Prevention of mutagen formation in heated meats and model systems

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Possible means for preventing mutagen formation in cooked meats and in heated model systems are described. One way to reduce mutagenicity in cooked meats is to control cooking temperature, time and method. Another way is to increase water content or to avoid loss of water in meats during cooking. Addition of an excessive amount of reducing sugars to meats before cooking is effective in minimizing mutagen formation, which may be due to suppression of generation of the pyrazine cation radical. Addition of a small amount of ascorbate or erythorbate is also effective, which may be the result of scavenging the intermediary pyrazine cation radical.

Introduction

Heterocyclic amine (HCA) mutagens are produced in a wide variety of cooked or processed meats and fish and are known to be carcinogenic in rodents and considered to be probable human carcinogens (Sugimura and Sato, 1983; Layton et al., 1995; Skog et al., 1998). Epidemiological studies have demonstrated that the risks of colorectal adenoma (Sinha et al., 1999, 2001) and lung cancer (Sinha et al., 2000) are increased by the intake of well-done or grilled meats containing HCA mutagens. It is an important objective to reduce or minimize mutagen formation due to HCAs in cooked or processed meats and fish. Here, possible means for preventing or minimizing the mutagenicity of cooked meats due to HCAs are briefly reviewed.

Mechanisms of mutagen formation in heat-dried meats and model systems

It was found that addition of sugars and creatine/creatinine to ground beef increased the mutagenicity of cooked beef and that the precursors of HCAs were sugars, amino acids and creatine/creatinine (Jägerstedt et al., 1983). The precursors of HCA mutagens in meats have been extensively studied (Jägerstad et al., 1984; Negishi et al., 1984; Reutersward et al., 1987; Skog et al., 1998). A dramatic increase in mutagenicity was observed on adding creatine to a recipe combining pork, beef, veal, starch and milk (Nes, 1986; Becher et al., 1988).

The heating temperature was shown to be important as an increase in temperature had a marked effect on overall mutagenicity of foods (Knize et al., 1985; Felton and Knize, 1991).

2-Amino-3,8-dimethylimidazo[4,5-f]quinocarcinone (MeIQx) is a common mutagenic HCA found in a wide variety of fried or heated meats (Negishi et al., 1991) and fish (Kikugawa and Kato, 1987b). Creatine or one of 15 amino acids was mixed with minced pork before broiling at 200°C (Overvik et al., 1989). Addition of 5% (w/w) creatine increased the overall mutagenicity of crust, pan residue and aerosol 4-fold. Amino acid addition (1% w/w) increased the overall mutagenicity between 1.5 (lysine) and 43 times (threonine). In most cases the mutagenicity profiles on reversed phase HPLC separation of the crust and pan residues were changed by amino acid addition. Serine, threonine, phenylalanine, alanine, leucine and tyro-

iment were shown to be present in dry-heated meat juice. It was concluded that creatine and free amino acids were the main reactants in the mutagen-forming reactions that occur during frying of meat.

Mixtures of glucose, amino acids and creatinine simulating the composition of six different kind of meats (beef, chicken breast, chicken thigh, turkey breast, pork and fish) were dry-heated to simulate the formation of HCAs in meat. The presence of 16 HCAs was investigated in the model systems and in the six meats and their corresponding meat drippings to determine the importance of meat composition to HCA formation (Pais et al., 1999). Nine HCAs, 2-amino-3-methylimidazo[4,5-f]quinoline (IQ), 2-amino-3,4-dimethyl-imidazo[4,5-f]quinoline (MeIQx), 4,8-DiMeIQx, PhIP, 2-amino-3-methylimidazo[4,5-f]quinocarcinone (IQx), 2-amino-

1,6-dimethylfuro[3,2-e]imidazo[4,5-b]pyridine (IFP), 2-amino-1,6-dimethylimidazo[4,5-b]pyridine (DMIP) and DMIP were found to be present at concentrations ≥0.1 ng/g in some of the model systems and in some of the meats of pan residues. HCA concentrations were affected by precursor composition in the model system and the same nine HCAs formed in the meat and in the model system, showing that the model system is a good surrogate for the reaction conditions occurring in meats during cooking.

