

CHEMICAL HAZARD ANALYSIS FOR SODIUM NITRITE IN MEAT CURING

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Cured meat has specific properties including a pink color and characteristic flavor and texture. Potassium nitrate and sodium nitrite have a long history of use as curing ingredients, and by the close of the 19th century the scientific basis of the process was becoming understood. It was realized, for example, that nitrate must be converted to nitrite in order for the curing process to proceed.

Regulations controlling the use of curing agents were established in the USA in 1926 (see USDA, 1925; USDA, 1926), and the same rules are in effect at present, with slight modification. The critical feature of these rules is that a maximum use level of sodium nitrite is defined; but the meat processor may use less. Basically, no more than one-quarter ounce (7.1 g) may be used per 100 pounds (45.4 kg) of meat (resulting in 156 mg/kg or 156 ppm). While nitrate is still permitted, it is, in fact, not used by the industry. The regulations were changed for bacon so that ingoing nitrite is targeted at 120 ppm, and the maximum use of ascorbates (550 ppm) is mandated. The current routine use of ascorbates (ascorbic acid, sodium ascorbate, erythorbic acid and sodium erythorbate) by the meat processing industry is important not only because it accelerates and improves the curing process but also the use of ascorbates inhibits nitrosation reactions which might result in formation of carcinogenic nitrosamines (Mirvish et al, 1995).

Sodium nitrite is an effective antimicrobial agent for certain pathogens, particularly *Clostridium botulinum*. In the event that cured meats are temperature abused, sodium nitrite provides a degree of protection by delaying outgrowth of spores. Nitrite also functions as an antioxidant in cured meat. There is concern, however, that left-over or residual nitrite in the meat poses a health risk to humans.

One possible chemical hazard involved in producing processed meats would be an error in the use of sodium nitrite. If too much is added there is a risk of illness, even death, to the consumer. USDA recognized this concern when the regulations permitting the direct use of sodium nitrite were established. Levels of use and safeguards in handling it were established. The industry itself has devised further control methods. The remainder of this paper consists of a discussion of the chemistry and toxicity of sodium nitrite, an explanation of how it is handled and controlled and an analysis of the chemical hazard it presents in meat curing.

Nitrite is a reactive chemical, and when used in biologically complex meat system presents numerous reaction possibilities (Cassens et al, 1979). Nitrite acts as an oxidizing agent, and in the mildly acidic conditions of meat a small quantity of nitrous acid is formed. One of the reactive species formed subsequently is nitric oxide, and it is the active nitrosating agent.

The level of nitrite analytically detectable in cured meat is greatly reduced from the amount added because the nitrite reacts with components of the meat during processing and storage. Modern-day cured meats at retail have a residual nitrite content of about 10 ppm (Cassens, 1997).

Sodium nitrite is a toxic substance, and at sufficient dose levels, is toxic in humans. Fassett (1973) and Archer (1982) referenced the widely used clinical toxicology book of Gleason et al (1963) and estimated the lethal dose in humans is 1 g of sodium nitrite in adults (about 14 mg/kg). Ellenhorn and Barceloux (1988) in their text about medical toxicology also estimated the lethal dose to be 1 g (14 mg/kg).

The 1963 text by Gleason et al gives sodium nitrite a toxicity rating of “4”. In the 4th edition of the text (Gosselin et al, 1976) a toxicity rating of “4” is maintained on the basis of animal studies, but on the basis of clinical studies in man a toxicity rating of “5” is suggested. A rating of “4” is “very toxic” with a probable oral lethal dose in humans being 50-500 mg/kg. A rating of “5” is “extremely toxic” with a probable oral lethal dose in humans being 5-50 mg/kg.

