between processed meats and colorectal cancer incidence are as yet unknown. However, from the discussion the most probable hypothesis for explaining the damaging effect of processed meats at their present intake involves haem compounds, nitros(yl)ation and/or reactive oxygen radical formation, resulting ultimately in carcinogenic DNA damage. Indeed, processed meats are essentially produced by addition to fresh meat of 2–3% salt (NaCl) containing about 0.5% $NaNO_2$. For long-ripened products, nitrate rather than nitrite is used. Pending definite proof for this hypothesis, it is indicated to consider both the selection of raw materials containing less haem iron and the development of processing technologies involving the omission or lowering of nitrite and nitrate use. In addition, research is needed in our view to assess the damaging nitros(yl)ation reactions by haem and nitrite/nitrate present in the digestive tract. Subsequently, modified meat products might be developed to counteract this effect.

### *5.1. Omitting or lowering the use of nitrite and nitrate*

For nearly 30 years, the omission of nitrate/nitrite from meat processing has been proposed (Mirvish et al., 2002; Wirth, 1991) and was even effectively applied (Skjelkvåle, Tjaberg, & Valland, 1974), inspired by the long known cytotoxic effects of nitrosamines. It is now known that acceptable alternatives for the use of nitrate and nitrite exist in relation to color development, flavor and microbiological safety. There exists for example a purified red haem pigment for use in cooked meat products (Pegg, Fisch, & Shahidi, 2000). Nitrosomyoglobin can be formed in microbial metabolism in the absence of nitrate/nitrite as recently confirmed (Zhang, Kong, & Xiong, 2007). Useful and EU accepted coloring additives are available (Finkgremmels, Dresel, & Leistner, 1991) and attention should be given to Zn-porphyrin, the red substance formed in Mediterranean products such as Parma ham, in the absence of nitrite (Adamsen, Moller, Laursen, Olsen, & Skibsted, 2006; Laursen, Adamsen, Laursen, Olsen, & Moller, 2008). An adapted hurdle technology based on decreasing $a_w < 0.85$ can guarantee microbial safety, in the absence of nitrite (Chawla & Chander, 2004). Meat products of acceptable hygienic and sensory quality using vegetable juices as sources of nitrates and nitrites were recently developed in the USA (Sindelar, Cordray, Olson, Sebranek, & Love, 2007). Nevertheless, arguments for the widespread omission of nitrites/nitrates will have to be very convincing in view of the positive effects of these substances as antimicrobials, antioxidants, and desirable flavour and red colour developing ingredients.

### *5.2. Identification followed by inhibition of the damaging nitros(yl)ation reactions by haem and nitrite/nitrate present in the digestive tract and of the ensuing carcinogenic effects*

This approach is probably an important one because the past years have shown that nitrate/nitrite use in meat processing is hard to replace. A first possibility is of course a more frequent substitution of red meat by poultry raw materials. (Processed) poultry intake is not associated with colon cancer incidence and its use requires a minimal adaptation of technology. In fact, this supports the most probable hypothesis proposed earlier that nitrite alone is not the causal factor, but the combination of *N*-nitroso compounds and haem iron. The latter is present in higher amounts in red meats (beef, pork, lamb) than in white meats (poultry). There is also large variation in the haem content of red meats of different species, and between muscles within species. Genetic selection of meat producing animals for faster growth over the last decades has resulted in a more glycolytic muscle fibre type, and thus a lower muscle haem content. This may offer opportunities for lowering the haem and iron content of raw meat materials used for processing.

Inhibition of the hypothetical damaging nitros(yl)ation upon consumption of red meat or haem iron containing processed meats would involve the use of new ingredients in meat processing. This requires a distinction to be made between whole and comminuted or ground products sometimes referred to as prepared meats and meat preparations respectively. A number of new non-meat (raw) materials can indeed easily be added during the comminuting or chopping process characteristic of the latter, whereas this is

less straightforward for whole meat cuts in which addition will necessitate combining the new (raw) materials with brine (curing solution) or dry salt. Compounds or materials to be considered can be grouped as follows.