A mixture of glucose, glycine and creatinine in diethyleneeglycol–water generated imidazoquinocarcinone-type HCAs, including MeIQx. A pathway including condensation of the pyrazine cation radical (Namiki and Hayashi, 1975) generated in the Maillard reaction of sugar/amino acid with creatine/creatinine has been suggested for HCA formation (Pearson et al., 1992; Milic et al., 1993) and participation of the pyrazine cation radical (Figure 1) in the formation of imidazoquinocarcinone-type HCAs has been demonstrated (Kato

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Minimizing mutagen formation in cooked meats by controlling heating temperature and time

Heating temperature, heating time and heating method greatly affected HCA formation in the model systems and in cooked meats. Earlier studies showed that HCA formation was controlled in part by surface temperature and the length of time at a high temperature was maintained (Spingarn et al., 1980). One method of lowering the surface temperature was to mix ground meat with soy proteins or with other components that retain water. Thus, satisfactory cooking of an item, such as a soyburger, could be achieved at a lower maximum temperature and, therefore, with decreased production of HCAs (Wang et al., 1982). Mutagenicity in lean pork meat fried at 250°C was increased by the addition of fat, probably due to more efficient heat transfer from the frying pan to the meat (Nilsson et al., 1986). The effect of cooking time on the mutagenicity of the crust, pan residue and smoke from pan-broiled pork patties was studied (Berg et al., 1990). Meat was broiled at 200°C for various times between 2 and 10 min. Broiled meat was reheated up to 5 times at 200°C, each time to a centre temperature of 70°C. Reheating was performed in a microwave oven for 2 min and in an electric oven at 200°C for 10 min. In addition, broiled patties were kept warm at 60°C in an incubator for up to 9 h. Mutagenicity increased rapidly in all fractions except the volatile phase over the first 6 min of cooking, after which time only a slight increase was seen. At cooking times <4 min no mutagenicity was detected in the smoke. Reheating or keeping the meat warm had very little effect on the mutagenicity of the meat. Hence, during pan broiling at 200°C the major part of the mutagenicity was formed during the first 6 min of cooking. Reheating the meat or keeping it warm did not significantly affect the mutagenicity.

Microwaving meat for 1–1.5 min separated the juice appearing at the bottom of the dish from the meat itself. When the meat was cooked the resulting mutagenicity was ~50-fold lower than that in meat cooked without microwaving. When the juice was added back to the meat and recooked, the mutagenicity was restored (Taylor et al., 1986). This reduction in mutagenicity may be due to removal of water-soluble HCA precursors in the juice on microwaving. Pretreatment of beef patties by microwaving reduced the mutagenicity of cooked hamburgers, which may be due to the loss of HCA precursors (Felton et al., 1994). The beef patties received microwave treatment for various times before frying. Microwave pretreatment for 0, 1, 1.5, 2 or 3 min before frying at either 200 or 250°C for 6 min per side reduced HCA precursors (creatinine, creatinine, amino acids and glucose), water and fat by up to 30% and resulted in a decrease in mutagenicity of up to 95%. The sum of four HCAs, MeIQx, IQ, 4,8-DiMeIQx and PhiP, decreased 3- to 9-fold compared with non-microwaved beef patties fried under identical conditions. Hence, microwaving meat is effective in reducing the mutagenicity of cooked meat. However, much of the taste of the meat may be removed by this procedure.

Turning over beef patties repeatedly at lower temperatures reduced the mutagenicity of cooked hamburgers (Salmon et al., 2000).

Chemical analysis of foods has shown that flame grilling can form both polycyclic aromatic hydrocarbon and HCA mutagens and that frying forms predominantly HCA mutagens (Knize et al., 1999). Pan-roasted beef showed lower mutagenicity after various degrees of cooking than beef cooked over charcoal. The high mutagenicity of barbecued beef was due to the formation of more HCAs because of rapid and direct heating of the surface of the meat to a high temperature (Gu et al., 2001). Seasoning decreased the mutagenicity of pan-roasted beef but increased the mutagenicity of barbecued beef with decreased kinds of HCAs, probably due to a change in HCA precursors or, alternatively, to the formation of other mutagens (Gu et al., 2001). Seasoning was also reported to increase mutagenic activity in chicken and hamburger patties charcoaled at 220°C (Tikkonen et al., 1996).

