CHAPTER **10**

Toxicants Formed during Food Processing

The development of food processing technology—which includes frying, toasting, roasting, evaporation, smoking, sterilization, pasteurization, irradiation, pickling, freezing, and canning—expanded the potential of food supplies greatly in the modern era. For example, smoke treatment made a year-round supply of fish possible and canned foods could be sent anyplace in the world.

In the United States, commercial food processing is subject to regulation by the FDA and must meet specified standards of cleanliness and safety. Sometimes particular methods of food processing are considered under the category of food additives, since they may intentionally alter the form or nature of food.

Home cooking is one important method of food processing. Cooking increases the palatability (for example, flavor, appearance, texture) and stability of foods; it also improves the digestibility of foods. Moreover, it kills toxic microorganisms and deactivates such toxic substances as enzyme inhibitors. Since antiquity, people appreciated home-cooked food.

The chemical changes in food components, including amino acids, proteins, sugars, carbohydrates, vitamins, and lipids, caused by high-heat treatment have raised questions about the usual consequence of reducing nutritive values and even the formation of some toxic chemicals such as polycyclic aromatic hydrocarbons (PAHs), amino acid or protein pyrolysates, and *N*-nitrosamines. Among the many reactions occurring in processed foods, the Maillard Reaction plays the most important role in the formation of various chemicals (including toxic ones).

During processing, it frequently happens that some foreign materials are mixed into foods. Some of these materials are undesirable ones.

Although most modern food factories are engineered to avoid any occurrence of food contamination during processing, low-level contamination is hard to eliminate entirely. Many instances of accidental contamination of food by toxic materials have been reported. For example, in 1955 in Japan, a neutralizing agent (sodium phosphate) that was contaminated with sodium arsenite was added to milk during a drying process; the final commercial dried milk contained 10–50 ppm of arsenic. Subsequently, many serious cases of arsenic poisoning were reported.

It is a common misunderstanding that gamma irradiation, which is most often used for food irradiation, produces radioactive materials in foods. In fact, although the electromagnetic energy used for irradiation is sufficient to penetrate deep into foods and can kill a wide range of microorganisms, it is far below the range required to produce radioactivity in the target material. However, there are still uncertainties about the toxicity of chemicals that may be produced during irradiation. The energies used are sufficient to produce free radicals, which may in turn produce toxic chemicals.

I. Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons occur widely in the environment. The typical PAHs are shown in Figure 10.1. They are found in water, soil, dust, and in many foods. For over 200 years, carcinogenic effects have been ascribed to PAHs. In 1775, Percival Pott, an English physician, made the association between the high incidence of scrotal cancer in chimney sweeps and their continual contact with chimney soot. The research on the toxicity of PAHs, however, progressed somewhat slowly. In 1932, benzo[*a*]pyrene (BP) was isolated from coal tar and found to be highly carcinogenic in experimental animals.

A. Occurrence

One of the most abundant food sources of PAHs is vegetable oil. It is possible that the high levels of PAHs in vegetable oils are due to endogenous production, with environmental contamination playing only a minor role. However, some of the PAHs in vegetables are apparently due to environmental contamination because levels of these substances decrease with increased distance from industrialized centers and freeways. The

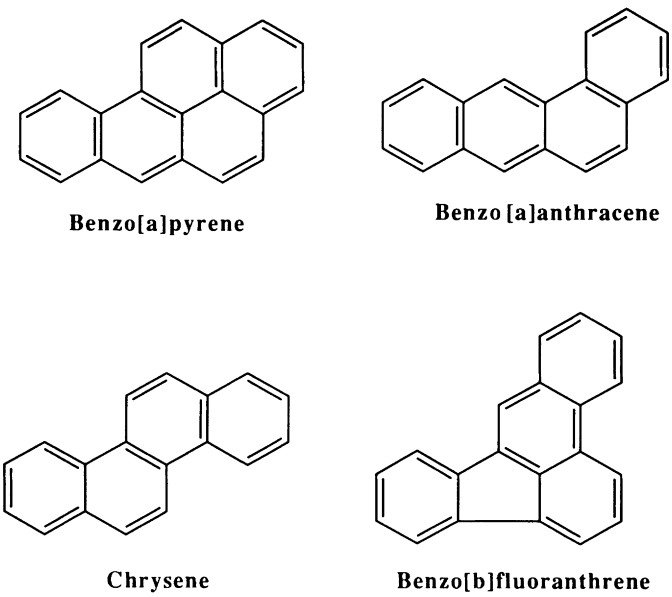


Figure 10.1 Polycyclic aromatic hydrocarbons.