(a) Compounds introducing fibre in the product (Münch & Honikel, 2007) or with known protective effect against colorectal cancer such as pre- and probiotics (Geier, Butler, & Howarth, 2006), resistant starch (amylose enriched) (Toden, Bird, Topping, & Conlon, 2007) and omega-3 polyunsaturated fatty acids (Hall et al., 2007) can be introduced in processed meats. Spanish work has shown that the substitution of pork fat (lard) by soy or fish oil or by inulin increases the health value of fermented sausages, not only through improvement of the fatty acid composition but also by increasing the fiber content. Even for the exigent Spanish products, this was done without harming the sensory quality, provided extra antioxidants (100 mg/kg of butylhydroxytoluene and butylhydroxyanisole each) were used with fish oil (Valencia, Ansorena, & Astiasaran, 2007; Valencia, Ansorena, & Astiasarán, 2006). A more generalized ‘‘functionalization" of processed meats has been recommended in Spain (Fernandez-Lopez et al., 2005).
(b) Compounds inhibiting the nitros(yl)ation and/or oxygen radical formation. Various antioxidants such as tocopherols and extracts from herbs such as rosemary may be valuable in this respect. Also the use of ascorbate or ascorbic acid which is very common in meat products in some countries will contribute to scavenge oxygen radicals. In a more general approach, plant biomass receives considerable attention as a possible functional component in processed meats. Not only the classic herbs and ingredients such as paprika (Revilla & Quintana, 2005) might be considered here, but also materials such as tea catechins (Yilmaz, 2006), citrus byproducts such as lemon peel (Fernandez-Lopez et al., 2005) as well as grape seed and pine bark (Grün, Ahn, Clarke, & Lorenzen, 2006) preparations might be used. Furthermore, such ingredients may also increase product safety because of their antimicrobial activities while they enrich the products in functional plant minor components such as lycopene (Osterlie & Lerfall, 2005).

It is known however that the uses of (herbal) antioxidant compounds in meat processing is delicate because, dependent on the amounts used, pro-oxidant as well as antioxidant effects may be initiated (Kondaiah & Pragati, 2005; Lee & Kunz, 2005). It may therefore be more feasible to use meat raw materials derived from animals fed on diets enriched with similar materials such as polyphenols (Demeyer, Raes, Fievez, & De Smet, 2004; Lopez-Bote, 2000; Tang, Kerry, Sheehan, Buckley, & Morrissey, 2000) and to evaluate such raw materials in relation to the protection against oxidative stress and haem catalyzed nitros(yl)ation in the gut. The risk for possible adverse effects of using all kinds of functional ingredients in meats is much smaller by the animal route for several reasons. Firstly, the animal feed legislation is very strict. Secondly, the levels of functional compounds or their metabolites deposited in edible tissues following animal feeding will be much lower due to the animal's metabolism. However, the potential benefits of adapted feeding strategies for improving raw meat materials for processing need to be better assessed.

Research and development initiatives searching for such products should be considered priority projects (Palanca et al., 2006). Deciding on which approach to take is obviously affected by cost, knowledge and feasibility. The introduction of functional foods in the dairy industry may be a source of inspiration. Whatever choice is made, it will require a multidisciplinary approach, including meat producers, meat scientists, meat technologists, nutritionists and toxicologists as well as physicians. New products developed by meat producers, scientists and technologists will have to be investigated during digestion in vitro but also evaluated in vivo during consumption by animals or humans. Experimental techniques for that purpose are available, including methods for evaluation of muscle protein oxidation (Astruc, Marinova, Labas, Gatellier, & Sante-Lhoutellier, 2007) and urinary markers for evaluation of oxidative stress and DNA damage in humans (Pierre et al., 2006) as described in the development of other foods (Azadbakht et al., 2007).