These studies demonstrate that understanding the cooking or processing conditions that lead to the formation of mutagens can give rise to methods to greatly reduce their occurrence in cooked or processed foods.

Avoiding loss of water minimizes mutagenicity of heated meats and model systems

A high water content in beef extract prevents mutagen formation (Taylor et al., 1986). Decreasing the water content of the model system and loss of water from fish increased the mutagenicity after heating (Kikugawa and Kato, 1987a). When mixtures of glucose, glycine and creatinine in diethyleneglycol–water or dioxane–water with varying water contents were heated in sealed tubes at 120°C for 2 h, the overall mutagenicity produced in the mixtures decreased as the water content increased (Figure 2), indicating that a higher water content in the model systems suppressed the formation of mutagenic compounds. Decreased mutagenicity with a higher water content may be due to disappearance of the intermediary pyrazine cation radical, because heating of glucose and glycine in water alone did not give the appropriate ESR signals whereas heating glucose and glycine in diethyleneglycol–water gave intense signals for the radical (Kato et al., 1996). Bonito,
tunny and mackerel gradually generated mutagenicity during heating at 100–110°C in air over a period of ~50 h, which was mainly due to MeIQx formation (Kikugawa and Kato, 1987a). Loss in weight and mutagenicity were followed over time under these conditions (Figure 3). The mutagenicity of fish increased after >60% weight loss, indicating that loss of water is a driver for the decrease in mutagenicity. The development of overall mutagenicity in the fish heated at 100–110°C for 24 h under air and steam was compared (Table I). Loss of weight of each fish on heating under steam was one-tenth that on heating in air and the overall mutagenicity of each meat heated under steam was less than one-fifth of that heated in air. These results indicate that formation of mutagenicity in fish depends not only on heating temperature and time but also on loss of water. Hence, avoiding loss of water during cooking is effective in minimizing the mutagenicity of meats. Boiling meats does not induce mutagenicity and roasting or frying meats under conditions of lowered water loss can minimize mutagenicity.

Controlling sugar content reduces mutagenicity of heated meats and model systems

It has been shown that the addition of reducing sugars such as glucose and fructose is effective in inhibiting HCA formation in cooked beef patties, resulting in a reduction in mutagenicity (Skog and Jägerstad, 1990, 1991; Skog et al., 1992). Although sugar is viewed as a major contributor to HCA formation, the addition of sugars to ground beef patties at levels ranging from 2 to 4% reduced HCA formation and overall mutagenicity of cooked ground meat. It has been thought that the addition of reducing sugars to meat beyond the optimum needed for the formation of HCAs results in the formation of Maillard reaction products that inhibit HCA formation.

The present author investigated whether sugar content affects generation of the intermediary pyrazine cation radical in the early stages of the Maillard reaction and the formation of imidazoquinazoline-type HCAs. When mixtures of 0-0.8 M glucose and 0.8 M glycine in polyethylene glycol–water were heated at 120°C for 5 min the intensities of the pyrazine cation radical ESR signals were highest in the mixture with glucose at 0.4 M (Figure 4) (Kikugawa et al., 1999). When mixtures of 0.025–0.8 M glucose, 0.4 M glycine and 0.4 M creatinine in diethylene glycol–water were heated at 120°C for 2 h, the mixture with glucose at 0.4 M gave the highest overall mutagenicity (Figure 5) (Kato et al., 1998). Hence, both pyrazine cation radical generation in mixtures of glucose and glycine and overall mutagenicity formation in mixtures of glucose, glycine and creatinine depend on the glucose concentration. The optimal glucose:glycine mixture was 1:2, suggesting that mutagen formation was reduced at higher glucose ratios.

