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Colorectal cancer-incidence in relation to consumption of red and processed meat

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Sammanfattning

Grov- och ändtarmscancer är både i Europa och globalt två vanliga cancerformer. Sammantaget är det den tredje vanligaste cancerformen i världen och den näst vanligaste i Europa, och den har dödlig utgång i nästan häften av fallen. Det finns i den vetenskapliga litteraturen en stor samstämmighet i att de livsmedel vi äter, och de ämnen som finns i dessa, har en viktig roll både för att hindra och initiera cancer i grov- och ändtarmen (kolorektalcancer). World Cancer Research Fund (WCRF) har tagit fasta på detta i sina två rapporter, vilka utkom först 1997 och senare i reviderad form 2007. I den senare rapporten ges, med avsikt att minska cancerinsjuknandet, tio rekommendationer baserade på nuvarande kunskap om den betydelse livsmedel, näring och fysisk aktivitet spelar för canceruppkomst. En av slutsatserna i WCRF 2007-rapporten är att det finns övertygande vetenskapliga bevis för att konsumtion av rött och processat kött ökar risken för uppkomst av kolorektalcancer. En konsekvens av denna slutsats är att WCRF rekommenderar konsumenter att begränsa konsumtionen av rött kött och undvika processat kött, med en personlig rekommendation till individer som äter rött kött, att konsumera högst 500 g per vecka av detta livsmedel, och att litet om ens något av detta ska vara processat. Motsvarande folkhälsomål innebär att populationens medelintag av rött kött ska vara högst 300 g per vecka, av vilket litet om ens något ska vara processat.

De nya rekommendationerna om kött från WCRF har lett till att nationella myndigheter behöver se över sina nuvarande råd om köttkonsumtion. I Sverige ansvarar Livsmedelsverket för denna uppdatering och denna rapport är en del av denna översyn. Ett komplement som beaktats vid utvärderingen av WCRF 2007-rapporten har varit fortlöpande uppdateringar från WCRF, samt en sammanställning av relevanta studier som Livsmedelsverket har låtit göra, gällande artiklar från 2005 och fram till idag. Den huvudsakliga frågan som ska besvaras i denna rapport är om rekommendationerna från WCRF-rapporterna är motiverade och relevanta för svenska konsumenter.

Den senaste svenska matvaneundersökningen bland den vuxna befolkningen (18-75 år), Riksmaten 2010-11, visade att medelintaget av tillagat rött och processat kött, speciellt hos de manliga konsumenterna, är avsevärt högre än den konsumtion som rekommenderas av WCRF. Med utgångspunkt från konsumtionsdata och scenarie-beräkningar gjordes en uppskattning av om en minskad köttkonsumtion till WCRFs nivå skulle få negativa näringsmässiga konsekvenser. Den generella slutsatsen var att en minskning av köttkonsumtionen enligt WCRF-rekommendationen inte får några negativa näringsmässiga konsekvenser för befolkningen.

De slutsatser som dras i WCRF 2007-rapporten är grundade på resultat från i första hand kohort-studier. Dessa studier sammantaget, som rapporterar om cancerrisker för grovtarm, ändtarm, eller kolorektum har, om man jämför den högsta konsumtionsgruppen med den lägsta, en relativ risk (RR) på över 1 i nästan samtliga fall. Ett dos-responssamband kunde observeras i kohortdata och en ökning av RR kunde observeras vid en konsumtion över 700 g per vecka av rött kött, eller vid en konsumtion av 350 g per vecka av

processat kött. Det är dock en mycket stor variation i vad som beskrivits som processat kött, och kategorierna av processat kött kan skilja från studie till studie. Detta innebär i många fall stora svårigheter när man ska utvärdera och jämföra huruvida ett samband finns i epidemiologiska studier mellan konsumtion av processat kött och uppkomst av kolorektalcancer.

Försöker vi sammanfatta de kohortstudier och review-artiklar som publicerats efter 2007, alltså efter den senaste WCRF-rapporten, framgår att dessa artiklar i stort stödjer slutsatserna för ett samband mellan hög konsumtion av rött och processat kött och en ökad risk för kolorektalcancer. Orsakssambandet är dock inte helt utrett, då det saknas en fastställd enskild mekanism för att förklara hur rött och processat kött kan ge upphov till kolorektalcancer. De mest diskuterade och undersökta föreslagna mekanismerna i vetenskaplig litteratur är fett, heterocykliska aminer, nitrit och nitrosaminer, hämjärn, kolesterol, salt, protein, virus samt indirekta mekanismer, såsom alkohol, högt energiintag och lågt grönsaksintag. Det är troligt att cancerprocessen fordrar att flera faktorer agerar tillsammans i en sekvens av händelser, genom initiering, promotion och progression. Möjliga faktorer och mekanismer bakom alla dessa steg kan antas finnas närvarande i rött och processat kött, men troligen i varierande mängd.

WCRF-rapporten har i sin sannanvägning kommit fram till att ett intag av mer än 500 g rött kött per vecka ökar risken för kolorektalcancer, medan processat kött anses som en högre risk på grund av dess högre cancerpotens per g kött och därför bör konsumeras mycket sparsamt eller helt undvikas. En svaghet i WCRFs bedömning är de skilda typer av processat kött som ingår i olika studier samt en osäkerhet bakom mekanismerna.

Sammanfattningsvis bedömer vi att detfinns det goda vetenskapliga skäl att begränsa konsumtionen av rött kött, men att det är tveksamt om konsumenter, i enlighet med WCRFs rekommendation, helt ska undvika processat kött. Vi har samtidigt att beakta de vetenskapliga rön som indikerar en högre kolorektalcancer-risk av processat jämfört med rött kött, räknat på viktsbasis, samt det faktum att konsumtionen av rött och processat kött i de nordiska länderna är avsevärt högre än vad WCRF rekommenderar. Baserat på WCRFs rekommendationer och andra vetenskapliga rapporter leder detta fram till vår slutsats att på individnivå begränsa intaget av rött och processat kött till högst 500 g per vecka, och då att särskilt begränsa konsumtionen av processat kött. Den aktuella risk- och nyttorapporten är ett stöd för riskhanterare när rekommendationer ska tas fram och kommuniceras.

Summary

Cancers in the colon and rectum are common worldwide, including in Europe. It is the third leading cause of cancer globally and the second leading cause of cancer in Europe, and it is fatal in just under half of all cases. Most scientific evidence judges that food and nutrition have a very important role in both the prevention and the causation of cancers of the colon and rectum. Indeed, the World Cancer Research Fund (WCRF) authoritative expert report, first published in 1997 and thoroughly revised in 2007, resulted in ten recommendations, which are derived from the evidence on food, nutrition, and physical activity. The conclusion in the updated WCRF 2007 report was that there is convincing evidence that exposure to red meat and processed meat increases risk for colorectal cancer (CRC). As a consequence, the WCRF report resulted in the recommendation to "Limit intake of red meat and avoid processed meat", with a personal recommendation saying "People who eat red meat to consume less than 500 g a week, very little if any to be processed". The public health goal on red and processed meat says "Population average consumption of red meat to be no more than 300 g a week, very little if any of which to be processed".

This new advice from WCRF resulted in an interest by national and international food and health agencies to look over their present recommendations in the light of this new meat-and-cancer advice. The present report is part of this process and has been conducted by the National Food Agency in Sweden. As a supplementary retrieval activity of recent studies on CRC and red/processed meat, we made a literature search on the Pub Med. The main question to be answered is if the conclusions of the WCRF report are justified and valid for Swedish consumers.

By use of the latest Swedish food survey for the adult population (18-75 yrs), Riksmaten 2010-11, the mean intake (obtained by food registration and questionnaire answers) of cooked red and processed meat, especially regarding male consumers, is considerably higher than what is recommended by WCRF. By use of these consumption data different scenarios were evaluated in which the meat consumption in the Nordic countries were adjusted to the WCRF, and whether this would have nutritional consequences for the consumers. The general conclusion was that an adjustment to the WCFR recommendation has negligible nutritional consequences for the general population.

The conclusions regarding red and processed meat and cancer in the WCRF 2007 Report are mainly based on cohort studies. Of these studies which reported analysis of colon, rectal, or colorectal cancer risk for the highest intake group compared to the lowest, the relative risk (RR) was above 1 in almost all studies. A dose-response relationship was also apparent from the cohort data and an increase in relative risk was shown in some studies with servings of 700 g red meat per week or 350 g processed meat per week, respectively. There is a huge variety of processed meat and it is difficult to sort them by categories. Consequently, epidemiologic studies on the relation between intake of pro-

cessed meat and colon cancer are many times difficult to evaluate and compare because of the huge variety of processed meat and an incomplete description of the products that have been specifically studied.

If we try to conclude the above present cohort and review papers published after 2007, it is obvious that the studies as a whole strengthen the evidence, although still not completely causative, for an a association between a high consumption of red and processed meat and an increased CRC risk. This possible association is somewhat weakened by the fact that no single mechanism can explain how red and processed meat act in the development of CRC. The most discussed and investigated mechanisms and associated factors that have been presented in the scientific literature are fat, heterocyclic amines, nitrite and N-nitroso compounds, heme iron, cholesterol, salt, protein, viruses, and indirect mechanisms (for example alcohol, high energy intake, low intake of vegetables). Thus, the process of carcinogenesis may require several different factors acting in concert in a sequential way during initiation, promotion and progression. Potential factors and mechanisms for all these steps seem to have been identified in red and processed meat.

After weighing all results the WCRF Report concluded that intake above 500 g/week of red meat increases the risk for CRC, whereas processed meat, due to its claimed higher CRC potency per weight basis, is regarded as a higher risk and intake should therefore be very little, if any. However, a weakness in the WCRF evalution is the variety of processed meat categories in the studies and the non-conclusive evidence of the actual mechanisms behind the cause of CRC, by both red and processed meat.

