

Concerning Nitrate and Nitrite's antimicrobial efficacy – chronology of scientific inquiry

9 May 2015

By Eben van Tonder



Introduction

Producing good bacon is simple, but the processes involved are complex. I am not a historian or a food scientist, but I work in the bacon industry as an entrepreneur. Understanding the environment is fundamental.

The best way for me to understand complex processes is to retrace the historical account of unraveling the system. This is the approach we follow related to bacon. One of the most exciting stories in bacon is that of saltpeter (potassium nitrate) and sodium nitrite. We now come to the question of their efficacy as antimicrobials or are these chemicals merely functional to provide the colour and taste to cured products such as bacon.

Background

Curing brines have been made with salt and a small quantity of saltpeter (potassium or sodium nitrate) for centuries. Nitrate changes into nitrite through microbial reduction. This step takes some time. Once it happens, nitrite starts to undergo further reduction which leads to chemical reactions in the meat, resulting in the cured colour and taste.

The idea started to develop from the mid 1800's that curing time could be sped up by using nitrite directly. A novel curing method was developed in Denmark which spread to places like Calne, Wiltshire, England where it was adopted and called the "*Danish method*".

Nitrite and nitrate were used in combination. A mixture of salt, sugar and saltpeter (potassium nitrate) was injected into the muscle with a single needle injector. The meat was then placed in an old cover brine, called the mother brine. Bacterial reduction had change nitrate (saltpeter) that was originally used in the curing brine into nitrite by bacterial reduction, over time and the mother brine therefore contained the nitrite. It entered the meat through capillary and osmotic forces. (see [The mother brine](#) and [The history of curing](#)) This mother brine was used over and over again, constantly being topped up by saltpeter and constantly undergoing bacterial reduction to nitrite.

In Germany, experiments were done with a compound called sodium nitrite as a possible replacement for nitrate in curing brines. This would be an improvement on the Danish method since the amount of nitrite added could be controlled. Sodium nitrite was a well known compound at this time, being used as part of an intermediary process in the manufacturing of dyes. It is however a very toxic compound and people generally frowned on the thought of adding a poisonous substance to food. (see [Concerning chemical synthesis and food additives](#))

The transition events that caused its wholesale application in the meat industry as a curing agent to replace nitrate are associated with conditions in Germany during World War I. The use of saltpeter (potassium nitrate) in meat curing was made illegal in Germany when war broke out since saltpeter is a key component in the manufacturing of explosives and all available saltpeter were used for the war-effort. The German Government allowed the use of sodium nitrite as a replacement for saltpeter. Unfortunate events in Leipzig where 34 people were killed due to the accidental consumption of sodium nitrite caused it to be banned for use in curing brines, but by this time its quick curing action was so popular that the ban was not heeded. After World War I its use was again permitted and soon its was legalised around the world for use in curing. (see [Concerning the direct addition of nitrite to curing brine](#))

Its application as curing agent remained functionally for colour and taste. Right from the start its use was not without controversy due to its high toxicity and consumer concern over food additives. (see [Concerning chemical synthesis and food additives](#))

Nitrate's role in curing brines

Conventional wisdom that surfaced in the 1920's suggested that nitrate and nitrite should continue to be used in combination in curing brines (Davidson, M. P. et al; 2005: 171) as was the case with the Danish curing method and the mother brine concept of the previous century. Nitrite gives the immediate quick cure and nitrate acts as reservoir for future nitrite and therefore prolongs the supply of nitrite and ensures a longer curing action. The question comes up if there are any other reasons why one should continue to use nitrate? Is there for example any preservative role of nitrate and while we are considering this question, what exactly is the preservative value of nitrite?

Clostridium Botulinum – a key organism to consider

The first thing to remember when considering the effectiveness of a preservative is that not all preservatives are equally effective against all microorganisms. A second point is that different microorganisms are generally associated with different kinds of food. When we look at bacon in particular, what are some of the microorganisms associated with it? Some of these are Lactobacillus, Pseudomonas, Clostridium, yeasts like Dabaryomyces and molds like Aspergillus and Penicillium. (Jay, J. M. et al.; 2005: 102) We then want to look at anti-microbials that are particularly effective against these and other organisms associated with bacon.