Gosselin et al (1976) have described in detail the toxicology of sodium nitrite. Sodium nitrite must be swallowed or injected to produce poisoning and has two basic modes of action. First, the relaxation of smooth muscle, especially in the small blood vessels causes reduced blood pressure; second, the oxidation of hemoglobin to methemoglobin. Methemoglobinemia renders the hemoglobin incapable of carrying oxygen. Very young children, because of the presence of the fetal form of hemoglobin, which is more sensitive to oxidation, are more susceptible than adults. Of interest is the fact that low doses are used therapeutically. For example, a dose of 30 mg is suggested as a vasodilator (Merck Index, 1968). Signs and symptoms of nitrite poisoning include intense cyanosis, nausea, vertigo, vomiting, collapse, spasms of abdominal pain, tachycardia, tachypnea, coma, convulsions and death.

Most reports of nitrite poisoning fall into two categories. In the first instance, sodium nitrite is mistaken for ordinary table salt. In the second instance, a source high in nitrate, such as contaminated well water, is consumed by infants. In the second instance, nitrate does not act directly, since it is eliminated fairly rapidly from the body, but it may be converted to nitrite and then cause methemoglobinemia.

The toxic effects of nitrate and nitrite were discussed in the 1981 National Academy of Sciences report; following are two of the paragraphs dealing with dose levels.

“The toxic effects of nitrate and nitrite have been extensively reviewed (Archer, in press; Corre and Breimer, 1979; Green et al, in press; National Academy of Sciences, 1978; World Health Organization, 1973). The committee has summarized this information and described the most prevalent toxic effects. Nitrate and nitrite are discussed together since most toxic reactions are due to nitrite derived from bacterial reduction of nitrate, either prior to ingestion or within the host (Chapter 8).”

“Corre and Breimer (1979) and Burden (1961) have summarized the literature documenting the toxic and lethal levels of nitrate and nitrite. Different studies have reported that the lethal level of nitrate for a 60-kg adult ranges from 4 to 50 g, whereas for nitrite, it ranges from 1.6 to 9.5 g (Corre and Breimer, 1979). Although the criteria for toxicity vary, most authors accept as a criterion for toxicity a single dose that will induce methemoglobinemia. In four studies, the listed toxic dose for nitrate ranged between 2 and 4 g, whereas for nitrite it ranged from 60 to 500 mg (Corre and Breimer, 1979).”

In his consideration of the literature, Schuddeboom (1993) observed the lowest acute oral lethal dose of nitrite reported for man varied from 33-250 mg nitrite/kg body weight, in which the lowest figures applied for children and elderly people. Nitrite is also more toxic to young infants (3 months of age or less) than to adults, giving rise to relatively higher methemoglobin levels in the blood. The lowest toxic dose reported was 1 mg nitrite/kg body weight, whereas in another study 0.5-5 mg nitrite/kg body weight did not cause any toxic effect.

The National Institute of Occupational Safety and Health (NIOSH, 1997) reported that there are data indicating nitrite is a primary irritant, tumorigen, mutagen and that it causes reproductive effects. The levels cited for toxic and lethal doses are in the range of those previously referenced.

The National Academy of Sciences (1981) concluded there is no definitive evidence to suggest that either nitrate or nitrite is carcinogenic. In animals, nitrate has not been shown to be directly carcinogenic or mutagenic. The limited data on nitrite indicate that it may not act directly as a carcinogen, but that it is mutagenic in microbial systems.

Also of interest, at this point, is an understanding of what is considered an acceptable intake of nitrate and nitrite by humans. The FAO/WHO Joint Expert Committee on Food Additives (JECFA) set an acceptable daily intake (ADI) of 0-5 mg/kg body weight for sodium nitrate and an ADI of 0-0.2 mg/kg body weight for sodium nitrite (see WHO 1973).

In view of the foregoing and for the purpose of making some example calculations we assume the lethal dose as 1 g sodium nitrite (14 mg/kg).

Assuming a worst case, if sodium nitrite were mistakenly substituted for salt (sodium chloride) in a cured meat formulation, and assuming no depletion of nitrite during the process, then the product would contain 2.5% sodium nitrite or 25 g/kg of product. A single 45 g hot dog would contain 1.1 g sodium nitrite. The conservative estimate for a lethal dose in humans is 14 mg/kg, meaning the dose would be 1 g for a 70 kg adult and 0.2 g for a 15 kg child. Obviously, the product would be toxic.