We conclude that scientific data support a restricted consumption of red meat, but it is questionable, as stated by WCRF, to totally avoid consumption of processed meat. Nevertheless, it has to be considered that scientific data indicate a higher CRC risk of processed meat compared to red meat on a weight basis and the fact that the Nordic intake of red and processed meat is considerably higher than the WCRF recommendation. Based on the WCRF Report and other scientific data our conclusion is to restrict the intake of red and processed meat, on a personal level, not to more than 500 g per week, and to particularly decrease the consumption of processed meat. The present risk- and benefit assessment report is a support for risk managers when recommendations will be formulated and communicated.

1 General introduction

The report Food, Nutrition and the Prevention of Cancer: a global perspective (produced by the World Cancer Research Fund together with the American Institute for Cancer Research), has been a very important and authoritative source concerning issues on food, nutrition, and cancer prevention for the last 10 years and more. On publication in 1997 it was regarded as the standard document worldwide for policy-makers in government at all levels, for civil society and health professional organisations, and in teaching and research centres of academic excellence (WCRF 1997). However, since 1997 the amount of scientific literature on this subject was dramatically increased and the need for a new report increased. In 2001 the WCRF International began the work with the new report, which was published in November 2007 (WCRF 2007).

The evidence and judgements that form the basis for the conclusions of the WCRF 2007 Report have been produced over a period of five years from 20 systematic literature reviews, compiled by nine independent centres of scientific excellence, covering 20 cancer sites. By use of a common methodology, these reviews give an examination of the relevant types of epidemiological and experimental evidence that are available today. The findings were subsequently assessed and judged by the WCRF Panel, and the compiled documentation is given in part 2 of the WCRF 2007 Report.

The WCRF Panel's work has resulted in ten recommendations (in part 3), which are derived from the evidence on food, nutrition, and physical activity. These ten recommendations are given in Table 1. The Panel has concluded that the evidence that high body fatness and also physical inactivity are causes of a number of cancer forms, also including common cancers, is particularly strong. For this reason, the first three recommendations (on Body fatness, Physical activity and Foods and drinks that promote weight gain) are produced as a basis for policies and programmes whose purpose is to maintain healthy body weights and to make people maintain physical activity, throughout life. The following five are given without order of priority, and the last two are given to specific groups, one to breastfeeding mothers and the other to cancer survivors. Other factors that modify the risk of cancer, and not included in the WCRF Report, are smoking, infectious agents, radiation, industrial chemicals, and medication. Nevertheless, the Panel emphasise the importance of not smoking and of avoiding exposure to tobacco smoke.

The Panel is also aware of that life and food patterns that could induce cancer could be influenced by social and environmental factors. These broader factors, as well as policies for creating healthier societies, are subject of the WCRF Policy Report from 2008 (found at www.dietandcancerreport.org)

Table 1. Recommendations of the WCRF Report of 2007

No.	Title	Recommendation
1	Body fatness	Be as lean as possible within the normal range of body weight
2	Physical activity	Be physically active as part of everyday life
3	Foods and drinks that promote weight gain	Limit consumption of energy-dense foods. Avoid sugary drinks
4	Plant foods	Eat mostly foods of plant origin
5	Animal foods	Limit intake of red meat and avoid processed meat
6	Alcoholic drinks	Limit alcoholic drinks
7	Preservation, processing, preparation	Limit consumption of salt. Avoid mouldy cereals (grains) or pulses (legumes)
8	Dietary supplements	Aim to meet nutritional needs through diet alone
9	Breastfeeding	Mothers to breastfeed; children to be breastfed
10	Cancer survivors	Follow the recommendations for cancer prevention

2 The WCRF Report – red and processed meat

Already in the first World Cancer Research Fund (WCRF) authoritative expert report from 1997 it is stated that "evidence shows that red meat probably increases risk and processed meat possibly increases risk of colorectal cancer" (WCRF 1997). This report was updated and new literature references included in a new report that was published by the World Cancer Research Fund in 2007 (WCRF 2007). The conclusion in the updated report was that there is convincing evidence that exposure to red meat and processed meat increases risk for colorectal cancer (CRC). It was also concluded that the evidence in this recent report is stronger than in the previous report from 1997. This resulted in the recommendation to "Limit intake of red meat and avoid processed meat", with a personal recommendation saying "People who eat red meat to consume less than 500 g a week, very little if any to be processed". Public health goals are also given, primarily aimed at health professionals, and these health goals focus on informing population groups. The public health goal on red and processed meat says "Population average consumption of red meat to be no more than 300 g a week, very little if any of which to be processed".

Of the recommendations given in the WCRF Report of 2007, the majority were already known by the broad public and in many instances similar to those given earlier (WCRF Report of 1997). However, in Swedish as well as international media much interest was given the recommendation on red and processed meat, probably because this advice was identified as being new, or at least much more restrictive, than those earlier given. This new advice from WCRF also resulted in an interest by national and international food and health agencies to look over their present recommendations in the light of this new meat-and-cancer advice. The present report is part of this process and has been conducted by the National Food Agency in Sweden. The main question to be answered is if the conclusions of the WCRF report are justified and valid for Swedish consumers. The present report is a support for risk managers when recommendations will be formulated and communicated.

3 Red and processed meat and colorectal cancer (CRC) – general comments

There were an estimated 3.45 million new cases of cancer in Europe 2012 (Ferlay et al., 2012). Cancers in the colon and rectum are common worldwide, including Europe. It is the third leading cause of cancer globally and the second leading cause of cancer in Europe, and it is fatal in just under half of all cases (Ferlay et al., 2013). Colorectal cancer is somewhat more common in men than in women. Individual country- and cancer site-specific studies suggest that the age-adjusted incidence of cancers in the colon and rectum has increased in the European population over the two past decades (Arnold et al., 2013). Thus, it seems that rates of these cancers increase with industrialisation and urbanisation. It has been suggested that the rapid societal and economic transition in many countries means that any reductions in infection-related cancers are offset by an increasing number of new cases that are more associated with reproductive, dietary and hormonal factors (Bray et al., 2012). Accordingly, cancers in the colon and rectum were earlier more frequent in countries with high mean income, but is now increasing in middle- and low-income countries, probably reflecting a general income increase. It still remains relatively uncommon in Africa and much of Asia. However, cancer is believed to become a major cause of morbidity and mortality in the coming decades in every region of the world (Bray et al., 2012).

Most scientific evidence, and consequently also the conclusions by the WCRF Panel, judge that food and nutrition have a very important role both in the prevention and in the causation of cancers of the colon and rectum. Of these factors, there is strong evidence that red meat and processed meat increase the risk, while e.g. dietary fibres will decrease the risk. Other factors that increase the risk for CRC are, according to the WCRF Report, alcoholic drinks, body (general) fatness, abdominal fatness and adult attained height.

4 Definitions

In the 2007 WCRF Report the following definitions are used:
Red meat: *refers to beef, pork, lamb, and goat meat from domestic animals, including that contained in processed foods.*

Meat from wild animals such as elk, deer, and boar may be considered as red meat, but are not included in the WCRF Report definitions, and studies on consumption of these meat types in relation to CRC risk are lacking.

Processed meat: *refers to meat preserved by smoking, curing of salting, or addition of chemical preservatives, including that contained in processed foods.*

There is a huge variety of processed meat and it is difficult to sort them by categories. Examples of parameters involved in the making of processed meat are curing (adding salt and other additives), drying, smoking, cooking, and packing. Processed meat includes for example bacon, ham (raw, smoked, or cooked), heated sausages like hot dogs, raw sausages (such as salami), bologna, blood sausages, liver paté (or liverwurst) and other patés and spread meat, luncheon meat and other cold cuts, canned meat and corned meat.

All these different processes may generate products with various potential health hazards. Consequently, epidemiologic studies on the relation between intake of processed meat and colon cancer are many times difficult to evaluate and compare because of the huge variety of processed meat and an incomplete description of the products that have been specifically studied (see Table 3b). No systematic epidemiological studies have compared different kind of processed meats in relation to CRC incidence. Moreover, national consumption patterns of the different categories of processed meat can be assumed to be quite different which means that results from studies performed in different countries are difficult to compare directly.

5 Basis for conclusions on red and processed meat in the WCRF Report

The results on red meat and colorectal cancer used in the WCRF 2007 Report come from 17 cohort and 71 case-control studies (in the Report, the number of cohort studies was incorrectly stated as 16). In the case of processed meat and colorectal cancer, 14 cohort and 44 case-control studies were used. In the majority of these cohort studies, the effects of both red and processed meat were studied, but in seven cohorts only red meat effects were studied and in 11 cohorts only those of processed meat were investigated. In the WCRF 2007 Report highest scientific confidence is given the cohort studies, and these are presented in Tables 2 (red meat) and 3 (processed meat). In these tables, details on consumption of red and processed meat are given for cancer incidence in colon and rectum, (Tables 2a, 3a), and for consumption details, meat definitions, significant effects, and trends (Tables 2b, 3b). In addition to the mentioned cohort studies, 11 meta-analyses were performed on the connection between red meat and colorectal cancer, and 6 meta-analyses were registered regarding processed meat and colorectal cancer. Because of the abundant data from the cohort studied, the Panel found no reason to summarise the case-control studies in the WCRF Report.

6 Results and conclusions based on the WCRF 2007 Report regarding red and processed meat and CRC risk

Red meat

The conclusions regarding red meat and cancer in the WCRF 2007 Report are mainly based on 17 cohort studies (Tables 2a,b). Of these, 11 are studies from USA, two from Finland, and the remaining studies come from Australia, Holland and Sweden, and from an inter-european research group (10 participating countries). The number of participants varied between about 23 000 (Chen et al., 1998) to up to 478 000 (Norat et al., 2004), and the gender were either mixed or representing males or females separately. In the WCRF 2007 Report, the data on red meat and colorectal cancer has been visualised as so called forests plots (Figures 4.3.1. to 4.3.3 in the Report). In the studies which reported analysis of colon, rectal, or colorectal cancer risk for the highest intake group, compared to the lowest, the relative risk was above 1 in all studies (n=13) and results were statistically significant in four of them. In meta-analyses of some of the cohort studies in which intakes were similarly expressed, the summary effects estimates were 1.43 (95 % CI 1.05-1.94) per times/week and 1.29 (95 % CI 1.04-1.60) per 100 g/day, respectively.