### *5.3. Reducing the use of salt*

Apart from the recommendation to ‘‘limit consumption of salty foods and foods processed with salt (sodium)" (WCRF, 2007), a possible association of the processed meat–colorectal cancer relationship with the ‘‘salt problem" should not be discarded. The generally accepted fact that salt intake largely exceeds recommended values (see above) has resulted in government initiatives to lower salt intake and in government requests for the food industry to consider methods for lowering salt use. Alternatives for salt in meat processing have also been investigated for decades (Gelabert, Gou, Guerrero, & Arnau, 2003; Schmidt, Means, & Clarke, 1986; Wirth, 1986; Wirth, 1988). Even more than for nitrate/nitrite, such intervention is a delicate one, because of the universal role of salt in texture as well as in flavor development and safety of processed meats (Ruusunen & Puolanne, 2005). In the author's experience (Honikel, unpublished results); there is no technological need for salt in pâté, liver, blood sausages and aspic products whereas the reduction of salt down to between 1% and 1.5% is possible in all heat treated products. According to the same author flavor formation requires about 1% (today about 2% is used), emulsion type sausages could be produced with 1.5% salt requiring a slight increase in meat raw material use only, whereas cooked ham may be feasible with 1.7–1.8% salt. Because of the lack of the heat safety hurdle, reduction will be more difficult with non heat-treated products but some reduction is still possible. A replacement of NaCl by KCl is limited to about 0.5–0.6% salt because KCl in concentrations above this value develops a bitter and metallic taste (Shahidi & Samaranayaka, 2004). A partial replacement of salt by calcium ascorbate lowered the salt content from 2.3% to 1.4% in dry fermented sausages, with limited effect on the sensorial quality and resulting in an increased content of calcium, an important nutrient that is only present in very low amounts in meat (Gimeno, Astiasaran, & Bello, 2001). Support for such replacement may also be found in the recently reported protective effect of calcium against colorectal carcinogenesis in rats (Pierre, Santarelli, Tache, Gueraud, & Corpet, 2008). This illustrates that salt reduction may improve the nutritional as well as the health value of processed meats. Salt reduction may be difficult to accept however because of supermarket concerns about shelf life as well as processing concerns about the label of traditional products (Honikel, 2008).

## References


Adamsen, C. E., Moller, J. K. S., Laursen, K., Olsen, K., & Skibsted, L. H. (2006). Zn–porphyrin formation in cured meat products: Effect of added salt and nitrite. *Meat Science, 72*, 672–679.

Ahmed, F. E. (2006). Gene–gene, gene–environment and multiple interactions in colorectal cancer. *Journal of Environmental Science and Health Part C, 24*, 1–101.

Astruc, T., Marinova, P., Labas, R., Gatellier, P., & Sante-Lhoutellier, V. (2007). Detection and localization of oxidized proteins in muscle cells by fluorescence microscopy. *Journal of Agricultural and Food Chemistry, 55*(23), 9554–9558.

Azadbakht, L., Kimiagar, M., Mehrabi, Y., Esmaillzadeh, A., Hu, F. B., Willett, W. C., et al. (2007). Dietary soya intake alters plasma antioxidant status and lipid peroxidation in postmenopausal women with the metabolic syndrome. *British Journal of Nutrition, 98*(4), 807–813.