Before we look at this list more carefully and how these organisms are managed, one organism is the starting point when considering antimicrobial efficacy of any chemical. The first and most important microorganism to begin with, associated with bacon and other foods is clostridium botulinum. (see [Concerning Clostridium Botulinum – the priority organism](#))

The reason for its priority in food safety is that certain types of its toxins counts as some of the most lethal substances on earth.

Nitrate Useful Against Botulism

April 20, 1972

A headline appeared in a newspaper in California in 1972, reporting that nitrite has been found effective against botulism. (Montclair Tribune; 20 April 1972: 28) The headline

incorrectly read “*Nitrate useful against botulism*”. The study it is reporting on deals with nitrite.

The discovery was news worthy. Botulism is a serious and potentially fatal disease that caused considerable alarm since it was identified in the early 1800’s by Justinus Kerner. (Emmeluth, D.; 2010: 16) It is caused by a toxin called botulin, a neurotoxic proteins produced by the bacteria *clostridium botulinum*. It is so poisonous that one millionth of a gram can kill an adult human. 500mL is enough to kill every person on earth. (Sterba, J. P.; 28 April 1982)

Preventing it remained a focus for the food industry throughout the 1900’s and into the 2000’s and any consideration of the anti-microbial effect of nitrate and nitrite must include its effectiveness in preventing it. It affects humans and animals and one of the ways we contract it is through food. Our article, [Concerning Clostridium Botulinum – the priority organism](#) describes the organism, its toxin formation, prevalence, spores and how it makes its way into food.

Clostridium botulinum was isolated as the microorganism causing botulism in 1895 by Emile Emergem, professor of bacteriology at the university of Ghent, in Belgium. (Emmeluth, D.; 2010: 19) The following year an article appeared in *The Centralia Enterprise and Tribune* in Centralia, Wisconsin, reporting on a warning issued by the the *Connecticut State Department of Health*, issued in its weekly bulletin, in response to two cases of botulism that occurred in New Haven, the week prior. The warning identified home canned foods as the usual source of the botulism. Especially “improperly processed, non-acid fruits and vegetables which are served cold”. The incidents of the previous week were traced back to improperly processed home canned figs. (*The Centralia Enterprise and Tribune*; 25 January 1896: 5)

Such was the public’s concern over botulism that in 1896 when in the US new Food and Drug Administration rules came into effect allowing low-level radiation of food, concern was raised by some consumer groups that this would destroy “*more common and more vulnerable spoilage bacteria*” while deadly botulism bacteria would grow undetected. The argument was that the more common spoilage bacteria would alert the consumer that the food has gone bad before the deadly botulism toxins could be produced. The FDA

responded to this concern by pointing out that at higher radiation levels it would share the concern, but that the levels were too low to completely destroy the spoilage bacteria. (The Laredo Times; 1 December 1896: 14)

It is interesting that this same principle is still a recognised hurdle against botulism where spoilage bacteria is allowed to be present in certain food in order to cause spoilage before clostridium botulinum toxin formation takes place.

The Montclair Tribune article of 20 April 1972 reported on work done by Dr. Richard A. Greenberg, director of research for Swift & Company, on behalf of the American Meat Institute. After studying canned ham he suggested that the unblemished botulism safety record of the curing industry in the USA may be due to the use of *nitrites*.

So, clostridium botulinum will feature prominently in our considerations of the efficacy of nitrate and nitrite as antimicrobial agents, but other bacteria will also be considered.