A dose-response relationship was also apparent from the cohort data, i.e. with five or more servings of red meat per week there was an increased risk for CRC (Figure 4.3.4 in the Report). However, from these studies it is difficult to read out more precisely at what consumption level the risk actually is increased. Nevertheless, an increase in relative risk was shown in some studies with servings of 700 g red meat per week (Figure 4.3.3 in the report).

Most of the studies considered to various degrees confounding factors in the assessment, although more or less detailed. At the same time, it could be noted that the consumption range sometimes was narrow, or higher than “normal”, or that the specificity of the meat was questionably described. Apart from the mentioned cohort studies, a larger number of case-control studies (71 studies mentioned in the Report) have been performed on this subject. These studies are also part of the general evaluation but have not been used in the same detail as the cohort studies.

Processed meat

The conclusions regarding processed meat and cancer in the WCRF 2007 Report are mainly based on 14 cohort studies, but there are also 44 case-control studies on this subject (Tables 3 a,b). Of the 14 cohort studies, 7 studies have been performed in USA, 2 are from Holland, and the remaining studies come from Australia, Finland, Japan, Sweden,

and from Europe. The number of participants varied between 1 524 and 478 000, and the studies represented both mixed and separated gender approaches. In the WCRF 2007 Report, the data on processed meat and colorectal cancer has been shown as forest plots (Figures 4.3.5 - 4.3.6) and dose response associations are given in Figures 4.3.7- 4.3.8.

Of the 14 studies, 12 studies reported an increased cancer risk (colon, rectum, colorectum), for the highest intake group when compared to the lowest, with a statistical significance in three studies. Meta-analysis (on five studies) gave a summary effects estimate of 1.21 (95% CI 1.04-1.42) per 50 g/day. A dose-response relationship was apparent from cohort studies that measured consumption in meals or times/day.

As for red meat, from studies on processed meat showing an increased risk for colorectal cancer it is difficult to read out at what consumption level the risk actually is increased. Nevertheless, an increase in relative risk was shown in some studies with servings of 350 g of processed meat per week (Figure 4.3.6 in the Report).

The studies generally considered confounding factors, but there were shortcomings or lack in confounding discussions in some studies. Thus, many studies did not define the servings by weights, there were lack of intake effect correlations and of meat definition data, or a general lack of information of the study design.

7 Epidemiological studies on red and processed meat and CRC, published after the WCRF 2007 Report

After the report was published by WCRF in 2007, some new studies have been published that should be considered in the overall evaluation of a possible association between colon cancer and the intake of red meat and processed meat. Thus, WCRF has produced several updated documents (Continuous Update Projects, CUP) to include new documents to an extended data base. In the CUP of WCRF 2011 it was concluded that current evidence indicate an elevated risk of colorectal adenomas with intake of red and processed meat, which is in accordance with the previous WCRF Report 2007. For both red and processed meat the conclusion is that these meat products are convincing causes of CRC. This was recently further supported by an updated systematic review and meta-analysis of epidemiological studies compiled within the WCRF project (Aune et al., 2013).

As a supplementary retrieval activity of recent studies on CRC and red/processed meat, we made a literature search on the Pub Med. On the 3rd of October 2013, a scientific literature search was executed by use of the following search string on the Pub Med search motor: “Cancer AND (colorectal OR colon OR rectum) AND meat AND (processed OR red) AND (cohort OR case-control)”. As a result, some 125 articles were found by limiting the search back to, and including, year 2005 (older paper are assumed be included in WCRF 2007 document). Of these articles, a selection of 57 papers was made based on article headings and abstract information. Of these 57, 17 were defined as cohort, or nested case-control studies, 26 were case-control studies, and 14 were reviews. The cohort and review papers are listed in Table 4, where also short conclusions of the papers are given. Obviously, the obtained papers were among those retrieved in the above mentioned WCRF CUP Reports.

The listed papers in Table 4 are in some cases focussed on polymorphism or other specific questions related to intake of meat and associated CRC risk, whereas in other papers, the dietary pattern as a whole, but also including consumption of red and processed meat, is studied in relation to CRC risk. Nevertheless, also these papers give additional information regarding associations between red and processed meat and colon/rectal cancer risk. In summary, the findings in the 17 recent cohort studies show that a majority of the papers indeed find associations between red and/or processed meat and CRC. Thus, 11 of the 17 papers show increases in morbidity or mortality in CRC or colon/rectal cancer separately, or a decreased risk if red meat is substituted with chicken or fish meat. From the papers showing an increased risk it was not possible, at least not without a deeper evaluation of the documents, to claim that either red or processed meat constituted the higher risk at least when based on serving frequencies. Notably, of the six papers that do not observe an increased CRC risk associated to red or processed meat intake, three de-

scribe the same Danish cohort, and for two other papers data from the same multi-ethnic cohort was used. Regarding the 14 review articles, the conclusion in 10 of these was that consumption of red and/or processed meat, alone or as part of a Western diet, was associated to CRC or to pre-cancer stages. In contrary, three papers concluded that an association between red/processed meat and CRC was not likely. Finally one paper could relate CRC risk only to some genotypes, which may indicate that only certain individuals are sensitive and therefore may explain at least part of the variation in results among different studies. The case-control studies (26 papers), considered to be of less significance in the evaluation compared to the cohort studies, were not evaluated in detail. A short overview of these studies suggests that the results regarding red and processed meat and CRC risk are generally in accordance with those given in the cohort compilation.

If we try to conclude the above present cohort and review papers published after 2007, it is obvious that the studies as a whole strengthen the evidence, although still not completely causative, for an a association between a high consumption of red and processed meat and an increased CRC risk. Notably, a study where red meat was substituted with chicken or fish meat (not associated with CRC) showed a decreased risk for development of CRC (Daniel et al., 2011). However, altogether these studies could not fully show that processed meat was a significantly more potent colorectal carcinogen than red meat. Thus, even if the studies largely confirm the conclusions given in the WCRF 2007 Report, the picture may be somewhat more complex.

8 Proposed mechanisms for association between meat consumption and CRC development

Epidemiological (WCRF, 2007) and experimental (Corpet, 2011) evidence supports the hypothesis that intake of red and processed meat is associated with an increased risk for the development of colorectal cancer (CRC). However, results from meta-analyses indicate that the risk associated with consumption of one gram of processed meat is two to ten times higher than the risk associated with one gram of fresh red meat (Santarelli et al., 2008). Based on these results it is reasonable to assume that processed meat, compared to red meat, contains a higher amount of the components that are responsible for carcinogenesis in the colorectum. In fact, processed meat, compared to red meat, generally contains more fat, more additives such as salt and nitrite, as well as oxidized cholesterol due to long-time storage.

Few experimental studies have been directly carried out on processed meat but the studies undertaken on red meat have made it possible to propose some possible mechanisms. Based on available epidemiological and experimental data there is evidently no known single mechanism that can explain how red and processed meat act in the development of CRC. It seems that colorectal cancer always arises in the context of genomic instability, where there is inactivation of tumor suppressor genes, as well as activation of oncogene pathways and growth factor pathways (Watson and Collins, 2011). In line with this, it has been suggested that development of CRC caused by red and processed meat requires presence of several factors acting in concert and by different mechanisms, i.e. it may be a multifactorial disease.

Cancer as a disease usually develops slowly, and the process of carcinogenesis involves a variety of biological changes in the cells which to a great extent reflect the structural and functional alterations in the genome of the affected cell. At present it is known that the pathogenesis of the disease consists of at least three operationally defined stages beginning with the initiation, followed by an intermediate stage of promotion, ending up in a final stage of progression.

Initiation is an early alteration in individual cells within the affected tissue, for example caused by different chemicals. In addition, spontaneous pre-neoplastic changes in cells continuously occur that has to be taken care of. In addition, the metabolism of initiating agents to nonreactive forms and the efficacy of DNA repair in tissue cells can alter the progress of initiation. Unlike chemicals that induce initiation, there is no evidence that promoting agents or their metabolites directly interact with DNA or that metabolism is required for their effectiveness. A distinctive characteristic of promotion, as opposed to initiation and progression, is the reversible nature of this stage. The final stage in carcinogenesis is the irreversible progression that is characterized by changes in growth rate,

invasiveness, metastatic frequency, hormonal responsiveness and morphological characteristics.

The exact mechanisms of CRC development are not known, but several potential factors present in red and processed meat, as well as possible pathways for cancer induction, have during the years been suggested. Consequently, the process of carcinogenesis may require several different factors acting in concert in a sequential way during initiation, promotion and progression. Potential factors and mechanism for all these steps seem to have been identified in red and processed meat. The most discussed and investigated mechanisms and associated factors that have been presented in the scientific literature are fat, heterocyclic amines, nitrite and N-nitroso compounds, heme iron, cholesterol, salt, protein, viruses, and indirect mechanisms (for example alcohol, high energy intake, low intake of vegetables) (Santarelli et al., 2008; Corpet, 2011). The background and support for these possible mechanisms are summarized and discussed below.

Heterocyclic amines (HCAs) and Maillard reaction products. At high cooking temperatures potentially carcinogenic HCAs are formed from creatinine with specific amino acids, which means that only fried, broiled or barbecued meat contains significant amounts of these compounds (Santarelli et al., 2008). Thus, to adequately assess an individual's exposure to HCA epidemiological studies, investigating the relationship between HCA intake and cancer risk, need to specify type of meat, cooking method and degree of doneness/surface browning (Sinha et al., 1998). A proposed mechanism that well-done red meat consumption concomitant to intestinal inflammation, such as colitis, could initiate CRC, a mechanism potentiated by heme (Lakshmi et al., 2005). However, Santarelli et al. (2008) concluded that HCAs are not an important determinant of CRC in humans partly because chicken meat is the major contributor of HCA intake, but its consumption is not associated with increased CRC risk in epidemiologic studies (WCRF, 2007).