Baghurst, P. A. (2007). Colorectal cancer. *Nutrition & Dietetics, 64*(Suppl. 4), S173–S180.
Baines, S., Powers, J., & Brown, W. J. (2007). How does the health and well-being of young Australian vegetarian and semi-vegetarian women compare with non-vegetarians? *Public Health Nutrition, 10*(5), 436–442.
Bauchart, C., Remond, D., Chambon, C., Mirand, P. P., Savary-Auzeloux, I., Reynes, C., et al. (2006). Small peptides (<5 kDa) found in ready-to-eat beef meat. *Meat Science, 74*(4), 658–666.
Bauer, F., & Honikel, K. O. (2007). Meat – A food of high nutritional density. *Fleischwirtschaft International, 22*(5), 39–42.
Bjerregaard, B. K., Raaschou-Nielsen, O., Sorensen, M., Frederiksen, K., Tjonneland, A., Rohrmann, S., et al. (2006). The effect of occasional smoking on smoking-related cancers – In the European Prospective Investigation into Cancer and Nutrition (EPIC). *Cancer Causes & Control, 17*(10), 1305–1309.
Bryan, N. S., Calvert, J. W., Elrod, J. W., Gundewar, S., Ji, S. Y., & Lefer, D. J. (2007). Dietary nitrite supplementation protects against myocardial ischemia-reperfusion injury. *Proceedings of the National Academy of Sciences of the United States of America, 104*(48), 19144–19149.
Butler, L. M., Millikan, R. C., Sinha, R., Keku, T. O., Winkel, S., Harlan, B., et al. (2008). Modification by *N*-acetyltransferase 1 genotype on the association between dietary heterocyclic amines and colon cancer in a multiethnic study. *Mutation Research – Fundamental and Molecular Mechanisms of Mutagenesis, 638*(1-2), 162–174.
Cai, H., Shu, X. O., Gao, Y. T., Li, H. L., Yang, G., & Zheng, W. (2007). A prospective study of dietary patterns and mortality in Chinese women. *Epidemiology, 18*(3), 393–401.
Chawla, S. P., & Chander, R. (2004). Microbiological safety of shelf-stable meat products prepared by employing hurdle technology. *Food Control, 15*, 559–563.
Cross, A. J., Leitzmann, M. F., Gail, M. H., Hollenbeck, A. R., Schatzkin, A., & Sinha, R. (2007). A prospective study of red and processed meat intake in relation to cancer risk. *Plos Medicine, 4*(12), 1973–1984.
De Geeter, H. (2007). Tweede Rapport van het World Cancer Research Fund – Gezonde leefstijl helpt kanker te voorkomen. *Nutrinews, 15*(4), 18–24.
Deierling, H., Hemmrich, U., Grosch, N., & Taschau, H. (1997). Nitrosamine in Lebensmitteln. *Lebensmittelchemie, 51*, 53–55.
Demeyer, D., & Stahnke, L. (2002). Quality control of fermented meat products. In J. Kerry & D. Ledward (Eds.). *Meat processing, improving quality* (pp. 359–393). Cambridge: Woodhead Publishing.
Demeyer, D. (2007). Composition and nutrition. In Fidel Toldrá (Ed.), *Handbook of fermented meat and poultry*. *Part V. Product categories: General considerations* (pp. 243–256). Blackwell Publishing (Chapter 23).
Demeyer, D., Raes, K., Fievez, V., & De Smet, S. (2004). Radicals and antioxidants in relation to human and animal health: a case for functional feeding. *Communications in Agricultural and Applied Biological Sciences, Ghent University, 69*, 75–92.
Egeberg, R., Olsen, A., Autrup, H., Christensen, J., Stripp, C., Tetens, I., et al. (2008). Meat consumption, *N*-acetyl transferase 1 and 2 polymorphism and risk of breast cancer, in Danish postmenopausal women. *European Journal of Cancer Prevention, 17*, 39–47.
Faramawi, M. F., Johnson, E., Fry, M. W., Sall, M., & Yi, Z. (2007). Consumption of different types of meat and the risk of renal cancer: meta-analysis of case-control studies. *Cancer Causes & Control, 18*(2), 125–133.
Fernandez-Lopez, J., Fernandez-Gines, J. M., Aleson-Carbonell, L., Sendra, E., Sayas-Barberá, E., & Pérez-Alvarez, J. A. (2005). Application of functional citrus by-products to meat products. *Trends in Food Science and Technology, 15*(3–4), 176–185.
Ferrari, P., Jenab, M., Norat, T., Moskal, A., Slimani, N., Olsen, A., et al. (2007). Lifetime and baseline alcohol intake and risk of colon and rectal cancers in the European Prospective Investigation into Cancer and Nutrition (EPIC). *International Journal of Cancer, 121*(9), 2065–2072.