The historical perspective

There are many reviews of the antimicrobial efficacy of nitrate and nitrite. I rely exclusively on an review article written by Dr. R. Bruce Tompkin (1), the former Vice President for Food Safety, [ConAgra Refrigerated Prepared Foods](#), published as part of Davidson, M. P. et al's, 2005 publication, "*Antimicrobial in Food, Third edition.*" Dr. Tomkin is an exceptionally qualified man to write such a review. He is a "microbiologist with more than 45 years in the food processing industry and one of the developers of HACCP." (Maple Leaf Press release) He arranges the material chronologically which provided insight into why the research was conducted and why certain important points were missed early on.

It is in line with our approach of first understanding the historical background to any technology associated with the bacon industry.



Observations

In 1969, a major issue developed around the question of nitrite's formation of nitrosamines in cured meat and the question if nitrate or nitrites are carcinogenic. This immediately became the major issue that dominated research for the next 30 years. It is an issue of such importance that we will deal with it separately along with the question if one can produce bacon without adding nitrite. The general contention will be that considerable scientific evidence suggesting the safety of nitrite in cured meat, but that the consumers who are not swayed by the arguments should be afforded a choice between nitrite free and traditional nitrite containing products, as long as a nitrite-free option can be offered where the risk of botulin formation has been prevented.

For now we remain with the story of nitrite and nitrate as science started to unlock the fascinating secret of its full effect in cured meats since the 1930's. Most of the research focuses on canned and cured meat and we incorporate some of these important findings and see what can be applied to bacon. The focus on research of nitrite and its effectiveness in canned cured meat makes sense since botulin formation occurs mostly from canned food and due to its deadly nature it is the priority organism in food safety. All consideration of preservatives must therefore start with the question if its effective against clostridium botulism, its spores and toxins.

“Unlike most other antimicrobial agents, there has been a long, controversial history over whether nitrate and nitrite have antimicrobial properties.” (Davidson, M. P. et al.; 2005: 172)

An avalanche of investigations followed, elucidating the efficacy of these chemicals as antimicrobials.

Tanner and Evans (1933) said that sodium chloride (normal table salt), is the most effective component in curing mixtures and that sodium nitrite present, apparently produced no effect on organisms. They then cited MacNeal and Kerr who said that potassium nitrate (saltpeter), in acid solutions had marked inhibitory efficacy. They said that this effect was “incompatibly greater than that of salt.” They believed that the claim of meat packers that small amounts of nitrate in the pickle produced better preservation of the meat was born out by their results. It seemed that nitrate was especially valuable in preventing a high degree of acidity of souring of meat. (Davidson, M. P. et al.; 2005: 172)

Brooks et al (1940) looked at bacon curing in the United Kingdom and concluded that bacon can be produced with nitrite only. “They said that the characteristic cured flavour of bacon is primarily the result of the action of nitrite. The conversion of nitrate to nitrite in commercial bacon curing brines is mainly the result of growth of micrococci. The presence of nitrate or microbial action during the curing process is not essential for bacon flavour.” Rapid chilling, as was practiced in the United States, was also not detrimental, as some speculated. (Davidson, M. P. et al.; 2005: 172)

Tarr and Sutherland (1940) showed that nitrite delayed spoilage in fish. Tarr (1941) revealed the importance of pH to the efficacy of nitrite. At pH 7.01 there was little or no inhibition, but at pH 5.7 and 6.0, complete or strong microbial inhibition occurred”. (Davidson, M. P. et al.; 2005: 173)

Jensen and Hess (1941) insisted that nitrites role was purely colour development and said that nitrate “exerts a definite inhibitory effect upon bacteria”. They reported that nitrite reacts with protein during the heating process and is destroyed, “thus leaving the meat in much the same state as freshly cooked uncured meat”. Scott (1955) agreed. Jensen and Hess said that a combination of heat, nitrate, nitrite and salt caused destruction of anaerobic spores at much lower temperatures. (Davidson, M. P. et al.; 2005: 173)