Maillard reaction products are formed during non-enzymatic browning of food during cooking. It results from a chemical reaction between an amino acid and a reducing sugar, usually requiring heat. In the process, a complex mixture of poorly characterized molecules is created. At high temperatures, a number of maillard reaction products, including acrylamide, can be formed. Ingested acrylamide is metabolized to a chemically reactive epoxide, glycidamide. In February 2009, Health Canada announced that they were assessing whether acrylamide, which occurs naturally in processed foods, is a hazard to human health and whether any regulatory action needs to be taken. In addition, acrylamide is considered a potential occupational carcinogen by US government agencies. The World Health Organization (WHO) also has concern and has initiated activities to disentangle whether there is a health risk of acrylamide in food. Consequently, this is an interesting but less well studied group of chemical agents, formed in processed foods, that needs to be further investigated.

Polycyclic aromatic hydrocarbons (PAHs). PAHs are produced from the incomplete combustion of organic compounds. Many tested PAHs, like benzo(a)pyrene (BaP), are mutagens and animal carcinogens. Main sources of PAHs for humans are cooked and smoked meat and fish, especially barbecued meat. Although based on insufficient data,

case-control studies suggest that PAHs may possibly be better candidates than HCAs, to explain that overcooked meat may cause CRC (Santarelli et al., 2008).

Nitrite and N-nitroso compounds. Nitrite gives the meat the desirable red color by combining with heme iron forming nitrosylmyoglobin. There is intake of nitrite present in meat products, intake of vegetables containing nitrate and the subsequent gastrointestinal formation of nitrite, as well as an endogenous production of nitrite. In the gastrointestinal tract this nitrite may through nitrosation of amines and amides be involved in the formation of potentially carcinogenic N-nitroso compounds (NOCs). According to the International Agency for Research on Cancer (IARC 2007) ingested nitrite, under the conditions that result in endogenous nitrosation, is probably carcinogenic to humans (classified as 2A). Human studies have shown that dietary beef meat, but not poultry, strikingly increases NOC excretion in feces (Santarelli et al., 2008). However, the nature of the NOCs formed in the gut is not fully known (Zhou et al., 2006). Consequently, even though many tested N-nitroso compounds have been shown to induce cancer in rodents, it is not yet clear whether red and processed meat-induced N-nitroso compounds are colon carcinogens.

Heme iron. Heme iron (Fe) is included in myoglobin, in red blood cell hemoglobin, and in cytochromes. Blood products (sausage, pudding) and liver pate is particularly rich in heme, followed by dark red meat products, whereas chicken meat contains little heme. It has experimentally been shown that heme iron in red meat can promote carcinogenesis by increasing cell proliferation in the mucosa, mediated by lipoperoxidation and/or cytotoxicity of fecal water. In addition, it has been reported that nitrosation might also increase the toxicity of heme iron in processed meat products. Santarelli et al. (2008) has proposed three mechanisms that may explain heme promotion of cancer, i.e. heme is metabolized in the gut into a cytotoxic and promoting factor; heme induces peroxidation of fat in foods in the gut, and the lipoperoxides would promote CRC; heme catalyzes the endogenous N-nitrosation, which increases the formation of NOCs (see above) and activation of HCAs. That heme is a link between meat intake and CRC risk is consistent with epidemiologic studies showing that red meat, but not white meat, is associated with an increased risk (Bastide et al., 2011; WCRF, 2007).

Protein. According to certain experimental findings proteins derived from meat do not promote carcinogenesis, but other results suggest that a fraction of such proteins, i.e. those that are subject to slow and thereby protracted gastrointestinal digestion, may carry such potency (Santarelli et al., 2008; Corpet et al., 1995; Le et al., 2007). Although partly degraded protein fragments and/or peptides produced by microbial activity in the gastrointestinal tract have not hitherto been associated with growth factor-like bioactivity, this risk cannot altogether be dismissed.

We therefore believe that the issue of whether potentially bioactive peptides, either derived from digested red meat or indirectly produced from such foods by intestinal microbiotic activity, can promote or sustain malignancy is far from exhaustively studied and thus needs further investigation. In addition, the level and nature of such peptides, assuming their intestinal appearance as a consequence of the actual consumption pattern, could also differ between processed and red meat.

Fat. Experimentally it has been shown that high-fat diets could promote carcinogenesis via insulin resistance or fecal bile acids (Santarelli et al., 2008). High fat intake favors the secretion of bile acids which subsequently undergo bacterial hydroxylation into deoxycholic acid and lithocholic acid. These bile acids, which are present in human stools, have in animal experimental models been shown to promote colon carcinogenesis (Bruce, 1987). In addition, fatty acids can damage the colonic epithelium and increase cell proliferation, an effect blocked by dietary calcium (Lapre et al., 1993). The suggestion that fat explain the link between CRC and meat intake is not fully supported because results from experimental studies are not consistent and epidemiologic studies have failed to confirm this association (Santarelli et al., 2008). Notably, studies have revealed that diets rich in monounsaturated fats or n-3 fatty acids were inversely correlated with the development of cancer (Rediger et al., 2009).

Cholesterol. During long-time storage, fermentation, and/or frying of fatty meat products in the presence of oxygen there are formation of oxidation products of cholesterol, a process that is inhibited by nitrite addition (Santarelli et al., 2008). In vitro studies have shown that oxysterols could excert mutagenic and genotoxic properties. The oxysterols may also have a role in oxidative stress and inflammation and to interfere in signalling pathways, which both could have roles in carcinogenesis (Jusakul et al., 2011). However, even if hydroxycholesterol has been associated with certain cancer forms such as lung cancer, there is not much evidence for the hypothesis that oxidized sterols might induce or promote CRC. Indeed, results from epidemiological studies indicate that the CRC risk associated with consumption of processed meat, where nitrite usually is added, is higher than the risk associated with fresh red meat (Santarelli et al., 2008). Consequently, dietary cholesterol does not seem to be associated to an increased CRC risk.

Salt. Salt preserves meat by stopping bacterial growth because it diffuses inside the muscle and reduces the water activity. Processed meat contains more salt than red meat. Studies have shown that intake of salty diet and salted foods seem to be associated with stomach cancer (Joossens et al., 1996), and this is particularly the case in Japan (Tsugane, 2005). Proposed mechanisms for stomach cancer induction are potentiation of colonization of Heliobacter pylori, a known risk factor for stomach cancer, and changes in the mucous viscosity of the stomach which could result in an increased exposure to carcinogens (Wang et al., 2009). However, so far no scientifically convincing link between salt and an increased risk for CRC has been published.

Viruses and bacteria. The majority of viral associated human cancers are related to infection with human papillomaviruses, hepatitis B and C viruses, and Epstein-Barr viruses (Riley et al., 2013). Together these viruses were 2002 reported to be associated with ap-

proximately 11 % of the global cancer burden (Parkin, 2006). Thus, papilloma and polyoma viruses are potentially tumorigenic viruses (Bosch and Sanjose, 2007) and it is also well-known that liver cancer may develop after concomitant exposure to hepatitis virus infection and aflatoxin (Bannasch et al., 1995). One of the key mechanisms by which some viruses promote carcinogenesis is through their integration into the human genome causing somatic mutations (Riley et al., 2013). A hypothesis has been forwarded that potentially oncogenic bovine viruses (e.g. polyoma-, papilloma- or possibly single-stranded DNA viruses) may contaminate beef preparations and lead to latent infections in the colorectal tract (zur Hausen, 2012). Moreover, if such potential carcinogenic viruses exist a synergistic effect could occur between these infections and chemical carcinogens arising in the processing steps prior to consumption of red and processed meat. This is an interesting but less well studied area that needs further investigation.

A less well studied area is whether bacteria also are potential risk factors in the development of cancer. There are some data supporting such a hypothesis. The bacteria *Bartonella henselae*, a human opportunistic pathogen, has been shown to transform human cells *in vitro*, and is together with *B. quintana* the only known bacteria to cause angiomatosis, i.e. the formation of benign tumors in blood vessels (Riley et al., 2013). Moreover, evidence was recently published that bacterial DNA integrates in the human genome and that such integration are detected more frequently in a) tumors than normal samples, b) RNA than DNA samples, c) the mitochondrial genome than the nuclear genome. From this the authors concluded that bacterial integrations occur in the human somatic genome and may play a role in carcinogenesis. This is an interesting area that needs further investigation.

Indirect mechanisms. It is possible that individuals who eat more processed meat and/or red meat also have another life style and that their consumption pattern of other food stuffs is different. However, there is no clear link to any specific factor that could explain the majority of CRC cases in epidemiological studies of intake of red meat and processed meat and the increased risk for CRC (Santarelli et al., 2008; WCRF, 2007).

Summary. It can be concluded that based on available epidemiological and experimental data there is no known single mechanism that can alone explain how red and processed meat act in the development of CRC. Instead, several different factors, of which some are mentioned above, may be needed for tumor occurrence. The presence of genotoxic agents such as heterocyclic amines and PAHs could well initiate colorectal cells. Nevertheless, it is felt that the strongest scientifically plausible mechanism involved is heme-associated induction of CRC. It has been suggested that heme iron can promote cancer by three different pathways, the formation of cytoxic heme species in the gut, the fat peroxidation pathway and the N-nitroso pathway (Corpet, 2011). According to this suggestion fat peroxidation mainly explains tumor promotion by fresh meat and the N-nitroso pathway mainly explains that nitrite-cured meat favors cancer. Such a difference in mechanisms between red meat and processed meat could explain the suggested difference in potency for CRC between processed meat and red meat (Santarelli et al., 2008). However, this hypothesis needs to be further supported and other potential mechanisms also need to be further evaluated. For example, in hem-rich meat products, the carcinogenic potency of

HCAs and PAHs may play a role. Also, whether partly degraded protein fragments and/or peptides formed by protein digestion and/or microbial activity in the gastrointestinal tract could act as growth factors for cancer cells in the colorectum, or the importance of a presence of the microorganism per se, has to be further investigated.