The effect of adding reducing sugars to ground beef on the overall mutagenicity of cooked hamburgers was also examined (Figure 6) (Kato et al., 2000b). The initial reducing sugar content of ground beef was 0.07% (w/w). Addition of 0.08% (w/w) glucose to ground beef (total glucose content 0.15% w/w) increased the mutagenicity ~2-fold, but addition of >0.67% (w/w) glucose to ground beef (total glucose content 0.74% w/w) decreased the mutagenicity by 50% (Figure 6A). Addition of other reducing sugars, such as fructose and lactose, to ground beef gave rise to similar sugar-dependent mutagen formation (Figure 6B and C). However, addition of a...
It was found that addition of foods that contain reducing sugars to ground beef affects the mutagenicity of cooked hamburgers. Addition of wine containing reducing sugars to ground beef increased the mutagenicity of the cooked hamburger (Kato et al., 2000b). When a brand of red wine containing reducing sugars at 0.10% (w/v) or a brand of white wine containing reducing sugars at 0.13% (w/v) was added to ground beef at 33% (v/w), the total reducing sugar content of the ground beef was close to the optimum for the mutagen formation and mutagenicity of the cooked hamburger increased 2-fold. Addition of brandy (containing no sugars) to ground beef had no effect on mutagen formation in cooked hamburgers.

In Japan, as elsewhere, onion is added to ground beef when preparing homemade hamburgers. This cooking style was found to be a beneficial way to reduce hamburger mutagenicity (Kato et al., 1998). Onions obtained in Japan contain ~7.6% (w/w) reducing sugars and addition of 33% (v/w) onion to ground beef raised the sugar content well above the optimal ratio. The resulting mutagenicity of the cooked hamburger, estimated on the basis of weight of ground beef, was about one-fifth of that cooked without onion.

Addition of a large amount of corn starch to ground beef (1.5% w/w) also reduced the overall mutagenicity of cooked hamburgers (Kato et al., 1998). The effects of different oligosaccharides, fructooligosaccharides, galactooligosaccharides and isomaltooligosaccharides and inulin on HCA formation and overall mutagenicity in fried ground patties were evaluated (Shin et al., 2003a). The addition of a large amount of saccharides to ground beef (1.5%, w/w) inhibited total HCA formation by 46–54%. It also reduced overall mutagenicity by 48–59%. The studies confirmed that saccharides have the potential to reduce HCA formation in cooked beef patties, although the reasons for this effect are not known.

**Table I. Effect of heating conditions on mutagen formation in fish**

<table>
<thead>
<tr>
<th>Meat</th>
<th>Heated at 100–110°C for 24 h in air</th>
<th>Heated at 110°C for 24 h in steam</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Weight loss (%)</td>
<td>His&lt;sup&gt;+&lt;/sup&gt; revertants (per g)</td>
</tr>
<tr>
<td>Bonito</td>
<td>69.6</td>
<td>612</td>
</tr>
<tr>
<td>Tunny</td>
<td>73.3</td>
<td>391</td>
</tr>
<tr>
<td>Mackerel</td>
<td>65.1</td>
<td>227</td>
</tr>
</tbody>
</table>

A piece of each meat (40–50 g) was heated in a stainless steel box in air at 100–110°C or in an autoclave under steam at 100°C for 24 h. The mutagens were extracted by the blue cotton method and mutagenicity (His<sup>+</sup> revertants) was assayed on Salmonella typhimurium TA98 with S9 mix. The mutagenicity is presented on the basis of weight dried at 120°C for 2 h (Kikugawa and Kato, 1987a).

**Fig. 4.** ESR spectra of a mixture of 0–0.8 M glucose and 0.4 M glycine in polyethylene glycol–water (6:4 v/v) heated at 120°C for 5 min (Kikugawa et al., 1999).

Non-reducing sugar, sucrose, at the concentrations studied did not affect mutagen formation (Figure 6D). Hence, a glucose content in ground beef of >0.7% (w/w) can reduce the overall mutagenicity of cooked hamburgers.