Yesair and Cameron (1942) took up this concept and reached the conclusion that curing salts do not assist in thermal destruction but inhibit outgrowth. Stumbo et al. (1945) reported that nitrite delayed germination, although salt was the stronger inhibitor. Nitrate alone or in combination with other ingredients did not “appreciably influence spoilage.” (Davidson, M. P. et al.; 2005: 173)

Jensen et al. (1949) looked at the combination of heat and curing salts. The magical temperature range where increased inhibition occurs in tubes of pork was between 50 deg C and 65 deg C, for 30 minutes. Raising the temperature and heating it for longer times did not increase the effect. However, looking at the effect in canned ham, increasing salt and nitrite increased inhibition. Studying these effects of *C. sporogenes* 369 showed that increasing nitrate did not increase the inhibition. (Davidson, M. P. et al.; 2005: 173)

Steinke and Foster 1951 found salt to be major factor retarding botulinal outgrowth in temperature-abused products. Having a moderately high brine of 5.05% to 5.37% and a pH range of 6.1 to 6.5. A combination sodium nitrate, nitrate and nitrite was the most inhibitory. (Steinke and Foster 1951) (Davidson, M. P. et al.; 2005: 174)

Bulman and Ayres (1952) found that a mixed cure of salt, nitrate and nitrite yielded the maximum inhibition. (Davidson, M. P. et al.; 2005: 174)

“Henry et al. (1954) found that at pH 7.5 or above, nitrite enhanced bacterial grow in curing brine. A pH of 5.6 to 5.8 was optimal for antibacterial efficacy. At pH 5.3 or below, nitrite rapidly disappeared and was ineffective. Nitrite was more inhibitory in the presence of ascorbate.” (Davidson, M. P. et al.; 2005: 175)

Castellani and Niven (1955) said that nitrite was not known to have any practical preservative value against those organisms not inhibited by high salt in cured meat. They also found that if a broth medium (pH 6.55) was autoclaved with glucose, a very small amount added nitrite prevented staphylococcal growth when incubated anaerobically. (Davidson, M. P. et al.; 2005: 175)

Lechowich (1956) showed that *S. aureus* growth can occur in any combination of salt, nitrite, and nitrate that is palatable and permissible. (Davidson, M. P. et al.; 2005: 175)

Scott (1955) said that because nitrate exhibited relatively poor antimicrobial inhibition and nitrite, although effective, has been shown to be unstable, the control of salt concentration and resultant water activity is the most reliable bacteriostatic system for cured meats. (Davidson, M. P. et al.; 2005: 175)

As late as in 1957, Eddy was very cautious when expressing an opinion about the antimicrobial ability of nitrite. He wrote: "Taken in their totality, these observations leave no doubt inhibition by nitrite is at least a possibility". (Davidson, M. P. et al.; 2005: 176)

Tomkin summarizes the findings from 1950 to 1960 and state that it was found that nitrite, per se, had no antimicrobial effect, other than its possible influence on water activity. (Davidson, M. P. et al.; 2005: 176)

He further states that by the end of the 1960's nitrite was recognized as an effective antimicrobial agent, but its value as a preservative in perishable meat was still in doubt. The majority of studies focused and proved its effectiveness in shelf-stable canned meat. (Davidson, M. P. et al.; 2005: 176)

Brine content was shown to be an important factor in botulinal outgrowth and toxin formation. (Davidson, M. P. et al.; 2005: 177)

Following 1960, the focus shifted towards the role of nitrite in the total inhibitory system in cured meat. (Davidson, M. P. et al.; 2005: 177)

In 1962, Eddy and Ingram investigated "the survival of *S. aureus* in vacuum packed, sliced bacon. They found that staphylococci grew among the natural microflora of the bacon but growth was better when the number of saprophytic microorganisms was low and the storage temperature was high. (Doyle, M. 1989. : 476)

Gould (1964) showed that the toxicity of nitrite was 3 to 5 times greater at pH 6 than at pH 7. (Davidson, M. P. et al.; 2005: 177)