9 Meat consumption data in the Nordic countries in relation to the WCRF recommendations

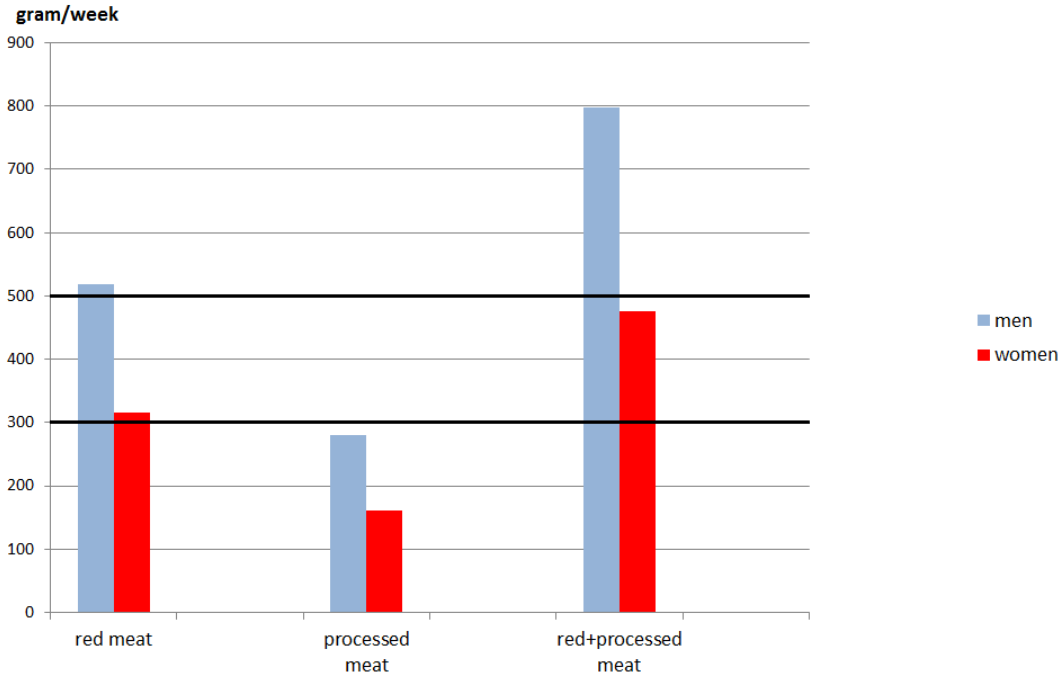

Figure 1. Estimated mean consumption of red and processed meat in the Swedish population, for men and women separately, based on the Swedish dietary survey Riksmaten 2010-11. Public health goal and personal recommendation of weekly intake of red meat (of which little or any to be processed), according to WCRF, are indicated in the figure, i.e. 300 g/week respectively 500 g/week.

By use of the latest Swedish food survey for the adult population (18-75 yrs.), Riksmaten 2010-11, the mean intake (obtained by food registration and questionnaire answers) of cooked red and processed meat by women was calculated to 315 g and 161 g per week, respectively, whereas the corresponding figures for men was considerably higher, i.e. 518 g and 280 g per week (TemaNord, 2013). This means that the total Swedish intake of red and processed meat, on a population basis, is at least twice as high as the WCRF recommendation, i.e. a public health goal of 300 g red meat per week, of which little or any to be processed (Fig. 1). According to the same calculations, the mean total meat consumption for women and men was 623 g and 959 g per week, respectively, also including consumption of white meat. There is of course a range in meat intake among women and men, and some consumers have a meat intake that will considerably exceed the calculated mean intake. For example, according to these calculations and assuming a normal

distribution of the meat consumption, roughly one half of the male consumers will have a consumption of red meat above 500 g per week, which is the recommended limit for red meat consumption set by the WCRF Report, on an individual basis (see Fig. 1). The report evaluated the different scenarios in which the mean meat consumption in the Nordic countries were adjusted to the levels suggested by the WCRF, and what nutritional consequences this would lead to. The general conclusion was that an adjustment to the WCFR recommendation has negligible nutritional consequences. These conclusions were similar for the different Nordic countries, except for Iceland.

The Swedish intake of meat could be calculated by the use of different data sources, and these could give somewhat different results depending on e.g. reporting bias, food losses at different stages, inclusion/exclusion of inedible carcass details, differences in calculation on meat contents in meat products, and weight differences in raw and cooked meat/ meat products (unpublished data). Nevertheless, in the presented Swedish food survey all data on consumption of red and processed meat are based on prepared products, i.e. weight given as consumed.These results clearly show that the mean total meat consumption in the Nordic countries, especially regarding male consumers, is considerably higher than what is recommended by WCRF.

10 Conclusion regarding red and processed meat in the Nordic Nutrition Recommendation

In the last revision of the Nordic Nutrition Recommendation (NNR5), one chapter addresses health effects of certain food that are characteristic of the Nordic diet, in the form of a systematic literature review (NNR, 2012). In this review, health effects were reviewed regarding potatoes, berries, whole grains, milk and milk products, and red and processed meat. In the case of red and processed meat, the review largely based its conclusion of the WCRF Report instead of, as was made for the other food groups, producing their own review statements. Thus, the review document concludes (based on WCRF) that red and processed meat is a convincing cause of colorectal cancer. However, no recommendation is presented regarding the amount of red and processed meat that can safely be consumed. In addition, the review states that there were too few studies to draw any conclusion regarding cardiovascular disease and consumption of red meat.

11 Information on restrictive dietary recommendations in EU/Europe

Some countries (Norway, Denmark and France) have implemented the WCRF advice in their National recommendations. In general, in these recommendations consumers are advised a weekly intake of not more than 500 g of red meat, sometimes including also processed meat, and to especially limit their intake of processed meat.
Norway: http://www.matportalen.no/matvaregrupper/tema/fjorfe_og_kjott/#tabs-1-2-anchor
Denmark: http://www.altomkost.dk/Anbefalinger/De_officielle_kostraad/Vaelg_magert_koed_og_koedpaalaeg/Vaelg_magert_koed_og_koedpaalaeg.htm
France: http://www.anses.fr/en/content/nutrition-and-cancer-recommendations ;
http://www.anses.fr/sites/default/files/documents/NUT-QR-NutritionCancerEN.pdf

There is also on-going activity in this area in Belgium where the WCRF Report is under evaluation. A preliminary evaluation has concluded that lowering the average consumption of red and processed meat from 95 to 50 g/day might save 1200 cases of CRC (15 % of total cases in Belgium), (info from the Belgian Superior Health Council).

12 Actions taken by the Commission and EFSA

The Panel on Dietetic Products, Nutrition and Allergies (NDA) at EFSA (European Food Safety Authority) has concluded that studies in the WCRF report show an association between intake of red and processed meat and CRC, but that the causality is questionable. Moreover, EFSA feels that it may be more of a life style effect and that it primarily may be a risk management issue (precautionary principle) due to insufficient but still alarming data.

13 General conclusions regarding the possible association between intake of red and processed meat and CRC

Studies described in the scientific literature, as a whole, give a strong, but not completely conclusive, evidence of an association between high intake of red and processed meat and development of CRC. This possible association is somewhat weakened by the fact that no single mechanism can explain how red and processed meat act in the development of CRC. However, the process of carcinogenesis may require several different factors acting in concert in a sequential way during initiation, promotion and progression. Potential factors and mechanisms for all these steps seem to have been identified in red and processed meat. According to the WCRF report, intake above 500 g/week of red meat increases the risk for CRC, whereas processed meat, due to its claimed higher CRC potency per weight basis, is regarded as a higher risk and intake should therefore be very little, if any. However, the variety of processed meat categories in the studies and a not conclusive evidence of the actual mechanisms behind the cause of CRC, by both red and processed meat, make it difficult to see the rationale for the great differences in the WCRF personal recommendations of intake amounts for red (500 g/week) compared to processed meat (avoid intake).

To conclude, it may be logical to recommend a restricted consumption of red meat, but questionable to recommend consumers to totally avoid processed meat. However, it has to be considered that scientific data indicate a higher CRC risk of processed meat compared to red meat on a weight basis and the fact that the Nordic intake of red and processed meat is considerably higher than the WCRF recommendation. Based on the WCRF Report and other scientific data our conclusion is to restrict the intake of red and processed meat, on a personal level, not to more than 500 g per week, and to particularly decrease the consumption of processed meat.