Finkgremmels, J., Dresel, J., & Leistner, L. (1991). Use of monascus extracts as an alternative to nitrite in meat-products. *Fleischwirtschaft, 71*(10), 1184–1186.
Friedenreich, C., Norat, T., Steindorf, K., Boutron-Ruault, M. C., Pischon, T., Mazuir, M., et al. (2006). Physical activity and risk of colon and rectal cancers: The European Prospective Investigation into Cancer and Nutrition. *Cancer Epidemiology Biomarkers & Prevention, 15*(12), 2398–2407.
Geier, M. S., Butler, R. N., & Howarth, G. S. (2006). Probiotics, prebiotics and synbiotics – A role in chemoprevention for colorectal cancer? *Cancer Biology & Therapy, 5*(10), 1265–1269.
Gelabert, J., Gou, P., Guerrero, L., & Arnau, J. (2003). Effect of sodium chloride replacement on some characteristics of fermented sausages. *Meat Science, 65*, 833–839.
Gimeno, O., Astiasaran, I., & Bello, J. (2001). Calcium ascorbate as a potential partial substitute for NaCl in dry fermented sausages: Effect on colour, texture and hygienic quality at different concentrations. *Meat Science, 57*, 23–29.
Gonzalez, C. A., & Riboli, E. (2006). Diet and cancer prevention: Where we are, where we are going. *Nutrition and Cancer – An International Journal, 56*(2), 225–231.
Gonzalez, C. A., Jakszyn, P., Pera, G., Agudo, A., Bingham, S., Palli, D., et al. (2006). Meat intake and risk of stomach and esophageal adenocarcinoma within the European Prospective Investigation into Cancer and Nutrition (EPIC). *Journal of the National Cancer Institute, 98*(5), 345–354.
Grillenberger, M., Murphy, S. P., Neumann, C. G., Bwibo, N. O., Verhoef, H., & Hautvast, J. G. A. J. (2007). The potential of increased meat intake to improve iron nutrition in rural Kenyan school children. *International Journal for Vitamin and Nutrition Research, 77*(3), 193–198.
Grün, I. U., Ahn, J., Clarke, A. D., & Lorenzen, C. L. (2006). Reducing oxidation of meat. *Food Technology, 60*(1), 36–43.
Hall, M. N., Campos, H., Li, H. J., Sesso, H. D., Stampfer, M. J., Willett, W. C., et al. (2007). Blood levels of long-chain polyunsaturated fatty acids, aspirin, and the risk of colorectal cancer. *Cancer Epidemiology Biomarkers & Prevention, 16*(2), 314–321.
Hambidge, K. M., & Krebs, N. F. (2007). Zinc deficiency: A special challenge. *Journal of Nutrition, 137*(4), 1101–1105.
Hansen, R. D., Sorensen, M., Tjonneland, A., Overvad, K., Wallin, H., Raaschou-Nielsen, O., et al. (2007). XPA A23G, XPC Lys939Gln, XPD Lys751Gln and XPD Asp312Asn polymorphisms, interactions with smoking, alcohol and dietary factors, and risk of colorectal cancer. *Mutation Research – Fundamental and Moleclar Mechnisms of Mutagenesis, 619*(1–2), 68–80.
He, F. J., Nowson, C. A., Lucas, M., & MacGregor, G. A. (2007). Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: Meta-analysis of cohort studies. *Journal of Human Hypertension, 21*(9), 717–728.
Honikel, K. O. (2008). The use and control of nitrate and nitrite for the processing of meat products. *Meat Science, 78*, 68–76.
Huang, X. (2003). Iron overload and its association with cancer risk in humans: Evidence for iron as a carcinogenic metal. *Mutation Research, 533*, 153–171.
Huang, K., Sandler, R. S., Millikan, R. C., Schroeder, J. C., North, K. E., & Hu, J. (2006). GSTM1 and GSTT1 polymorphisms, cigarette smoking, and risk of colon cancer: A population-based case-control study in North Carolina (United States). *Cancer Causes & Control, 17*(4), 385–394.
Huang, C. C., Chien, W. P., Wong, R. H., Cheng, Y. W., Chen, M. C., Chou, M. C., et al. (2007). NAT2 fast acetylator genotype is associated with an increased risk of colorectal cancer in Taiwan. *Diseases of the Colon & Rectum, 50*(7), 981–989.
Jakszyn, P., Bingham, S., Pera, G., Agudo, A., Luben, R., Welch, A., et al. (2007). Endogenous versus exogenous exposure to *N*-nitroso compounds and gastric cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC-EURGAST) study. *Carcinogenesis, 27*(7), 1497–1501.