Brownlie (1966) indicated that at pH 7.0, the presence of nitrite caused very little or no inhibition. At pH 6.0 and below, increasing the amount of nitrite from 25 to 200 µg/g caused progressively greater inhibition. (Davidson, M. P. et al.; 2005: 178)

Brownlie (1966) has shown that nitrite was more inhibitory at 0°C than at the other temperatures tested (10°C and 25°C) Several studies showed that salt becomes more inhibitory as storage temperatures are decreased in perishable vacuum packed cured meat. (Davidson, M. P. et al.; 2005: 177)

Brownlie (1966) showed the inhibitory effect of sodium nitrite concentration, pH and temperature. Brine content was shown to be an important factor in botulinal outgrowth and toxin formation. (Davidson, M. P. et al.; 2005: 177)

According to studies by Riemann Anon (1968), *C. botulinum* type A, the most toxic form, seemed to be completely inhibited by 4.5% brine at pH 5.3, 5.5% brine at pH 6.1, and 8.6% brine at pH 6.5. (Davidson, M. P. et al.; 2005: 180)

Studies by Baird-Parker and Baillie (1974) indicated that when adding sodium nitrite and L-ascorbic acid as filter-sterilized solutions, the number of strains showing growth in broth was found to decrease with increasing nitrite (50, 100, 150, 200 µg/g), decreasing temperature (25°C, 20°C, 15°C), decreasing pH (7.0, 6.5, 6.0, 5.5), increasing salt (1.5%, 3.0%, 4.5%, 6.0% w/v), and decreasing inoculum level (106, 103, 101). Adding L-ascorbic acid (1.0%) markedly increased the effectiveness of nitrite. (Davidson, M. P. et al.; 2005: 180)

Adding hemoglobin resulted in a lower level of residual nitrite after processing, decreasing botulinal inhibition. (Davidson, M. P. et al.; 2005: 181)

Tompkin et al. concluded that Isoascorbate, ascorbate, cysteine, and ethylenediaminetetraacetic acid (EDTA) share a common function in meat, which later was demonstrated to be the sequestering of iron. (Davidson, M. P. et al.; 2005: 180)

Grever (1974) indicated that *Bacillus* species are less sensitive to nitrite than clostridia. (Davidson, M. P. et al.; 2005: 187)

Tompkin et al (1979) also showed that although isoascorbate enhances the antibotulinal effect of nitrite in freshly prepared perishable cured meat that is temperature abused, isoascorbate also reduces the efficacy of nitrite by causing more rapid depletion of residual nitrite. (Davidson, M. P. et al.; 2005: 187)

According to Crowther et al. (1976), studying mixtures of nitrite, nitrate, ascorbate and brine levels and their effect on botulinal toxins in vacuum packed back bacon, a higher percentage of samples analysed were toxic with the addition of 200 µg/g of nitrite than with 100 µg/g of nitrite. The addition of ascorbate enhanced the antibotulinal effect of 100 µg/g but not 200 µg/g of nitrite. These values raise a question concerning the conclusions that (1) protection was greater if the level of nitrite was increased to 200 µg/g and (2) sodium ascorbate at a level up to 2000 µg/g did not reduce the protection afforded by nitrite against *C. botulinum*. (Davidson, M. P. et al.; 2005: 187)

Crowther et al. (1976) also reported that *S. aureus* grew well in the medium-salted bacon, regardless of the level of nitrite or ascorbate. (Davidson, M. P. et al.; 2005: 189)

Shaw and Harding (1978) studied the effect of nitrate and nitrite on the microbial flora of Wiltshire bacon. The predominant flora of the bacon after curing consisted of micrococci, *Moraxella* species, and *Moraxella*-like bacteria. Omitting nitrate led to higher numbers of *Moraxella* species in the cured bacon. However, bacon that was sliced and vacuum packaged developed a flora mainly of micrococci and lactics. Including nitrate in the bacon enhanced the growth of micrococci. (Davidson, M. P. et al.; 2005: 189)