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Table 2a. Cancer incidence (colon, rectum) in relation to consumption of RED meat (prospective cohort studies evaluated in the WCRF 2007 Report; for reference data see Report)

Author, journal	Country, region	Participants (no., gender, age),	Specific aim of study	Time length of cancer reg. (yr)	No. of cancer cases	Confounding factors
Bostick et al. 1994, Cancer Causes Control	USA, Iowa	35 215 women, age 55-69 (1986)	Diet and colon cancer study	4 yr	Colon: 212	Considered
Chen et al. 1998, Cancer Res.	USA	22 971 men, age 40-84 (1982)	Physicians' Health Study –genotype and colorectal cancer	13 yr	Colon: 212	Considered (to some degree)
English et al. 2004, Cancer Epid. Biomarkers Prev.	Australia, Melbourne	37 112 men + women, age 27-75 (1990)	Meat and colorectal cancer study	9 yr	Colon: 283 Rectum: 169	Considered
Feskanich et al. 2004, Epidemiol. Biomarkers Prev.	USA	121 700 women, age 30-55 (at start 1976)	NHS – vitamin D and colorectal cancer risk	11-12 yr	Colorectal: 193	Considered
Fraser 1999, Clin. Nutr.	USA, California	34 182 mixed gender (approx 40% men), age ≥25yr (1976)	7th Day Adventists Cohort – diet and cancer study	6 yr	Colon: 107	?? (insufficiently described)
Giovannucci et al. 1994, Cancer Res.	USA	47 949 men, age 40-75 (1986)	Health professionals follow-up study	6 yr	Colon: 205	Considered
Jarvinen et al. 2001, Br. J. Cancer	Finland	27 340 men, 24 170 women, age unknown, start 1967	Finnish Health Examination Study	27-33 yr	Colon: 63 Rectum: 46	Considered
Kato et al. 1997, Nutr. Cancer	USA	14 727 women, age 34-65 (1986)	Womens health study	7.1 yr	Colon: 100	Considered
Larsson et al. 2005, Int. J. Cancer	Sweden, central part	61 433 women, age 40-75 (1987-1990)	Mammography cohort	13.9 yr	Colon, prox.: 234 Colon, dist.: 155 Rectum: 230	Considered

Author, journal	Country, region	Participants (no., gender, age),	Specific aim of study	Time length of cancer reg. (yr)	No. of cancer cases	Confounding factors
Norat et al. 2005, J. Natl. Cancer Inst.	Europe, 10 countries	478 040, approx. 2/3 women, 1/3 men (start 1992)	EPIC study on cancer and nutrition	4.8 yr	Colon: 1 329	Considered
Pietinen et al. 1999, Cancer Causes Control	Finland, SW part	27 111 men, age 50-69 (1987)	Betacarotene cancer prevention study	8 yr	Colon: 185	Considered
Phillips 1975, Cancer Res.	USA, California	12 250 men 23 210 women Age 35->85 (1958)	7th Day Adventists Cohort – diet and cancer study	8 yr	Missing data!	Not considered?
Tiemersma et al. 2002, Cancer Causes Control	Holland	>36 000 women, age 20-59 yr (men + women), start: 1987-1991	Cardiovascular disease factors (nested case-control study)	8.5 yr	Colorectal: 102	Considered
Sellers et al. 1998, Cancer Causes Control	USA, Iowa	35 216 women, age 55-59 (1986)	Diet and colon cancer among older women	9 yr	Colon: 241	Considered
Singh, Fraser 1998, Am. J. Epidemiol.	USA, California	34 198, both sexes, mean age 52-54 (at start 1976)	7th Day Adventist Cohort – diet and colon cancer	6 yr	Colon: 157 (of which 22 in rectosigmoid junction)	Considered
Wei et al. 2004, Int. J Cancer	USA, 11 states	NHS: 87 773, HPFS: 46 632 (women), age 30-75, start 1980	NHS and HPFS combined	NHS: 20 yr, HPFS: 14 yr	Colon: 1 139 Rectum: 339	Considered
Willet et al. 1990, N. Engl. J. Med	USA, 11 states	88 751 women, age 24-75 (1980)	NHS	6-7 yr	Colon: 150	Considered

Table 2b. Details of consumption, meat definitions, significant effects, and trends in relation to consumption of RED meat (prospective cohort studies evaluated in the WCRF 2007 Report; for reference data see Report)

Author, journal	Definition of red/processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments
Bostick et al. 1994, Cancer Causes Control	Beef, pork or lamb and main or mixed dish/sandwich	<4 - >11 servings/wk	No increased risk (colon cancer)	RR 1.13-1.04	p=0.78	Servings not defined by weight. Processed meat also studied
Chen et al. 1998, Cancer Res.	Beef, pork, or lamb as main or side dish/sandwich, and hot dogs (!)	<3.5 - >7 servings/wk	No increased risk, but trend (colorectal c.)	RR= 2.15 (0.95-4.86) (for >7 servings/wk	p=0.06	Narrow consumption range. Specific issue on polymorphism (acetylation)
English et al. 2004, Cancer Epid. Biomarkers Prev.	Beef and veal main dish; minced meats; mixed dishes with beef, pork or lamb; rabbit and game	<3 - >6.5 servings/wk	Increased risk (colorectal c., rectal c)	Colorectal RR =1.4 (1.0-1.9) Rectal RR= 2.0 (1.1-3.4)	Colorectal p=0.2 Rectal p=0.07	Servings not defined by weight Processed meat also studied.
Feskanich et al. 2004, Epidemiol. Biomarkers Prev.	Red meat not defined	Controls and cases, both 7.7 servings/wk	No increased risk (colorectal cancer)	No information	No information	Not designed to study colorectal cancer risk
Fraser 1999, Clin. Nutr.	Beef	0 – 2.98 servings/wk (veg. vs. nonvegetarians)	Increased risk (colon cancer)	RR=1.88 (1.24-1.87) (for ≥1 serving/wk)	p=0.0032	Data gaps re. intake, meat definitions, confounding

Author, journal	Definition of red/processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments
Giovannucci et al. 1994, Cancer Res.	Beef, pork or lamb as main or side dish/sandwich; hamburger, hotdog, preserved meats and bacon Also grouped: beef, pork or lamb as main dish	Red meat: 130-906 g/wk (median) Beef, etc: 0->5 servings/wk	Increased risk (colon c.; both red meat and beef, etc)	Red meat RR= 1.71 (1.15-2.55) at 906 g/wk Beef, etc RR= 2.11 and 3.57 at 2.4 resp. >5 servings/wk	p (red meat)=0.005 p(beef, etc.)=0.01	Red meat def. could be questioned (also processed meat). Beef, etc. servings not defined by wt. Processed meat also studied
Jarvinen et al. 2001, Br. J. Cancer	Definition: referring to separate paper	Men: <658 - >1 442 g/wk Women: <469 - >938 g/wk	No increased risk (colorectal, colon, rectum) “Nonsignificant trend” for meat and colorectal c.	Colorectal: RR=1.50 0.77—2.94) at highest consumption group	No data	Red meat definition not (yet) available High consumption figures
Kato et al. 1997, Nutr. Cancer	Not defined	Not given (grouping in quartiles)	No increased risk (colorectal c.)	RR= 1.23 (0.68-2.22)	p=0.545	Data gaps re. meat def. and consumption. Sign. neg. association protein intake - colorectal c.

Author, journal	Definition of red/processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments
Larsson et al. 2005, Int. J. Cancer	Whole beef, chopped or minced meat, bacon, lunch meat, blood pudding, kidney or liver, pate	<350 – 658 g/wk	Increased risk (colorectal, distal colon c.)	Colorectum RR=1.32 (1.03-1.68) Distal colon RR=2.22 (1.34-3.68) (highest intake group)	Colorectum p=0.23 Distal colon p=0.20 (highest intake group)	Rather narrow consumption range. Processed meat also studied
Norat et al. 2005, J. Natl. Cancer Inst.	Fresh, minced and frozen beef, veal, pork, and lamb.	<70 - >560 g/wk	No increased risk (but sign. before more extensive conf. adj.)	Colorectal c. RR=1.42 (1.09-1.86) at >560 /g/wk	p=0.02	Variability in national data Processed meat also studied
Pietinen et al. 1999, Cancer Causes Control	1)Beef, pork, and lamb' 2) Total red meat (no. 1 + processed meat)	1) 245 - 693 g/wk 2) 553 – 1 421 g/wk (medians)	No increased risk (colorectal cancer)	1) RR= 0.8 (0.5-1.2) 2)RR= 1.1 (0.7-1.8) at highest consumption group	1) p=0.74 2) p=0.73	Selected group Comp. high meat intake
Phillips 1975, Cancer Res.	1) Beef products 2) Beef hamburgers 3) Lamb	Any vs. none	Increased risk (colon c.)	1) RR=2.3 2) RR=2.5 3) RR=2.7	No data (?)	Selected group. No consumption figures. Only two consumpt. Categories
Tiemersma et al. 2002, Cancer Causes Control	Beef and pork	0 - >5 servings/wk	Increased risk, men (colorectal c.)	RR= 2.7 (>5 servings/wk)	p=0.06	Servings not defined by wt. Processed meat also studied

Author, journal	Definition of red/processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments
Sellers et al. 1998, Cancer Causes Control	Liver, hamburger, beef, beef stew, and venison	<3.5 - >7 servings/wk	No increased risk (colon c.)	RR=1.3 (0.8-1.9)/ 1.0 (0.5-2.1) for highest consumpt. (no family history/history)	p=0.3/1.0	Servings not defined by wt. Specific focus on family history. Processed meat also studied
Singh, Fraser 1998, Am. J. Epidemiol.	Beef and pork	0->1 servings/wk	Increased risk (colon c.). (Also for white meat and total meat)	RR=1.67 (1.11-2.51) for the "middle" cons. group	p=0.7	Servings not defined by wt. Narrow consumpt. range. No clear dose-effect; no significance in highest group
Wei et al. 2004, Int. J Cancer	Beef, pork, and lamb, as main dish	0 - >5 servings/wk	Increased risk (colon c.), borderline, in quintile 2 and 5 (in combined cohort)	MVRR=1.43 (1.00-2.05) in highest cons. group	p=0.25	Servings not defined by wt. Borderline significance, no dose-effect relationship. No sign. for rectal c.
Willet et al. 1990, N. Engl. J. Med	Beef, pork, or lamb, as main dish	"0" (< 1/mo) - >7 servings/wk	Increased risk (colon c.),	RR=2.49 (1.24-5.03) in highest cons. group	p=0.01	Servings not defined by wt. Nice trend

Table 3a. Cancer incidence (colon, rectum) in relation to consumption of PROCESSED meat (prospective cohort studies evaluated in the WCRF 2007 Report; for reference data see Report)