Jiménez-Colmenero, F., Carballo, J., & Cofrades, S. (2001). Healthier meat and meat products: Their role as functional foods. *Meat Science, 59*, 5–13.
Kimura, Y., Kono, S., Toyomura, K., Nagano, J., Mizoue, T., Moore, M. A., et al. (2007). Meat, fish and fat intake in relation to subsite-specific risk of colorectal cancer: The Fukuoka Colorectal Cancer Study. *Cancer Science, 98*(4), 590–597.
Kiss, I., Orsos, Z., Gombos, K., Bogner, B., Csejtei, A., Tibold, A., et al. (2007). Association between allelic polymorphisms of metabolizing enzymes (CYP 1A1, CYP 1A2, CYP 2E1, mEH) and occurrence of colorectal cancer in Hungary. *Anticancer Research, 27*(4C), 2931–2937.
Kondaiah, N., & Pragati, H. (2005). Meat sector and its development. *Indian Journal of Animal Science, 75*, 1453–1459.
Krebs, J. R. (2001). Science, uncertainty and policy: food for thought. *Toxicology Letters, 120*(1–3), 89–95.
Kuhnle, G. G. C., & Bingham, S. A. (2007). Dietary meat, endogenous nitrosation and colorectal cancer. *Biochemical Society Transactions, 35*(Part 5), 1355–1357.
Kuhnle, G. G. C., Story, G. W., Reda, T., Mani, A. R., Moore, K. P., Lunn, J. C., et al. (2007). Diet-induced endogenous formation of nitroso compounds in the GI tract. *Free Radical Biology and Medicine, 43*(7), 1040–1047.
Kury, S., Buecher, B., Robiou-du-Pont, S., Scoul, C., Sebille, V., Colman, H., et al. (2007). Combinations of cytochrome p450 gene polymorphisms enhancing the risk for sporadic colorectal cancer related to red meat consumption. *Cancer Epidemiology Biomarkers & Prevention, 16*(7), 1460–1467.
Larsson, S. C., Bergkvist, L., & Wolk, A. (2006). Processed meat consumption, dietary nitrosamines and stomach cancer risk in a cohort of Swedish women. *International Journal of Cancer, 119*(4), 915–919.
Larsson, S. C., & Wolk, A. (2006). Meat consumption and risk of colorectal cancer: A meta-analysis of prospective studies. *International Journal of Cancer, 119*(11), 2657–2664.
Larsson, S. C., & Wolk, A. (2007). Obesity and colon and rectal cancer risk: A meta-analysis of prospective studies. *American Journal of Clinical Nutrition, 86*(3), 556–565.
Laursen, K., Adamsen, C. E., Laursen, J., Olsen, K., & Moller, J. K. S. (2008). Quantification of zinc-porphyrin in dry-cured ham products by spectroscopic methods. Comparison of absorption, fluorescence and X-ray fluorescence spectroscopy. *Meat Science, 78*(3), 336–341.
Lee, J. Y., & Kunz, B. (2005). The antioxidant properties of baechu-kimchi and freeze-dried Kimchi-powder in fermented sausages. *Meat Science, 69*, 741–747.
Lee, R. A., Kim, H. A., Kang, B. Y., & Kim, K. H. (2006). Hemoglobin induces colon cancer cell proliferation by release of reactive oxygen species. *World Journal of Gastroenterology, 12*(35), 5644–5650.
Lewin, M. H., Bailey, N., Bandaletova, T., Bowman, R., Cross, A. J., Pollock, J., et al. (2006). Red meat enhances the colonic formation of the DNA adduct O6-carboxymethyl guanine: implications for colorectal cancer risk. *Cancer Research, 66*, 1859–1865.
Linseisen, J., Rohrmann, S., Norat, T., Gonzalez, C. A., Iraeta, M. D., Gomez, P. M., et al. (2006). Dietary intake of different types and characteristics of processed meat which might be associated with cancer risk – results from the 24-hour diet recalls in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Public Health Nutrition, 9*(4), 449–464.
Lopez-Bote, C. J. (2000). Dietary treatment and quality characteristics in Mediterranean meat products. In E. Decker, C. Faustman, & C. J. Lopez-Bote (Eds.), *Antioxidants in muscle foods* (pp. 345–366). New York: Wiley Interscience.
Martinez, M. E., Jacobs, E. T., Ashbeck, E. L., Sinha, R., Lance, P., Alberts, D. S., et al. (2007). Meat intake, preparation methods, mutagens and colorectal adenoma recurrence. *Carcinogenesis, 28*(9), 2019–2027.