occurrence of PAHs in margarine and mayonnaise appears to be due to contamination of the oils used to make these products.

Levels of PAHs in soil can be quite high, even in areas distant from industrialized centers. Levels of PAHs of 100–200 ppm in the soil were found in some locations distant from human populations. It is thought that these levels result primarily as a residue from decaying vegetation. The significance of these relatively high levels of potentially carcinogenic substances in the soil is not fully understood.

Charcoal broiling or smoking of food also causes PAHs contamination (Table 10.1). PAHs are formed mainly from carbohydrates in foods at high temperatures in the absence of oxygen. Broiling meat over hot ceramic or charcoal briquettes allows the melted fat to come into contact with a very hot surface. PAHs are produced in the ensuing reactions. These products rise with the resulting cooking fumes and are deposited on the meat. Similarly, the presence of PAHs in smoked meats is due to the presence of these substances in smoke. PAHs levels in meat that is cooked at a greater distance from the coals are lower than in meat that is cooked close to the coals. Obviously, food processing produces PAHs in certain levels. It is of major importance to be aware of the presence of carcinogenic PAHs in our foods, and the overall public health hazard should be evaluated and controlled.

TABLE 10.1
Polycyclic Aromatic Hydrocarbons Found in Smoked Foods (ppb)

Food	Benzo[*a*] anthracene	Benzo[*a*] pyrene	Benzo[*e*] pyrene	Fluoranthene	Pyrene
Beef	0.4	—	—	0.6	0.5
Cheese				2.8	2.6
Herring				3.0	2.2
Dried herring	1.7	1.0	1.2	1.8	1.8
Salmon	0.5	—	0.4	3.2	2.0
Sturgeon	—	0.8	—	2.4	4.4
Frankfurters	—	—	—	6.4	3.8
Ham	2.8	3.2	1.2	14.0	11.2

B. Benzo[a]pyrene

The most commonly known carcinogenic PAH is benzo[*a*]pyrene (BP), which is widely distributed in various foods (Table 10.2). BP was reportedly formed at a level of 0.7 and 17 ppb at 370 to 390 and 650°C, respectively, when starch was heated. Amino acids and fatty acids also produced BP upon high-temperature treatment (Table 10.3). Many cooking processes utilize the 370–390°C range; for example, the surface temperature of baking bread may approach 400°C and deep fat frying is 400–600°C, suggesting that cooking produces some PAHs, including BP. The meat inspection division of the USDA and FDA analyzed 60 assorted foodstuffs and related materials for BP. Samples that contain relatively high levels of BP are shown in Table 10.1.

TABLE 10.2
Benzo[a]pyrene Found in Various Foods

Food	Concentration (ppb)
Fresh vegetables	2.85–24.5
Vegetable oil	0.41–1.4
Coffee	0.31–1.3
Tea	3.9
Cooked sausage	12.5–18.8
Smoked hot sausage	0.8
Smoked turkey fat	1.2
Charcoal-broiled steak	0.8
Barbecued ribs	10.5

TABLE 10.3
Amounts of PAHs Produced from Carbohydrates, Amino Acids, and Fatty Acids Heated at 500 and 700°C (μg/50 g)

	Starch		D-Glucose		L-Leucine		Stearic acid	
PAH	500	700	500	700	500	700	500	700
Pyrene	41	965	23	1680	—	1200	0.7	18,700
Fluoranthene	13	790	19	1200	—	320	—	6,590
Benzo[*a*]pyrene	7	179	6	345	—	58	—	4,440

1. Toxicity

BP has been subjected to extensive carcinogenic testing. It is a reasonably potent contact carcinogen. Table 10.4 shows the relative carcinogenicity of BP and other PAHs. A diet containing 25 ppm of BP fed for 140 days to mice produced leukemias and lung adenomas in addition to stomach tumors. Skin tumors developed in over 60% of the rats treated topically with approximately 10 mg of benzo[*a*]pyrene three times per week. The incidence of skin tumors dropped to about 20% when treatment was about 3 mg × 3 per week. Above the 10-mg range, however, the incidence of skin tumors increased dramatically to nearly 100%.