**Antioxidants and reductones reduce the mutagenicity of heated meats and model systems**

Food additives that minimize the mutagenicity of model systems and cooked meats have been investigated. Addition of tryptophan, indole derivatives and proline (Jones and Weisburger, 1988a,b,c) to the model system or to beef reduced mutagen formation. The mechanism for this inhibition has not yet been elucidated. Phenolic antioxidants, butylated hydroxyanisole (BHA) and chlorogenic acid (Wang et al., 1982; Weisburger, 1991) and EDTA (Barnes and Weisburger, 1984) prevent mutagen formation in beef, while ferrous ion enhances formation (Barnes and Weisburger, 1984). A contribution of lipid peroxidation to the mutagen formation has been suggested and prevention of lipid peroxidation by antioxidants was thought to be effective in reducing mutagen formation. Defatted glandless cottonseed flour inhibits mutagen formation (Rhee et al., 1987). Diallyl disulfide and dipropyl disulfide, minor components in onion, were shown to prevent HCA formation in boiled pork juice (Tsai et al., 1996). Garlic-related sulfur compounds were also inhibitory (Shin et al., 2002a,b). Concentrations of HCAs in and overall mutagenicity of fried ground beef patties can be reduced by the addition of substances such as the whole cherry tissue (Britt et al., 1998), antioxidant spices (Murkovic et al., 1998), vitamin E (Balogh et al., 2000) and unifloral honeys (Shin et al., 2003b).

The effect of fresh made virgin olive oil containing a high level of dihydroxyphenylethanol derivatives on the formation...
of HCAs in an aqueous model system composed of glucose, glycine and creatinine was examined (Monti et al., 2001). Virgin olive oil inhibited mutagen formation by between 30 and 50% compared with control 1-year-old oil. The inhibition of HCA formation was verified using phenolic compounds extracted from virgin olive oil.

We looked for scavengers of the pyrazine cation radical formed in the early stages of the Maillard reaction of glucose and glycine in diethyleneglycol–water or polyethylene glycol–water. The results are summarized in Table II. The antioxidants BHA, sesamol and epigallocatechin gallate (EGCG), most abundant polyphenolic in green tea, were effective at relatively high concentrations (Kato et al., 1996). Thiol compounds, including cysteine (CySH) and N-acetyl cysteine (NAC) (Kikugawa et al., 1999), and reductones, including ascorbate and erythorbate (Kikugawa et al., 2000a,b), were effective radical scavengers at relatively lower concentrations. The scavenging effects of ascorbate and erythorbate are shown in Figure 7. Unsaturated fatty acids were also effective radical scavengers (Kikugawa et al., 1999). The reductones generated in the Maillard reaction, 4-hydroxy-3-(2H)-furanones, promote formation of the pyrazine cation radical by interaction with glycine (Kikugawa et al., 2000b).

Scavengers of the pyrazine cation radical were tested as inhibitors of mutagen formation in the model system composed of glucose, glycine and creatinine in diethyleneglycol–water (Table II). The antioxidants BHA, propyl gallate (PG), sesamol and EGCG were effective in reducing the overall mutagenicity at relatively high concentrations (Kato et al., 1996). This observation is consistent with earlier ones showing that a wide variety of flavonoids, including EGCG, inhibit HCA formation in the model system (Weisburger et al., 1994; Oguri et al., 1998). Thiol compounds and unsaturated fatty acids also inhibit mutagen formation in the model system.
Ascorbate and erythorbate inhibit mutagen formation, but the Maillard intermediates 4-hydroxy-3-(2H)-furanones were not inhibitory (Kikugawa et al., 2000b). A possible pathway for generation of the pyrazine cation radical and formation of imidazoquinoxaline-type HCAs and the effects of antioxidants and reductones are shown in Figure 8.

We attempted to reduce the mutagenicity of smoked-and-dried bonito (katsuobushi in Japanese), a common seasoning in Japan. When a small piece of bonito meat (~20 g) was boiled in a 0.5% EGCG solution or in a 5% green tea extract and subsequently heated in air at 100°C for 24 h, the mutagenicity of the heated-and-dried bonito meat was reduced to <50% (Kato et al., 1996). Decreasing the mutagenicity of katsuobushi by boiling bonito meat in green tea extract was attempted. A large piece (~500 g) of bonito meat was boiled in 2.5 and 5% (w/v) green tea extract, then smoked-and-dried in the usual manner of katsuobushi processing. The mutagenicity of katsuobushi was only slightly decreased, thus there was a limit to the use of green tea extract to decrease the mutagenicity of katsuobushi (Kato and Kikugawa, 1999).