Shaw and Harding (1978) showed that because higher numbers of lactics were present in bacon with the lowest initial nitrite concentration, it was suggested that nitrite could be important in delaying the sour spoilage caused by the growth of lactics. (Davidson, M. P. et al.; 2005: 189)

Various botulinal studies were conducted in the USA in the 1970's. It showed that vacuum-packaged bacon prepared with 0.7% sugar (sucrose) or more provides sufficient fermentable carbohydrate that naturally occurring lactics cause a decline in pH to inhibitory levels. (Davidson, M. P. et al.; 2005: 190)

The botulinal studies in the '70's also showed that brine levels below 4.0% are not inhibitory to botulinal outgrowth. As the brine level exceeds 4.0%, outgrowth is increasingly delayed. If a lactic fermentation develops in the interim, the combination of relatively higher brine and decreasing pH can prevent botulinal outgrowth. (Davidson, M. P. et al.; 2005: 190)

These same studies showed that the level of residual nitrite at the time the bacon is abused influences the extent of the delay in botulinal outgrowth. The level of nitrite added to the product is not important, aside from the fact that the amount of added nitrite partially determines the level of residual nitrite. (Davidson, M. P. et al.; 2005: 190)

It also showed that the addition of ascorbate or isoascorbate can act in concert with residual nitrite to retard botulinal outgrowth in freshly produced bacon. However, ascorbate and isoascorbate can also have a negative effect by causing more rapid loss of residual nitrite during processing and storage. (Davidson, M. P. et al.; 2005: 190)

Nurmi and Turunen (1970) studied the effect of adding nitrite to a previously autoclaved broth medium (pH 6.0). Lactobacilli (78 strains), micrococci and staphylococci (24 strains), and *Pediococcus cerevisiae* (1 strain) were examined for their tolerance to nitrite in the presence and absence of 4.01% salt. At 200 µg/g growth was delayed or slower. At 40 µg/g growth was comparable to that in the control without nitrite, results were subsequently reported that showed the production of enterotoxin A to decrease as pH decreased, salt increased, and nitrite increased (Tompkin et al., 1973).

Morse and Mah (1973) studied the effect of glucose on enterotoxin B synthesis in a broth medium buffered to an alkaline pH (7.7). Adding glucose caused decreased toxin production. Glucose repression of enterotoxin B production was also reported to occur at pH 6.0 but to a lesser degree than at pH 7.7 (Morse and Baldwin, 1973).

Bean and Roberts (1974, 1975) The inhibitory effect of nitrite in the recovery medium increased with increasing salt content, decreasing incubation temperature, and decreasing pH. (Davidson, M. P. et al.; 2005: 190)

Zeuthen (1980) conducted studies on the effect of pH on the rate of microbial growth in sliced ham. They found that the lower pH meat resulted in a ham with a pH of 6.0 with

residual nitrite after processing and the higher pH meat resulted in a ham with a pH of 6.35 with a higher residual nitrite level. The brine level of both products were equal. During 7 – 8 weeks of storage at 5 deg C, the rate of microbial growth was considerably slower in the sliced ham prepared with the lower pH meat. (Davidson, M. P. et al.; 2005: 203)

In the 1980's, the USDA adopted a regulation for bacon that requires a maximum of 120 µg/g sodium nitrite and the addition of 550 µg/g sodium ascorbate or isoascorbate. (Davidson, M. P. et al.; 2005: 203)

It was also shown during this period that the mechanism of nitrite inhibition differs in different bacterial species. (Davidson, M. P. et al.; 2005: 203)

In 1988, the USDA initiated a series of increasingly restrictive policies on the rate of chilling for perishable cured meat manufactured under USDA inspection. Dr Tompkin continues that this is a case where the epidemiologic data indicate a negligible public health concern for cured meats but the evidence from challenge studies and predictive modeling suggests otherwise. He notes that the situation is a reminder of Morris Ingram's frustration with the increase in research on nitrite's role in botulinal inhibition in the 1970's. At the time he stated, "What we need at the present time, in my opinion, is not more inoculated pack experiments but a rationale for interpreting them" (Ingram, 1974).