Author, journal	Country, region	Participants (no., gender, age),	Specific aim of study	Time length of cancer reg. (yr)	No. of cancer cases	Confounding factors
Bostick et al. 1994, Cancer Causes Control	USA, Iowa	35 215 women, age 55-69 (1986)	Diet and colon cancer study	4 yr	Colon: 212	Considered
Chao et al. 2005, JAMA	USA, 21 states	148 610 men + women, age 50-75 (1992/93)	Cancer Prevention study II - nutrition cohort	8-9 yr	Colon: 1 197 Rectum + r.-sigmoid junction: 470	Considered
English et al. 2004,Cancer Epid. Biomarkers Prev.	Australia, Melbourne	37 112 men + women, age 27-75 (1990)	Meat and colorectal cancer study	9 yr	Colon: 283 Rectum: 169	Considered
Flood et al. 2003, Am. J. Epidemiol.	USA	45 496 women, age <50 - ≥80 (mean 61.9)	Breast cancer screening, follow-up cohort	8.5 yr (mean)	Colorectal: 487	Considered
Giovannucci et al. 1994, Cancer Res.	USA	47 949 men, age 40-75 (1986)	Health professionals follow-up study	6 yr	Colon: 205	Considered
Goldbohm et al. 1994, Cancer Res.	Holland	120 852 men + women, age 55 – 69 (1986)	Netherlands Cohort Study	3.3 yr	Colon: 215 (105 men, 110 women)	Considered (shortages)
Kato et al. 1997, Nutr. Cancer	USA	14 727 women, age 34-65 (1986)	Womens health study	7.1 yr	Colon: 100	Considered
Khan et al. 2004, Asian Pacific J. Cancer	Japan, Hokkaido	1 524 men + 1 634 women, age >40 (start 1984)	Broad screening of dietary factors and cancer	18 yr	Colorectal: 15 men + 14 women	Not considered

Author, journal	Country, region	Participants (no., gender, age),	Specific aim of study	Time length of cancer reg. (yr)	No. of cancer cases	Confounding factors
Larsson et al. 2005, Int. J. Cancer	Sweden, central part	61 433 women, age 40-75 (1987-1990)	Mammography cohort	13.9 yr	Colon, prox.: 234 Colon, dist.: 155 Rectum: 230	Considered
Norat et al. 2005, J. Natl. Cancer Inst.	Europe, 10 countries	478 040, approx. 2/3 women, 1/3 men (start 1992)	EPIC study on cancer and nutrition	4.8 yr	Colon: 1 329	Considered
Pietinen et al. 1999, Cancer Causes Control	Finland, SW part	27 111 men, age 50-69 (1987)	Betacarotene cancer prevention study	8 yr	Colon: 185	Considered
Tiemersma et al. 2002, Cancer Causes Control	Holland	>36 000 women, age 20-59 yr (men + women), start: 1987-1991	Cardiovascular disease factors (nested case-control study)	8.5 yr	Coloractal: 102	Considered
Wei et al. 2004, Int. J Cancer	USA, 11 states	NHS: 87 773, HPFS: 46 632 (women), age 30-75, start 1980	NHS and HPFS combined	NHS: 20 yr, HPFS: 14 yr	Colon: 1 139 Rectum: 339	Considered
Willet et al. 1990, N. Engl. J. Med	USA, 11 states	88 751 women, age 24-75 (1980)	NHS	6-7 yr	Colon: 150	Considered

Table 3b. Details of consumption, meat definitions, significant effects, and trends in relation to consumption of PROCESSED meat (prospective cohort studies evaluated in the WCRF 2007 Report; for reference data see Report)

Author, journal	Definition of red/ processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments (rating, 0-3)
Bostick et al. 1994, Cancer Causes Control	Bacon, hotdogs, other proc. meats (sausage, salami, bologna etc)	O - >3 servings/wk	No increased risk (colon cancer)	RR=1.51 (>3 servings/wk)	p=0.45	Servings not defined Also red meat studied
Chao et al. 2005, JAMA	Ham, lunch meat, hot dogs, salami etc.	0 – >240 g/wk	Increased risk (colon cancer) significant in combined studies	RR=1.50 ("high intake")	No data	Intake categories not defined (**?**) Also red meat studied
English et al. 2004, Cancer Epid. Biomarkers Prev.	Salami, sausage, bacon, ham, lunch meat	0 – 3.9 servings/wk	Increased risk (rectal cancer)	RR=2.0 (2 - 3.9 serv./wk)	p=0.09 (similar trend for colon cancer)	Servings not defined Also red meat studied
Flood et al. 2003, Am. J. Epidemiol.	Bacon, ham/lunch meat, hot dogs, sausage	0.2 – 186 g/wk	No increased risk (colorectal cancer)	RR=1.0	p=0.22	Also red meat studied
Giovannucci et al. 1994, Cancer Res.	Hot dogs, sausage, salami, bologna, bacon	0 -5 servings/wk	Increased risk (colon cancer)	RR=1.67 (2-4 serv./wk)	p=0.06	No sign. effect at highest intake Also red meat studied
Goldbohm et al. 1994, Cancer Res.	Mainly sausages	0 - >140g/wk	Increaed risk (colon cancer) sexes combined	RR=1.72 (>140 g/wk)	p=0.02	No sign. effect in separate sexes

Author, journal	Definition of red/ processed meat	Consumption data	Conclusion re. cancer risk (type)	Sign. effect; at what intake?	Trends	Comments (rating, 0-3)
Kato et al. 1997, Nutr. Cancer	Sausage, ham	Dietary intake in four quartiles	No increased risk (colorectal cancer)	RR=1.39-1.09	p=0.74	Badly described study Selection of proc. meat evaluated
Khan et al. 2004, Asian Pacific J. Cancer	Sausage, ham	Missing	No increased risk (colorectal cancer)	RR=0.5 (0.1-2.2)	No data	Badly described study No intake data
Larsson et al. 2005, Int. J. Cancer	Bacon, sausage, ham, lunch meat, blood pudding	<84 – 234 g/wk	No increased risk (colorectal cancer; also for rectal c.)	RR=1.07 (>234 g/wk) colorectum combined	p=0.23	Diff. cancer forms specifically analysed
Norat et al. 2005, J. Natl. Cancer Inst.	Sausage, meat cuts, liver pate, tinned meat	>70 - ≥560 g/wk	Increased risk (colorectal cancer)	HR=1.42 (highest intake)	p=0.02	Variability in national data
Pietinen et al. 1999, Cancer Causes Control	Mainly sausage	182 – 854 g/wk (median)	No increased risk (colorectal cancer)	RR=1.2	p=0.73	Selected group High but selected intake of proc. meat
Tiemersma et al. 2002, Cancer Causes Control	Meat snacks, sausage	188 g/wk (controls) 192 g/wk (cases)	No increased risk (colorectal cancer)	RR=0.9	No data	Badly descr. selection of proc. meat. Intake not sufficiently described
Wei et al. 2004, Int. J Cancer	Not specified (ref. to other studies)	0 - >5servings/wk	Increased risk (colon cancer)	MVRR=1.33	p=0.008	Increased risk by add. servings
Willet et al. 1990, N. Engl. J. Med	Not specified (ref. to other studies)	0 - ≥7 servings/wk	Increased risk (colon cancer)	RR=1.86 (2-4 serv./wk)	p=0.04	No dose-effects relationship re. colon cancer risk

Table 4. Compilation of cohort and review papers with focus on CRC and red/processed meat, retrieved from 2005 and onwards (not included in the WCRF 2007 Report)

No. (ref list)	Authors, year	Type of study	Region/country	Cancer cases	Result	Comments
Cohort/nested case-control studies						
1	McCullough et al., 2013	Cohort study	The Cancer Prevention Study II Cohort	Cases: 2315 CRC diagnosis, 966 died during follow-up	Red and processed meat associated with higher mortality risk among patients with CRC	
2	Egeberg et al., 2013	Cohort study	Danish Diet, Cancer and Health Cohort	Cases: 644 colon c., 345 rectal c.; 53 988 patients	No association between intake of red or processed meat and colon/rectal c.	Substitution of fish for red meat reduced risk
3	Parr et al., 2013	Cohort study	The Norwegian Women and Cancer Cohort study	Cases: 459 colon and 215 rectal c.; 84 538 women part.	Processed meat significantly increased cancer in both colon and rectum	No association with red meat
4	Zhu et al., 2013	Cohort study	Follow-up CRC patients Newfoundland	Cases: 529 diagnosed patients, follow-up ca, 10 yr	Processed meat dietary pattern associated with higher risk of tumour recurrence and death in CRC patients	
5	Ollberding et al., 2012	Cohort study	The Multiethnic Cohort Study	Cases: 3 404 CRC, 165 717 part.	Results do not support a role for meat in the etiology of CRC	Heterocyclic amines studied: also negative findings
6	Takaichi et al., 2011	Cohort study	Japanese cohort	Cases: 1 145 CRC; 80 658 part.	Association red meat and colon cancer, women; total meat and colon cancer men	Processed meat gave no significant associations to colon or rectal cancer risk

No. (ref list)	Authors, year	Type of study	Region/country	Cancer cases	Result	Comments
Cohort/nested case-control studies						
7	Daniels et al., 2011	Case-cohort -nested case-contr	U.S. cohort	74 418 cancer cases; 492 186 part.	Substitution red meat with white meat: 3-20% reduction in cancer risk (many forms incl CRC)	Simply increasing fish/poulty intake, without reducing red meat, less beneficial
8	Cross et al., 2010	Cohort study	U.S. prospective cohort	Cases: 2 719 CRC; 300 948 part.	Positive association was found for red and processed meat and colorectal cancer	Heme iron, nitrat/nitrite, and heterocyclic amines may explain the associations
9	Andersen et al., 2009	Case-cohort -nested case-contr	Denmark - Diet, cancer and health cohort	364 cases (CRC) 772 controls	No direct result on association meat-CRC	Polymorphism study – MDR1 effective modulating meat-CRC association
10	Wei et al., 2009	Cohort study	USA - Nurses' Health Study	701 cases (CRC); 83767 particip.	Red plus processed meat among risk factors for CRC (OR 1,20 ej sign)	
11	Nötlings et al., 2009	Case-cohort -nested case-contr	Hawaii, California, USA (multiethnic)	1009 cases (CRC), 1522 controls	No significant associations	Polymorphism study (NAT2 genotype and meat certain assoc.)
12	Morita et al., 2009 (abstract!)	Case-cohort -nested case-contr	Japan – Fukuoka CRC Study	685 cases (CRC), 778 controls	Association red meat – colon c. only for certain genotype (CYP2E1)	Polymorphism study (CYPE21)
13	Flood et al., 2008	Cohort study	USA – NIH AARP cohort	Cases: 2151 men, 959 women (ca 500 000 partic)	High red meat scores assoc with CRC risk: RR men 1.17, women 1.48 (both sex sign.)	Dietary patterns identified by factor analysis
14	Sörensen et al., 2008	Case-cohort -nested case-contr	Denmark – Diet, cancer and health cohort	379 cases, 769 controls ("subcohort memb.")	No stat. sign. association between red, processed or fried meat and CRC	Polymorphism study (NAT1 and 2)