The compound is also carcinogenic when administered orally. In one experiment, weekly doses of greater than 10 mg administered for 10 weeks induced stomach cancers, although no stomach cancers were produced at the dose of 10 mg or less. At 100-mg doses, nearly 70% of the animals had developed stomach tumors by the completion of the experiment.

TABLE 10.4
Relative Carcinogenicity of Typical Polycyclic Aromatic Hydrocarbons (PAH)

PAH	Relative activity
Benzo[*a*]pyrene	+++[a]
5-Methylchrysene	+++
Dibenz[*a,h*]anthracene	++
Dibenzo[*a,i*]pyrene	++
Benzo[*b*]fluoranthene	++
Benz[*a*]anthracene	+
Benzo[*c*]phenanthrene	+
Chrysene	+

[a] +++, high; ++, moderate; +, weak.

2. Mode of Toxic Action

BP is transported across the placenta and produces tumors in the offspring of animals treated during pregnancy. Skin and lung tumors appear to be the primary lesions in the offspring.

The biochemical mechanisms by which benzo[*a*]pyrene initiates cancer have been studied in some detail. Benzo[*a*]pyrene is not mutagenic and carcinogenic by itself, but must be first converted to active metabolites. This metabolic conversion involves initially a cytochrome P450-mediated oxidation, producing a 7,8-epoxide. The 7,8-epoxide, in turn, undergoes an epoxide hydrolase-mediated hydration, producing the 7,8-diol which, upon further oxidation by cytochrome P450, produces the corresponding diolepoxide. This diolepoxide is highly mutagenic without metabolic activation and is also highly carcinogenic at the site of administration. The benzo[*a*]pyrene diolepoxide can react with various components in the cells, including DNA, in which case it is possible that a mutation will occur. This is thought to be the primary event in benzo[*a*]pyrene-induced carcinogenesis.

II. Maillard Reaction Products

In 1912, the French chemist L. C. Maillard hypothesized the reaction that accounts for the brown pigments and polymers produced from the reaction of the amino group of an amino acid and the carbonyl group of a sugar. Maillard also proposed that the reaction between amines and carbonyls was implicated in *in vivo* damages; in fact, the Maillard reaction was later proved to initiate certain damages in biological systems. Some products formed by this reaction in processed foods exhibited strong mutagenicity, suggesting the possible formation of carcinogens.

The summary of the Maillard reaction is shown in Figure 10.2. Many chemicals form from this reaction in addition to the brown pigments and polymers. Because of the large variety of constituents, a mixture obtained from a Maillard reaction shows many different chemical and biological properties: brown color, characteristic roasted or smoky odors, pro- and anti-oxidants, and mutagens and carcinogens, or perhaps anti-mutagens and anti-carcinogens. It is common practice to use the so-called Maillard browning model system consisting of a single sugar and an amino acid to investigate complex, actual, food systems. The results of much mutagenicity testing on the products of Maillard browning model systems have been reported. Some Maillard model systems that produced mutagenic materials are shown in Table 10.5.

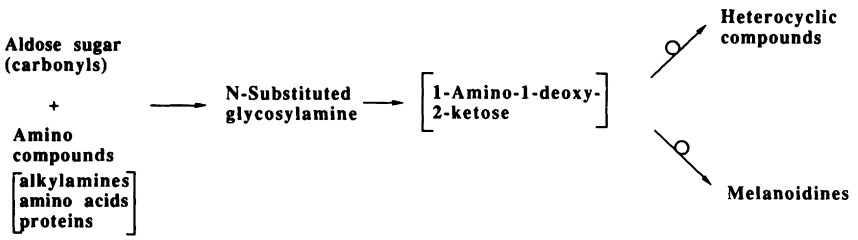


Figure 10.2 Summary of the Maillard reaction.