"Since 1990 there has been increased interest of *L. monocytogenes* in ready-to-eat foods. McClure et al (1991) found the efficacy of sodium nitrite to be temperature and pH dependent. At a pH value of 6.0 sodium nitrite had little effect in delaying the time to detect visible growth except at the highest level tested (200 ppm) and a temperature of 15 deg C or below. At pH 6.0 and 5 deg C no growth was observed with any of the levels of sodium nitrite evaluated (50, 100, 200, 400 µg/g). Buchanan and Golden, 1995; Buchanan et al., 1997) conducted an extensive series of experiments that led to the conclusion that nonthermal inactivation of *L. monocytogenes* by sodium nitrite is pH dependant and related to the concentration of undissociated nitrous acid. (Davidson, M. P. et al.; 2005: 203)

Duffy et al. (1994) inoculated a variety of vacuum-packaged cooked sliced meat with *L. monocytogenes* and found the lag time increased and the rate of growth decreased at 0 deg

C and 5 deg C with the addition of sodium nitrite (0 to 315 µg/g). The effectiveness of sodium nitrite was significantly increased with the addition of sodium ascorbate. (Davidson, M. P. et al.; 2005: 203)



Applications to our bacon curing

We skipped over the entire N-Nitrosamine issue and the possibility that nitrite in bacon is carcinogenic. According to Dr. Tompkin, the fear of N-nitrosamine formation in the early 1970's and 1980's is probably the main reason why bacon curers in the USA opted to leave nitrate out of curing brines and use only nitrite. (from private correspondence) We will return to the nitrosamine issue in a future article.

Lets first have a look at legislation in South Africa and then draw some practical applications that can be applied in the bacon curing plant.

SA REGULATIONS

The South African max allowed limits on nitrite, nitrate and some of the chemicals mentioned in our survey are:

from Regulation R965 of 1977(18):

- Potassium and sodium nitrate: 200mg/ kg
- Potassium or sodium nitrite: 160mg/kg

Where nitrate and nitrite are used in combination they must be added together and proportionally neither one can exceed the max limit (section 2b of Regulation R965 of 1977).

For using erythroic acid or sodium erythroate: 550 mg/kg

L Ascorbic Acid: 550 mg/kg.

One other matter must be considered before we make our applications and that is the status of bacon. Is it a cooked (ready-to-eat) or a raw product? This is an important point since uncooked products assume that heat will be applied as a final barrier before consumption. In the case of a ready-to-eat product, these are produced in such a way that it assumes no further barrier before consumption.

In South Africa bacon is not a cooked product. It is similar to the situation in the USA where “commercial bacon is cooked and smoked to an internal temperature of about 128F (53.3C). It is not considered ready-to-eat as in some European countries where a trichinae control program has been in place for about a century.” In South Africa, as in the USA, bacon is cooked before it is consumed. (private communication with Dr. Tompkin) This means that bacon is handled as a low risk product from a food safety perspective.

Now that we have the proper legal position of bacon and the maximum allowed limits of nitrate, nitrite and some chemicals often associated with them we can move on to our list of applications.

POINTS OF APPLICATION

Here are a few practical applications that flows from the consideration of nitrite and nitrate in bacon.

- We suggest a combination of nitrate and nitrite with a maximum of salt (as much as is palatable).
- An important economic and food safety consideration is shelf life. In order to extend shelf life, good manufacturing practices, a thorough food safety program and using the correct heat, freezing and pH during processing are as important as antimicrobial chemicals. Some argue that these may have the ability to replace most antimicrobials in food. An example of this is the contention that much of the improved shelf life in the US on bacon and poultry products is “attributed to improvements in sanitation between cooking and packaging as a requirement to control *Listeria* contamination”. (private communication with Dr. Tompkin)