**Table II. Effect of antioxidants and reductones on the generation of pyrazine cation radical and mutagen formation**

<table>
<thead>
<tr>
<th>Antioxidant/reductone</th>
<th>ESR signals of the model system (%) (mM)a</th>
<th>Mutagenicity of the model system (%) (mM)b</th>
<th>Mutagenicity of cooked hamburger (%)c</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Phenolic antioxidants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BHA</td>
<td>0 (100)</td>
<td>20 (100)</td>
<td></td>
</tr>
<tr>
<td>Sesamol</td>
<td>0 (100)</td>
<td>40 (100)</td>
<td></td>
</tr>
<tr>
<td>EGCG</td>
<td>0 (100)</td>
<td>40 (100)</td>
<td>97 (0.33)</td>
</tr>
<tr>
<td>Thiol compounds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CySH</td>
<td>10 (10)</td>
<td>30 (50)</td>
<td>100 (0.33)</td>
</tr>
<tr>
<td>NAC</td>
<td>0 (10)</td>
<td>94 (0.33)</td>
<td></td>
</tr>
<tr>
<td>Reductones</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascorbate</td>
<td>0 (50)</td>
<td>60 (50)</td>
<td>65 (0.33)</td>
</tr>
<tr>
<td>Erythorbate</td>
<td>0 (50)</td>
<td>60 (50)</td>
<td>60 (0.33)</td>
</tr>
<tr>
<td>Maillard reductone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-Hydroxy-3(2H)-furanones</td>
<td>200 (20)</td>
<td>100 (100)</td>
<td></td>
</tr>
</tbody>
</table>

aThe model system was composed of 0.2 M glucose and 0.4 M glycine in diethyleneglycol–water (8:2 v/v) or in polyethyleneglycol–water (6:4 v/v) heated at 120°C for 5 min.
bThe model system was composed of 0.2 M glucose, 0.4 M glycine and 0.4 M creatinine in diethyleneglycol–water (8:2 v/v) heated at 120°C for 2 h.
cHamburger prepared by heating ground beef at 200°C for 20 min (Kikugawa et al., 2000a).

**Fig. 7.** ESR spectra of mixtures of 0.2 M glucose and 0.4 M glycine in diethyleneglycol–water (8:2 v/v) in the presence of ascorbate or erythorbate at the indicated concentration heated at 120°C for 5 min. Two signals are due to the monodehydroascorbyl (or erythorbyl) radical with $g_H=0.18$ mT (Kikugawa et al., 2000b).
We then tried to reduce the mutagenicity of cooked hamburger by the introduction of antioxidants or reductones to ground beef (Table II). EGCG, CySH and NAC at doses lower than 0.33% (w/w) were not effective in reducing mutagen formation (Kato et al., 2000a), whereas tea polyphenols have been shown to prevent mutagen formation in cooked meat (Weisburger et al., 2002). Ascorbate and erythorbate at 0.33% (w/w) were effective in reducing the mutagenicity of cooked hamburger by 50% (Figure 9) (Kato et al., 2000a). The taste and smell of the hamburger were not affected by cooking with ascorbate and erythorbate at these doses.

Antioxidants and reductones are effective in reducing overall mutagenicity due to HCA formation in the heated model system and cooked meat through scavenging the intermediary pyrazine cation radical in the early stages of the Maillard reaction. However, the potencies of the antioxidants and reductones differ by compound. Ascorbate and erythorbate are the most potent scavengers of the radical and the most potent inhibitors of HCA formation at practicable low doses.

Conclusion

In this review various methods for minimizing mutagenicity derived from HCA formation in heated model systems and cooked meats have been described. Because the temperature, time and cooking method greatly affect mutagen formation, controlling these factors can reduce the mutagenic activity. Avoiding loss of water from model systems and meats is another way to reduce mutagen formation. Inhibition of the formation of the intermediary pyrazine cation radical in the early stages of the Maillard reaction or scavenging the radical is also effective in reducing mutagenicity. Thus, addition of an excess amount of reducing sugars or of a small amount of ascorbate or erythorbate to meats before cooking is effective in minimizing mutagen formation.

References


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