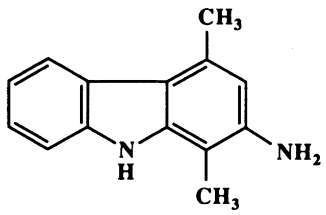
III. Amino Acid Pyrolysates

In the late 1970s, mutagenicity of pyrolysates obtained from various foods was reported that could not be accounted for by PAHs formed on the charred surface of certain foods such as broiled fish and beef. The mutagenic principles of the tryptophan pyrolysates were later identified as nitrogen-containing heterocyclic compounds. A group of polycyclic aromatic amines is produced primarily during the cooking of protein-rich foods. Their structures are shown in Figure 10.3. The early work on the isolation and production of these substances was based on their mutagenicity. Some classes of cooked protein-rich foods tended to be more

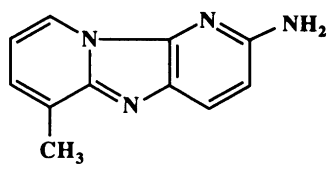
TABLE 10.5
Mutagenic Materials Produced from the Maillard Model System

Model system	*Salmolella typhimurium* strains
D-Glucose/cysteamine	TA 100 without S9[a]
	TA 98 with S9
Cyclotene/NH_3	TA 98 without S9
	TA 1538 without S9
L-Rhamnose/NH_3/H^2S	TA 98 with S9
Maltole/NH_3	TA 98 with S9
	TA 100 with S9
Starch/glycine	TA 98 with S9
Lactose/casein	TA 98 with S9
Potato starch/$(NH_4)_3CO_4$	TA 98 with S9
	TA 100 with S9
Diacetyl/NH_3	TA 98 with S9
	TA 100 with S9

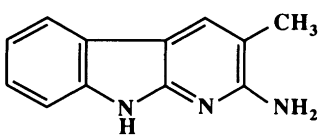
[a] Metabolic activation.



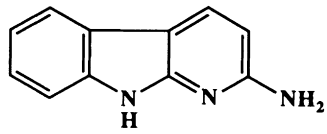
Trp-P-1



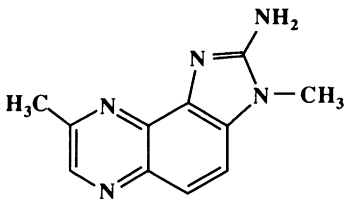
Glu-P-1



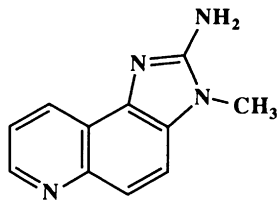
MeAαC



AαC



MeIQx



IQ

Figure 10.3 Mutagenic heterocyclic amines.

mutagenic than others, and the extent of heating influenced the level of mutagenic activity. The most highly heated samples of milk, cheese, tofu, rock cod, and several varieties of beans, although heavily charred, were only weakly mutagenic. Hamburger cooked at high temperatures was reported as mutagenic. The mutagenecity was, however, limited to the surface layers where most pyrolysates are found. On the other hand, no mutagenic activity was found in comparable samples of uncooked hamburger meat. The formation of these mutagenic substances seems to depend on temperature, and the temperature dependence of mutagen formation in heated beef stock has been determined quantitatively.