- Reduce the pH in the meat. We suggest manipulating the pH of the meat to levels of between 5.6 and 5.8. Not below 5.3 since reducing the pH will increase the rate of nitrite depletion (private communication with Dr. Tompkin) and 5.3 has been shown to be a threshold.
- Use nitrate, nitrite and salt in combination with a low temperature, targeting an internal core temp of between 50 and 65 deg C for at least 30 minutes.
- The goal of keeping the meat temperature below 5 deg C from receiving of meat till before smoking/ cooking and then rapid chilling and freezing and keeping the finished product below 5 deg C is an excellent way of increase the lag time and the reduce the rate of growth of *L. monocytogenes*. As a general policy, meat must be kept below this during processing.
- Related to the greening of bacon. “Greening is due to the growth of certain other lactobacilli which also occur on cured meats and is a very old problem. It is a major problem at times if cooked product is held in storage allowing for the lactobacilli to multiply and then the product is used as rework into new product. Over time the repetitive addition of aged rework leads to a high population of lactobacilli that are exceptionally heat resistant. They are microaerophilic meaning they can not tolerate much oxygen and grow well under the perimeter of sausages or in vacuum packaged meats. Upon opening the packages the product turns green.” (private communication with Dr. Tompkin)

Another reason often cited for a green discolouration in cured meat is nitrite burn. It is caused by a combination of excessive levels of nitrite and reduced pH (Deibel and Evans, 1957). The levels that nitrite is used in cured meat is so low that greening in bacon is unlikely to occur as a result of nitrite and reduced pH. (private communication with Dr. Tompkin)



Conclusion

Nitrite's role in cured meat is far more than only colour and taste. It is a key component of a very complex environment with definite antimicrobial efficacy. It is an effective hurdle against *Clostridium butulinum*. Its antimicrobial efficacy extends to other organisms, the level of which differs from organism to organism. It is definitely an important general antimicrobial hurdle.

Regarding nitrate, enough early research has been done that show efficacy if its used in conjunction with nitrite and salt to warrants its inclusion in brine curing mixes.

The efficacy of nitrate and nitrite is strongly tied to brine content, pH, heat treatment and adding complementary chemicals.

The story of saltpeter (potassium nitrate) and sodium nitrite is epic in the true sense of the word.

(c) eben van tonder

“Bacon & the art of living” in book form

Stay in touch

Like our Facebook page and see the next post. [Like](#), [share](#), [comment](#), [contribute!](#)

Bacon and the art of living



[Promote your Page too](#)

Notes:

(1) Dr. Tompkin is retired and currently associated with the [School of Applied Technology](#) as part of the [Illinois Institute of Technology](#). For his background, see <https://appliedtech.iit.edu/people/bruce-tompkin>.

References

Davidson, P. M. et al. 2005. Antimicrobials in Food, Third Edition. CRC Press.

Doyle, M. 1989. Bacterial Pathogens. Marcel Dekker, Inc.

Emmeluth, D. 2010. Botulism. Infobase Publishing.

Jay, M. J. et al. 2005. Modern Food Microbiology. Springer Science + Business Media.

The Centralia Enterprise and Tribune. Centralia, Wisconsin. 25 January 1896.

The Laredo Times. Laredo, Texas. 1 December 1896.

Maple Leaf Press release: <http://investor.mapleleaf.com/phoenix.zhtml?c=88490&p=irol-newsArticle&ID=1363993&highlight=>

McCarthy, M. Chairman of the Committee of nitrite and alternative curing agents in food. Et al. 1981. The Health Effects of Nitrate, Nitrite, and N- Nitroso Compounds. National Academy Press.

Montclair Tribune. Montclair, California. 20 April 1972.

Sterba, J. P.. 28 April 1982. The History of Botulism. The New York Times.

<http://medical-dictionary.thefreedictionary.com/Clostridium+putrificum>

Images

Image 1: Clipping from newspaper article: Montclair Tribune (Montclair, California), 20 April 1972.

All other images by Willem Klynveld.