Another case, explained by Tompkin (1997), illustrates the advantage of using a preblended curing mix (see subsequent definition of preblended cure). The preblend is designed to provide the USDA permitted level of 156 ppm sodium nitrite based upon the meat content. Considering that the formulation includes water and other nonmeat ingredients, the actual amount of sodium nitrite added to the product is only 110 ppm. Even if an error occurred and twice the amount of preblend were added, this would result in a product with only about 220 ppm of added sodium nitrite. Furthermore, the additional amount of salt would be objectionable and would limit consumption. If it is assumed the human oral lethal dose of sodium nitrite is 22-23 mg/kg of body weight (note—slightly different from our assumption of 14 mg/kg), then a 50 pound child would have to consume about 5 pounds of franks containing twice the permitted level of sodium nitrite i.e., 220 ppm). Another report indicates the oral lethal dose for sodium nitrite is 1 g. This would be equivalent to a 2 ounce frank being formulated with more than 18,000 ppm. This would be impossible when using the salt-nitrite blend.

Cassens (1997) reported that commercially prepared meats in the United States contain approximately 10 mg/kg sodium nitrite when consumed. This is the result of the normal depletion (from 156 ppm or less added nitrite) which occurs due to the mild acidity of the meat, the presence of curing adjuncts such as ascorbates, heat processing and depletion over time during storage. Consumption of one 45 g wiener therefore results in ingestion of 0.45 mg of sodium nitrite, over 400 times less than the lethal dose for a child and over 2,000 times less than the lethal dose for an adult.

Nitrite may be obtained and used in two forms—as the pure chemical sodium nitrite, or as a so-called curing salt or pre-blend in which the sodium nitrite is distributed in and diluted by ordinary salt (sodium chloride). In this latter instance the blend is made so that the use of salt at the normal level in a given product (2.5% for example) results in the correct level of nitrite. The use of curing salts avoids the direct use of nitrite and the attendant concerns about correct

weighing, distribution and validation. If, for example, a mistake is made and too much curing salt is added the consumer is alerted because the product will be too salty.

It must be kept in mind that preblended curing mixes may have different concentrations of nitrite. In other words, they could be manufactured for different applications or for specific formulations, so as to make them optimally useful for a variety of products and applications.

Germany and other European Community countries recognized the concern associated with keeping and handling a chemical such as sodium nitrite. They have mandated that only diluted forms of sodium nitrite such as those in pre-blends or curing salt mixtures be allowed in meat processing establishments.

USDA recognizes the toxic nature of sodium nitrite and states in the regulations, “supplies of sodium nitrite and potassium nitrite and mixtures containing them must be kept securely under the care of a responsible employee of the establishment. The specific nitrite content of such supplies must be known and clearly marked accordingly” (USDA CFR 9.318.7).

There are numerous types of cured products and processes used to manufacture those products. The required level of nitrite may be added as a dry powder and mixed, chopped or ground into the meat. Alternatively, the nitrite may be dissolved in water and thus incorporated into the meat by mixing, injecting or immersion of pieces.

It is obvious from a summary of the above information that if an establishment is using sodium nitrite diluted with sodium chloride, which is received from the manufacturer with a continuing letter of guarantee, then acute nitrite toxicity is not a problem. In the case of using a preblended cure, nitrite addition should not be considered a critical control point in a HACCP plan. Good manufacturing practices for the control and use of diluted sodium nitrite should, however, be established and referenced as a prerequisite program in the HACCP plan. On the other hand, extreme caution must be exercised if pure sodium nitrite is used. If a company chooses to use pure sodium preblends, then a stand-alone HACCP plan should be developed for the confined nitrite operation. In this case, the use of sodium nitrite should be considered a critical control point.

In the event that both nitrite and nitrate are used, as for example in a dry cured ham, the same conclusions would stand. That is no critical control point if a diluted preblend is used but a critical control point declared in the confined nitrite operation if the pure chemicals are used.

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