The identities of the mutagens produced under normal cooking con-

ditions have been established in some cases. The major mutagens in broiled fish are the heterocyclic amines imidazoquinoline (IQ) and methylimidazoquinoline (MelQx) (Figure 10.3). They are also minor components of fried beef. Several other mutagens of this class are also present in cooked meat. Beef extracts, which contain IQ and MelQx, are metabolically converted to active mutagens by liver tissue from several animal species and humans. Although these substances are highly potent mutagens, they are fairly weak carcinogens in rats. Following the mutagenicity studies on these pyrolysates, carcinogenicity of tryptophane (Trp-P-1 and Trp-P-2) and glutamine (Glu-P-1) were demonstrated using animals such as rats, hamsters, and mice. For example, a high percentage of tumor incidence was observed in mice fed a diet containing Trp-P-1 or Trp-P-2. The various reports indicate that both amino acid and protein pyrolysates may act as carcinogens in the alimentary tracts of experimental animals. Extensive research is presently being conducted to determine whether heterocyclic amines produced during the cooking process are hazardous to humans.

IV. *N*-Nitrosamines

Mixtures of inorganic salts, such as sodium chloride and sodium nitrite, have been used to cure meat for centuries. Only relatively recently has it been recognized that the curing action results from the nitrite ion. Bacterial reduction can produce nitrite from nitrate ions, although today nitrite is used directly. Certain fish products are also cured with nitrite. Some countries (but not the United States) also permit the addition of nitrate in the production of some varieties of cheese.

A. Precursors

1. Nitrite and Nitrate

The nitrite ion plays at least three important roles in the curing of meat. First, it has an antimicrobial action. In particular, it inhibits the growth of the microorganism that produces botulism toxin, *Clostridium botulinum*. The mechanism and cofactors of this antimicrobial action are not understood. However, since cured meats are often stored under anaerobic conditions for extended periods, it is very important in ensuring the safety of these foods. Nitrite also imparts an appealing red color

to meats during curing. This color arises from nitrosylmyoglobin and nitrosylhemoglobin pigments. These pigments are formed when nitrite is reduced to nitric oxide, which then reacts with myoglobin and hemoglobin. If these pigments did not form, cured meats would have an unappetizing grayish color. Finally, nitrite gives a desirable "cured" flavor to bacon, frankfurters, ham, and other meat products. The levels of nitrites that are permissible in cured foods vary from country to country and range from 10 to 200 ppm. The major portion of nitrite in humans results from reduction of dietary nitrate by bacteria in the mouth and in the intestinal tract. Nitrate is encountered in the diet, often in relatively high levels (1000–3000 ppm), in vegetables such as cabbage, cauliflower, carrots, celery, and spinach; the levels are variable and the exact causes are uncertain.

Nitrate is widely found in foods. The dietary intake for adult Americans has been estimated at 100 mg per day. Vegetables, especially leaf and root vegetables, account for over 85% of the total, while cured meats contribute about 9%. In certain areas, well water contains high levels of nitrate. While exposure from meat products may have dropped in recent years, the use of nitrate in fertilizers, and its concomitant widespread occurrence in soils and water, means that vegetables continue to be significant sources.

The reduced nitrite is not found in significant amounts in most foods. The chief dietary source is cured meats, in which it is present as an intentional food additive because of its desired antimicrobial, flavor, and color properties. Most ingested nitrite comes from saliva, which is estimated to contribute 8.6 mg of the total daily intake of 11.2 mg from the diet.

B. Occurrence

Nitrosation of secondary and tertiary amines produces stable nitrosamines. Unstable nitroso compounds are produced with primary amines. The reaction rate is pH-dependent and is maximum near pH 3.4. The nitrosation of weakly basic amines is more rapid than that of more strongly basic amines. Several anions, such as halogens and thiocyanate, promote the nitrosation process; on the other hand, antioxidants, such as ascorbate and vitamin E, inhibit the reaction of destroying nitrite. Diethylnitrosamine (DEN) and dimethylnitrosamine (DMN) occur in the gastric juice of experimental animals and humans fed diets containing amines and nitrite. The nitrosation reaction is also known to occur during high-temperature heating of foods, such as bacon, which contain nitrite and certain amines.