No. (ref list)	Authors, year	Type of study	Region/country	Cancer cases	Result	Comments
Cohort/nested case-control studies						
15	Wu et al., 2006	Cohort study	USA – HPFS cohort (men)	581 cases, 51 129 participants	Association between colon adenoma and meat-derived mutagenicity (OR=1.3-1.4). Red meat, processed meat intake??	Meat mutagen focus
16	Balder et al., 2006	Cohort study	Netherlands – the Netherl. Cohort Study	Cases: 869 men, 666 women (120 852 part.)	No associations between fresh meat and CRC cancer (but assoc. with heme iron)	Heme and chlorophyll focus
17	Luchtenborg et al., 2005	Case-cohort -nested case-contr	Netherlands – the Netherl. Cohort Study	Cases: 434 colon, 154 rectal; sub-cohort 2948	Total meat consumption not associated to CRC; meat subgroups associated to CRC forms, at certain genotype conditions	Genotype conditions for meat – CRC associations
Reviews						
1	Hjartåker et al., 2013	Review (32 prospective cohort studies)	(Norway)		Meat gave stronger association to distal colon and rectal cancer, compared to proximal colon cancer	
2	Aune et al., 2013	Review (seven prospective and 19 case-control studies incl.)	(England)		Results indicate elevated risk of CR adenomas with intake of red and processed meat	

No. (ref list)	Authors, year	Type of study	Region/country	Cancer cases	Result	Comments
Reviews						
3	Rosato et al., 2013	Review (three Italian and Swiss case-control studies)	(Italy, Switzerland)		Processed meat increases CRC risk, also in young-onset patients	Familial history of CRC is a particularly strong risk factor in young subjects
4	Yusof et al., 2012	Review (six cohort studies)	(Malaysia)		Elevated risk for CRC development for Western dietary pattern (red and processed meat, refined grains)	
5	Xu et al., 2013	Meta-analysis (five cohort/nested case-control and 16 case-control studies)	(China)		Increased intake of red and processed meat is associated with significantly increased risk of CR adenomas	
6	Magalhaes et al., 2012	Review (Eight cohort and eight case-control studies)	(Portugal)		Colon cancer increased with high red and processed meat intake patterns	No significant associations were observed for rectal cancer
7	Spencer et al., 2010	Pooled data analysis	(England)		Little evidence of association between consumption of red and processed meat and CRC risk	

No. (ref list)	Authors, year	Type of study	Region/country	Cancer cases	Result	Comments
Reviews						
8	Miller at al., 2010	Review (CRC risk coupled to food-based dietary patterns)	(USA)		Higher intakes of red and processed meat, as well as potatoes and carbohydrates, may increase CRC risk	
9	McAfee et al., 2010	Review			Moderate consumption of lean red meat is unlikely to increase colon cancer risk.	
10	Bosetti et al., 2009	Review	(Italy)		High red meat intake related to some common neoplasms	
11	Huxley et al., 2009	Review	(Australia)		High meat intake is associated with a sign. 20% incr. risk of CRC	Data from 103 cohorts included
12	Ryan-Harshman and Aldoori, 2007	Review	(Canada)		CRC and red meat assoc. still not confirmed, any effect is likely moderate and related to processing/ cooking	Calcium and vitamin D reduce risk
13	Reszka et al., 2006	Review	(Poland)		CRC risk only discussed in connection to genetic polymorphism (difficult to see the data of meat and CRC association)	Genetic polymorphism review
14	Campos et al., 2005	Review	(Brasil)		Red and processed meat are implicated in CRC risk	Discusses many components of Western diet

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1. Contaminants and minerals in foods for infants and young children – analytical results, Part 1, by V Öhrvik, J Engman, B Kollander and B Sundström.

   Contaminants and minerals in foods for infants and young children – risk and benefit assessment, Part 2 by G Concha, H Eneroth, H Hallström and S Sand.

   Tungmetaller och mineraler i livsmedel för spädbarn och småbarn. Del 3 Risk- och nyttohantering av R Bjerselius, E Halldin Ankarberg, A Jansson, I Lindeberg, J Sanner Färnstrand och C Wanhainen.

   Contaminants and minerals in foods for infants and young children – risk and benefit management, Part 3 by R Bjerselius, E Halldin Ankarberg, A Jansson, I Lindeberg, J Sanner Färnstrand and C Wanhainen.
2. Bedömning och dokumentation av näringsriktiga skolluncher – hanteringsrapport av A-K Quetel.
3. Gluten i maltdrycker av Y Sjögren och M Hallgren.
4. Kontroll av bekämpningsmedelsrester i livsmedel 2010 av A Wannberg, A Jansson och B-G Ericsson.
5. Proficiency Testing – Food Microbiology, January 2013 by L Nachin, C Normark and I Boriak.
6. Från jord till bord – risk- och sårbarhetsanalys. Rapport från nationellt seminarium i Stockholm november 2012.
7. Cryptosporidium i dricksvatten – riskvärdering av R Lundqvist, M Egervärn och T Lindberg.
8. Proficiency Testing – Food Microbiology, April 2013 av L Nachin, C Normark, I Boriak and I Tillander.
9. Proficiency Testing – Drinking Water Microbiology, March 2013 by T Šlapokas and K Mykkänen.
10. Grönsaker och rotfrukter – analys av näringsämnen av M Pearson, J Engman, B Rundberg, A von Malmborg, S Wretling och V Öhrvik. 11. Riskvärdering av perfluorerade alkylsyror i livsmedel och dricksvatten av A Glynn, T Cantilana och H Bjermo.
12. Kommuners och Livsmedelsverkets rapportering av livsmedelskontrollen 2012 av L Eskilsson.
13. Kontroll av restsubstanser i levande djur och animaliska livsmedel. Resultat 2011 av I Nordlander, B Aspenström-Fagerlund, A Glynn, I Nilsson, A Törnkvist, A Johansson, T Cantillana, K Neil Persson Livsmedelsverket och K Girma, Jordbruksverket.
14. Norovirus i frysta hallon – riskhantering och vetenskapligt underlag av C Lantz, R Bjerselius, M Lindblad och M Simonsson.
15. Riksprojekt 2012 – Uppföljning av de svensk salmonellagarantierna vid införsel av kött från nöt, gris och fjäderfä samt hönsägg från andra EU-länder av A Brådenmark, Å Kjellgren och M Lindblad.
16. Trends in Cadmium and Certain Other Metal in Swedish Household Wheat and Rye Flours 1983-2009 by L Jorhem, B Sundström and J Engman.
17. Miljöpåverkan från animalieprodukter – kött, mjölk och ägg av M Wallman, M Berglund och C Cederberg, SIK.
18. Matlagningsfettets och bordsfettets betydelse för kostens fettkvalitet och vitamin D-innehåll av A Svensson, E Warensjö Lemming, E Amcoff, C Nälsén och A K Lindroos.
19. Mikrobiologiska risker vid dricksvattendistribution – översikt av händelser, driftstörningar, problem och rutiner av M Säve-Söderbergh, A Malm, R Dryselius och J Toljander.
20. Mikrobiologiska dricksvattenrisker. Behovsanalys för svensk dricksvattenförsörjning – sammanställning av intervjuer och workshop av M Säve-Söderbergh, R Dryselius, M Simonsson och J Toljander.
21. Risk and Benefit Assessment of Herring and Salmonid Fish from the Baltic Sea Area by A Glynn, S Sand and W Becker.
22. Synen på bra matvanor och kostråd – en utvärdering av Livsmedelsverkets råd av H Enghardt Barbieri.
23. Revision av Sveriges livsmedelskontroll 2012 – resultat av länsstyrelsernas och Livsmedelsverkets revisioner av kontrollmyndighete av A Rydin, G Engström och Å Eneroth.
24. Kött – analys av näringsämnen: hjort, lamm, nötdjur, ren, rådjur, vildsvin och kalkon av V Öhrvik.
25. Akrylamid i svenska livsmedel – en riktad undersökning 2011 och 2012 av Av K-E Hellenäs, P Fohgelberg, U Fäger, L Busk, L Abramsson Zetterberg, C Ionescu, J Sanner Färnstrand.
26. Proficiency Testing – Food Microbiology, October 2013 av L Nachin, C Normark and I Boriak.
27. Proficiency Testing – Drinking Water Microbiology, September 2013 by T Šlapokas and K Mykkänen.
28. Sammanställning av analysresultat 2008-2013. Halt av polycykliska aromatiska kolväten (PAH) i livsmedel – matfetter, spannmålsprodukter, kosttillskott, choklad, grillat kött och grönsaker av S Wretling, A Eriksson och L Abramsson Zetterberg.

Rapporter som utgivits 2014

1. Exponeringsuppskattningar av kemiska ämnen och mikrobiologiska agens – översikt samt rekommendationer om arbetsgång och strategi av S Sand, H Eneroth, B-G Ericsson och M Lindblad.
2. Fusariumsvampar och dess toxiner i svenskodlad vete och havre – rapport från kartläggningsstudie 2009-2011 av E Fredlund och M Lindblad.
3. Colorectal cancer-incidence in relation to consumption of red and processed meat by P O Darnerud and N-G Ilbäck.

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