Mirvish, S. S., Haorah, J., Zhou, L., Clapper, M. L., Harrison, K. L., & Povey, A. C. (2002). Total *N*-nitroso compounds and their precursors in hot dogs and in the gastrointestinal tract and feces of rats and mice: possible etiologic agents for colon cancer. *Journal of Nutrition, 132*, 3526S–3529S.

Münch, S., & Honikel, K. (2007). Abschlussbericht des Forschungsvorhabens "Gemüsewurstqualität: Fettreduktion bzw. Ballaststofferhöhung in Brühwurst durch den Einsatz von Gemüse und Obst" der Förderergesellschaft für Fleischforschung e. V. Institut für Chemie und Physik, Bundesforschungsanstalt für Ernährung und Lebensmittel, Standort Kulmbach (pp. 25).

Norat, T., Bingham, S., Ferrari, P., Slimani, N., Jenab, M., Mazuir, M., et al. (2005). Meat, fish, and colorectal cancer risk: The European prospective investigation into cancer and nutrition. *Journal of the National Cancer Institute, 97*(12), 906–916.

Norat, T., Bingham, S., & Riboli, E. (2005). Meat, fish, and colorectal cancer risk: The European prospective investigation into cancer and nutrition – Reply. *Journal of the National Cancer Institute, 97*(23), 1788–1789.

Norat, T., Lukanova, A., Ferrari, P., & Riboli, E. (2002). Meat consumption and colorectal cancer risk: Dose–response meta-analysis of epidemiological studies. *International Journal of Cancer, 98*(2), 241–256.

Osterlie, M., & Lerfall, J. (2005). Lycopene from tomato products added minced meat: Effect on storage quality and colour. *Food Research International, 38*, 925–929.

Paik, D. C., Wendel, T. D., & Freeman, H. P. (2005). Cured meat consumption and hypertension: an analysis from NHANES III (1988–94). *Nutrition Research, 25*, 1049–1060.

Palanca, V., Rodriguez, E., Senorans, J., & Reglero, G. (2006). Scientific bases for the development of functional meat products with combined biological activity. *Nutricion Hospitalaria, 21*(2), 199–202 (in Spanish).

Pavanello, S., Pulliero, A., & Clonfero, E. (2008). Influence of GSTM1 null and low repair XPC PAT+ on anti-B[a]PDE-DNA adduct in mononuclear white blood cells of subjects low exposed to PAHs through smoking and diet. *Mutation Research – Fundumental and Molecular Mechanisms of Mutagenesis, 638*(1–2), 195–204.

Pegg, R. B., Fisch, K. M., & Shahidi, F. (2000). The replacement of conventional meat curing with nitrite-free curing systems. *Fleischwirtschaft, 80*(5), 86–89.

Pierre, F., Tache, S., Petit, C. R., Van der Meer, R., & Corpet, D. E. (2003). Meat and cancer: haemoglobin and haemin in a low-calcium diet promote colorectal carcinogenesis at the aberrant crypt stage in rats. *Carcinogenesis, 24*, 1683–1690.

Pierre, F., Peiro, G., Tache, S., Cross, A. J., Bingham, S. A., Gasc, N., et al. (2006). New marker of colon cancer risk associated with heme intake: 1,4-dihydroxynonane mercapturic acid. *Cancer Epidemiology Biomerkers & Prevention, 15*(11), 2274–2279.

Pierre, F., Santarelli, R., Tache, S., Gueraud, F., & Corpet, D. E. (2008). Beef meat promotion of dimethylhydrazine-induced colorectal carcinogenesis biomarkers is suppressed by dietary calcium. *British Journal of Nutrition, 99*(5), 1000–1006.

Revilla, I., & Quintana, A. M. V. (2005). The effect of different paprika types on the ripening process and quality of dry sausages. *International Journal of Food Science and Technology, 40*(4), 411–417.

Rigault, C., Mazue, F., Bernard, A., Demarquoy, J., & Le Borgne, F. (2008). Changes in L-carnitine content of fish and meat during domestic cooking. *Meat Science, 78*(3), 331–335.

Ruusunen, M., & Puolanne, E. (2005). Reducing sodium intake from meat products. *Meat Science, 70*, 531–541.

Schmidt, G. R., Means, W. J., & Clarke, A. D. (1986). Using restructuring technology to increase red meat value. *Journal of Animal Science, 62*, 1458–1462.

Sentandreu, M. A., & Toldra, F. (2007). Evaluation of ACE inhibitory activity of dipeptides generated by the action of porcine muscle dipeptidyl peptidases. *Food Chemistry, 102*(2), 511–515.

Shahidi, F., & Samaranayaka, A. G. P. (2004). Brine. In W. K. Jensen, C. Devine, & M. Dikeman (Eds.), *Encyclopedia of meat sciences* (pp. 366–374). London: Elsevier Academic Press.