In Norway in 1962, following an epidemic of food poisoning in sheep, extremely high levels of nitrosamines were detected in herring meal treated with nitrite as a preservative. The sheep suffered severe liver disease and many of them died. It was later shown that the rate of spontaneous formation of nitrosamine in nitrite-treated fish was dependent on the temperature of preparation following the addition of nitrite. Thus, refrigerated fish treated with nitrite had no more nitrosamine than fresh fish treated with nitrite, but heat treatment of fish increased the rate of nitrosamine formation following addition of nitrite. It was suggested that increased levels of nitrosamines in heated fish are due—at least in part—to increased concentrations of secondary amines resulting from protein degradation during the heating process.

Heating of other nitrite-treated foods has also been shown to produce nitrosamines. Cured meats have all been shown to contain nitrosamines (Table 10.6), the higher levels appearing in cured meats that have been subjected to relatively high heating. It is important to note that the levels of nitrosamines detected in various foods are quite variable. The reasons for this variability are not clear but seem to be dependent on the type of food and the laboratory conducting the examination.

The levels of volatile nitrosamines in spice premixes, such as those used in sausage preparation, were found to be extraordinarily high. These premixes contained spices with secondary amines and a curing mixture that included nitrite. Volatile nitrosamines formed spontaneously in these premixes during long periods of storage. The problem was solved simply by combining the spices and the curing mixture just prior to use.

Analyses of certain beers have also shown considerable variability in levels of nitrosamines. Although the mean concentration of volatile nitrosamines in both American and imported beer is generally quite low, the levels in certain samples can be as high as 70 ppb of dimethylnitrosamine. It was soon found that beers produced from malt dried by direct

TABLE 10.6
Nitrosamine Content in Typical Cured Meats

Meat	Nitrosamine	Level (ppb)
Smoked sausage	Dimethylnitrosamine	< 6
	Diethylnitrosamine	< 6
Frankfurters	Dimethylnitrosamine	11–84
Salami	Dimethylnitrosamine	1–4
Fried bacon	Dimethylnitrosamine	1–40
	Nitrosoproline	1–40

fire rather than by air-drying had the highest levels of nitrosamine. The direct fire-drying process was shown to introduce nitrite into the malting mixture. Domestic beer manufacturers quickly converted to the air-drying process.

C. Toxicity

The carcinogenic activity of many nitrosamines has been examined. Of the over 100 food substances assayed so far, approximately 80% were shown to be carcinogenic in at least one animal species. Diethylnitrosamine is active in 20 animal species. Dimethyl- and diethylnitrosamine are two of the most potent carcinogens in this group. Administration of dimethylnitrosamine at 50 ppm in the diet produces malignant liver tumors in rats in 26–40 weeks. Higher doses produce kidney tumors. As the dose of diethylnitrosamine is reduced below 0.5 mg/kg, the lag period between dosing and onset of tumors increases, with the total tumor yield remaining roughly the same. With a dose of 0.3 mg/kg, the lag time is 500 days, whereas for a dose of 0.075 mg/kg, the lag time is increased to 830 days. No clear threshold dose for carcinogenicity of nitrosamines in the diet has yet been established.

D. Mode of Toxic Action

Nitrosamines, like other groups of chemical carcinogens, require metabolic activation to render them toxic. The activation process is mediated by enzymes and involves, at least in some cases, hydroxylation of the α-carbon (Figure 10.4). The nitrosamines exhibit a good deal of organ specificity in their carcinogenic effect (Table 10.7); for example, dimethylnitrosamine is an active liver carcinogen with some activity in the kidney, and benzylmethylnitrosamine is specific for the esophagus. This organ specificity is apparently due, at least in part, to site-specific metabolism.

Administration of certain nitrosamines to pregnant animals can result in cancer in the offspring. The time of administration seems to be critical. For example, in rats, administration of the carcinogens must occur later than 10 days into gestation to produce cancer in the offspring, and the fetuses are most sensitive just prior to term. This development of sensitivity coincides with the development of the metabolic activation system of the fetuses. In addition, compared to the adults, the fetuses seem to be unusually sensitive to the carcinogenic effects of these substances. For example, at a maternal dose of only 2 mg/kg, which is 2% of

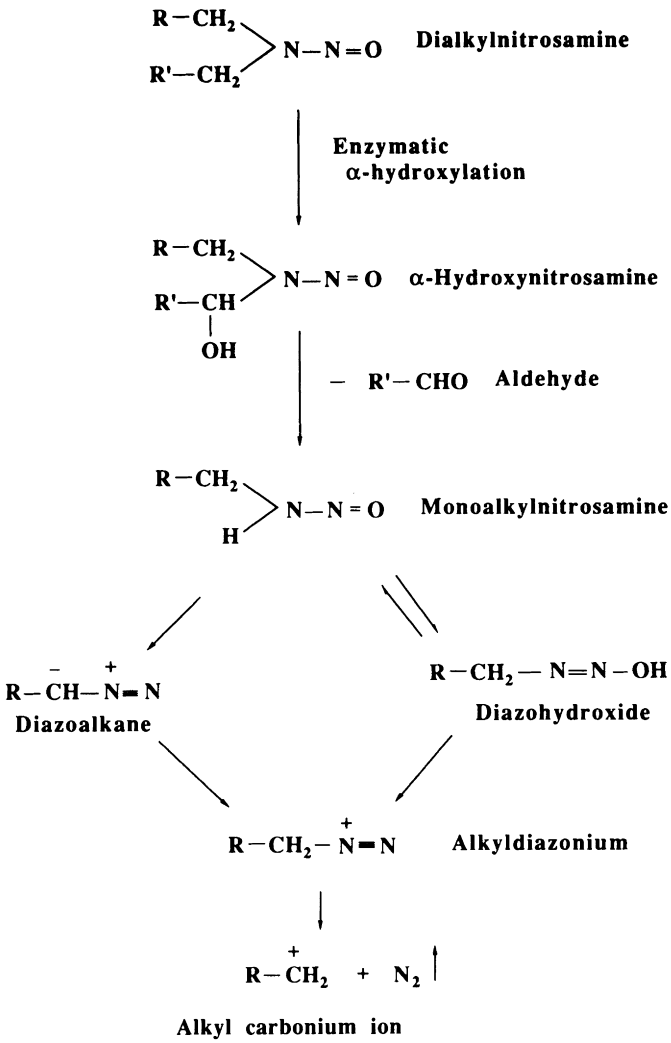


Figure 10.4 Formation of alkylating agent from nitrosamines.

the carcinogenic dose required for adults, N-nitrosoethylurea caused a carcinogenic response in the nervous system of offspring.

Under acidic pH, the nitrite ion can be protonated to form nitrous acid (HONO). The anhydride of nitrous acid, N_2O_3, present in equilibrium with nitrous acid, can nitrosate a variety of compounds, especially secondary and tertiary amines. Halide and thiocyanate ions, present in foods and digestive fluids, can catalyze the formation of *N*-nitroso compounds.

TABLE 10.7
Sites of Tumors Produced by *N*-Nitroso Compounds

Site	Compound
Skin	Methylnitrosourea
Nose	Diethylnitrosamine
Nasal sinus	Dimethylnitrosamine
Tongue	Nitrosohexamethyleneimine
Esophagus	Nitrosoheptamethyleneimine
Stomach	Ethylbutylnitrosamine
Duodenum	Methylnitrosourea
Colon	Cycasin
Lung	Diethylnitrosamine
Bronchi	Diethylnitrosamine
Liver	Dimethylnitrosamine
Pancreas	Nitrosomethylurethane
Kidney	Dimethylnitrosamine
Urinary bladder	Dibutylnitrosamine
Brain	Methylnitrosourea
Spinal cord	Nitrosotrimethylurea
Thymus	Nitrosobutylurea
Lymph nodes	Ethylnitrosourea
Blood vessels	Nitrosomorpholine

E. General Considerations

Efforts to reduce nitrosamine formation in cured meats have been quite successful. Simply adding a reducing agent, such as erythrobate or ascorbate, to the curing mix greatly reduced or eliminated nitrosamine formation in the final product. Domestic manufacturers of cured meat products now generally add these reducing agents to the curing mixture along with the minimum amount of nitrite necessary to achieve the desired effect. However, the nitrosamines found in foods are almost exclusively highly volatile. Very little is presently known about the concentrations of nonvolatile nitrosamines present in foods.