Shin, A., Shrubsole, M. J., Rice, J. M., Cai, Q., Doll, M. A., Long, J. R., et al. (2008). Meat intake, heterocyclic amine exposure, and metabolizing enzyme polymorphisms in relation to colorectal polyp risk. *Cancer Epidemiology Biomarkers & Prevention, 17*(2), 320–329.

Sindelar, J. J., Cordray, J. C., Olson, D. G., Sebranek, J. G., & Love, J. A. (2007). Investigating quality attributes and consumer acceptance of uncured, no-nitrate/nitrite-added commercial hams, bacons, and frankfurters. *Journal of Food Science, 72*(8), S551–S559.

Skjelbred, C. F., Saebo, M., Hjartaker, A., Grotmol, T., Hansteen, I. L., Tveit, K. M., et al. (2007). Meat, vegetables and genetic polymorphisms and the risk of colorectal carcinomas and adenomas. *BMC Cancer, 7*(228).

Skjelkvåle, R., Tjaberg, T. B., & Valland, M. (1974). Comparison of salami sausage produced with and without addition of sodium nitrite and sodium nitrate. *Journal of Food Science, 39*, 520–524.

Sun-Il, L., Hong-Young, M., Jung-Myun, K., Jin, K., Byung-Wook, M., Jun-Won, U., et al. (2008). Relationship between meat and cereal consumption and colorectal cancer in Korea and Japan. *Journal of Gastroenterology and Hepatology, 23*, 138–140.

Tang, S. Z., Kerry, J. P., Sheehan, D., Buckley, D. J., & Morrissey, P. A. (2000). Dietary tea catechins and iron-induced lipid oxidation in chicken meat, liver and heart. *Meat Science, 56*, 285–290.

Tarrant, P. V. (1998). Some recent advances and future priorities in research for the meat industry. *Meat Science, 49*, S1–S16.

Toden, S., Bird, A. R., Topping, D. L., & Conlon, M. A. (2007). High red meat diets induce greater numbers of colonic DNA double-strand breaks than white meat in rats: attenuation by high-amylose maize starch. *Carcinogenesis, 28*(11), 2355–2362.

Valencia, I., Ansorena, D., & Astiasarán, I. (2006). Nutritional and sensory properties of dry fermented sausages enriched with *n*-3 PUFAs. *Meat Science, 72*, 727–733.

Valencia, I., Ansorena, D., & Astiasaran, I. (2007). Development of dry fermented sausages rich in docosahexaenoic acid with oil from the microalgae Schizochytrium sp.: Influence on nutritional properties, sensorial quality and oxidation stability. *Food Chemistry, 104*(3), 1087–1096.

Ward, M. H., Cross, A. J., Divan, H., Kulldorff, M., Nowell-Kadlubar, S., Kadlubar, F. F., et al. (2007). Processed meat intake, CYP2A6 activity and risk of colorectal adenoma. *Carcinogenesis, 28*(6), 1210–1216.

WHO (2003). Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO expert consultation, WHO technical report series 919, 148 pp. <http://www.who.int/dietphysicalactivity/publications/trs916/summary/en/print.html>.

Wirth, F. (1986). Kochsalzverminderung bei Fleischerzeugnissen-Möglichkeiten und Grenzen. *Mitteilungsblatt Der Baff, 92*, 6914–6918.

Wirth, F. (1988). Salzen und Pökeln in Technologie der Kochwurst und Kochpökelware. *Bundesanstalt für Fleischforschung, Kulmbacher Reihe, 8*, 53–73.

Wirth, F. (1991). Restricting and dispensing with curing agents in meat-products. *Fleischwirtschaft, 71*(9), 1051–1054.

World Cancer Research Fund/American Institute for Cancer Research (2007). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective* (517 pp.). Washington, DC: American Institute for Cancer Research. <http://www.dietandcancerreport.org/?p=ER>.

Yilmaz, Y. (2006). Novel uses of catechins in foods. *Trends in Food Science and Technology, 17*, 64–71.

Yurchenko, S., & Mölder, U. (2007). The occurrence of volatile *N*-nitrosamines in Estonian meat products. *Food Chemistry, 100*, 1713–1721.

Zhang, X., Kong, B. H., & Xiong, Y. L. L. (2007). Production of cured meat color in nitrite-free Harbin red sausage by *Lactobacillus fermentum* fermentation. *Meat Science, 77*(4), 593–598.