The risk to human health of dietary nitrite and nitrosamines is difficult to assess. As discussed in the preceding section, *in vivo* reduction of the ubiquitous nitrate ion to nitrite appears to be the major source of ingested nitrite, contributing more than three times the nitrite ingested with cured meats in the average American diet. Both catalysts and inhibitors of nitrosation may be present in a typical meal. In addition, there

are significant nondietary sources of exposure to nitrosamines and nitrosatable compounds, including tobacco, some pharmaceuticals and cosmetics, and cutting oils used in industry. Isolating effects due to diet alone appears impossible. Nonetheless, it is prudent to minimize controllable exposures.

V. Food Irradiation

In the United States, commercial food processing is subject to regulation by the FDA and must meet specified standards of cleanliness and safety. In certain situations, methods of food processing are considered under the category of food additives since they may intentionally alter the form or nature of food. The use of ionizing radiation to preserve food falls into this category.

Gamma radiation is most often used for food irradiation. Gamma rays are a form of electromagnetic radiation produced by such radioactive elements as Cobalt-60 and Cesium-137. Such sources emit radiation with energies of up to 10 million electron volts (MeV). This is sufficient to penetrate deep into foods, but is far below the range required to produce radioactivity in the target material. Since there is no direct contact between the source and the target, there is no mechanism that can produce radioactivity in irradiated foods.

Studies in the use of ionizing radiation to preserve food began shortly after World War II. A large number of potential applications have been identified. Ionizing radiation can sterilize foods, control microbial spoilage, control insect infestations, and inhibit undesired sprouting. Food irradiation has the potential to substantially reduce postharvest applications of pesticides to prevent spoilage due to insects and fungi. Irradiation can be used to destroy *Salmonella* in cases where heat treatment is not possible, for example, in frozen chicken.

Despite the potential of food irradiation as a preserving technique, it is widely misunderstood and controversial. Some opposition arises from apparent confusion between "irradiated" and "radioactive." Gamma irradiation of foods is in some ways analogous to sterilization of medical equipment with ultraviolet light. Both of these processes can kill a wide range of microorganisms by radiation.

Some other critics have raised questions about the toxicity of chemicals that may be produced during irradiation. The energies used are sufficient to produce free radicals, which can combine with each other or form new bonds to other compounds that may be present. However, it is

important to remember that heat treatments commonly used in food processing are likely to produce a higher degree of chemical modification than is irradiation.

Suggestions for Further Reading

1. Ayres, J. C., and Kirschman, J. C. (eds.) (1981). "Impact of Toxicology on Food Processing." AVI Publishing Co., Westport Connecticut.
2. Diehl, J. F., (1990). "Safety of Irradiated Foods." Marcel Dekker, New York.
3. Doyle, J. (1985). "Altered Harvest: Agriculture, Genetics, and The Fate of The World's Food Supply." Viking, New York.
4. Hathcock, J. N. (ed.) (1982–1989). "Nutritional Toxicology." Academic Press, San Diego.
5. Miller, E. C., Miller, J. A., Hirono, I., Sugimura, T., and Takayama, S. (eds) (1979). "Naturally Occurring Carcinogens, Mutagens, and Modulators of Carcinogenesis." University Park Press, Baltimore.
6. Okun, M. (1986). "Fair Play in the Marketplace: The First Battle for Pure Food and Drugs." Northern Illinois University Press, Dekalb, Illinois.
7. Ory, R. L. (ed.) (1981). "Antinutrients and Natural Toxicants in Foods." Food & Nutrition Press, Westport, Connecticut.
8. Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences (1983). "Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects." Committee on Pyrene and Selected Analogues. National Academy Press, Washington, D.C.
9. Roberts, H. R. (ed.) (1981). "Food Safety." Wiley, New York.
10. Urbain, W. M. (1986). "Food Irradiation." Academic Press, Orlando.
11. Webb, T., and Lang, T. (1987). "Food Irradiation: The Facts." Thorsons, Rochester, Vermont.