Nitrite Additives—Harmful or Necessary?

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INTRODUCTION

The use of nitrites in the preparation of smoke-cured fishery products and processed meat products dates back to 1925. Prior to that, dating back to the early beginnings of the curing process, nitrates, usually containing nitrite impurities, were used. Nitrites are used in the curing of meats to impart red color, to enhance flavor, and to protect the product from the growth of *Clostridium botulinum* in case of contamination or mishandling. In the curing of fishery products, the addition of sodium nitrite ($NaNO_2$) prior to smoking to inhibit the growth of *C. botulinum*, thereby preventing formation of its toxin, is of primary importance. In recent years, the use of nitrites has been viewed with suspicion, and the continued use of $NaNO_2$ in the food industry has become a matter of controversy. The questions arising from the use of nitrites are: 1) Is their continued use necessary for the production of a product safe from deadly bacterial toxin? 2) Is their presence responsible for the occurrence and formation of highly potent carcinogens in foodstuffs and/or in the bodies of the consumers of these foodstuffs?

In recent years, the occurrence of trace amounts of *N*-nitrosamines has been indicated in many cured meat products (Fazio, White, and Howard,

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1971; Sen, 1972; Fazio et al., 1973; Panalaks, Iyengar, and Sen, 1973; Panalaks et al., 1974), cured fishery products (Ender and Ceh, 1968; Sen et al., 1970; Fazio, Damico, Howard, White, and Watts, 1971; Sen et al., 1972; Kawabata et al., 1973; Gadbois et al., 1975), in addition to other products such as cheese (Sen, Smith, Schwinghamer, and Marleau, 1969), soybean oil (Hedler et al.[1], 1971), milk (Hedler and Marquardt, 1968), alcoholic beverages (McGlashan, Walters, and McLean, 1968), and tobacco (Hoffman et al., 1974). They have also been found to occur in vivo. For a variety of animals tested in feeding studies, many of these compounds have been shown to be highly potent carcinogens (Preussmann[2], 1971; Swann and Magee, 1971; and Preussmann, 1974). The danger of these *N*-nitroso-compounds to human beings has not yet been established, as there is no direct evidence of their carcinogenicity in man. Although much remains to be learned concerning the chemistry of these compounds, they are known to form in foods and in the various organs of animals under favorable conditions of pH, time, and temperature from the reaction of secondary and tertiary amines with

[1]Hedler, L., H. Kaunitz, P. Marquardt, H. Fales, and R. E. Johnson. 1971. Detection of N-nitrosocompounds by gas chromatography (nitrogen detector) in soybean oil extract. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.
[2]Preussmann, R. 1971. On the significance of N-nitrosocompounds as carcinogens and on problems related to their chemical analysis. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.

nitrite (Epstein[3], 1971; Hill[4], 1971; Sander and Schweinsberg[5], 1971; Cantoni, Renon, and L'Acqua, 1974; Myslivy et al., 1974). Nitrosamines have been isolated from gastric juices of animals fed these precursors and under simulated human stomach conditions (Walters, Saxby, and Newton[6], 1971; Lane, Rice, and Bailey, 1974).

The presence of several amines such as methylamine, di- and trimethylamine, di- and triethylamine, *N*-propylamine, and *N*-butylamine has been reported in a variety of fishery products (Gruger, 1972). Their occurrence, primarily a result of bacterial and enzymic action, is known to vary considerably, quantitatively and qualitatively, depending on such factors as the species and the age of the fish, as well as storage and handling conditions. They may also arise during cooking. Freshwater species of fish of

[3]Epstein, S. S. 1971. In vivo studies on interactions between secondary amines and nitrites or nitrates. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.
[4]Hill, M. J. 1971. Bacterial production of nitrosamines in vitro and in vivo. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.
[5]Sander, J., and F. Schweinsberg. 1971. In-vivo and in-vitro experiments on the formation of nitrosocompounds from amines and amides and nitrate on nitrite. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.
[6]Walters, C. L., M. J. Saxby, and B. E. Newton. 1971. Nitrosation under simulated stomach conditions. Presented at the International Agency for Research on Cancer and the Deutshes Krebsforshungszentrum Joint Meeting on the Analysis and Formation of Nitrosamines.

the Great Lakes region, often used in the preparation of smoked fishery products, have higher amine contents than do marine fishes. If it is proven that the simultaneous consumption of these amines and nitrites present a real hazard to our health, then all possible steps must be taken to eliminate this hazard. By no means would it be possible for us to eliminate these amine compounds from the foods we eat. We can, however, to some extent, control the amount of nitrite we consume—at least that amount which is not a naturally occurring constituent of food.

The U.S. Food and Drug Administration (FDA) has issued regulations on the safe use of $NaNO_2$ for the preservation of smoked fishery products. As early as 1925, the U.S. Department of Agriculture (USDA) imposed an upper limit of 200 ppm residual sodium nitrite in any cured meat or fish product as it was known since the early part of this century that high doses of sodium nitrite had a toxic effect on humans causing methemoglobinemia, the result of nitrite combining with the hemogloblin of blood. Public concern and pressure for stronger and more effective food and drug control laws resulted in the enactment of the Federal Food, Drug, and Cosmetic Act of 25 June 1938. This legislation increased regulatory control over toxic substances added to food; but as the use of new food additives mushroomed, it became apparent that this law, too, was inadequate. In 1958, as a result of a study conducted by the Congressional Delaney Committee, the Food Additives Amendment of 1958 was passed. This amendment required FDA preclearance of all new food additives for safety before marketing. Prior to the passage of this amendment, when adulteration of food was charged, FDA was legally obliged to prove that the substance was poisonous; that it had been added; and that it was unnecessary in the production of the food. The Food Additives Amendment changed this situation. It shifted the burden of proof from FDA to the manufacturer, distributor, or user of a new food additive who was required to prove its safety. Under the amendment, FDA is responsible for determining the safety of all food additive uses, publishing regulations,

approving such uses, and establishing safe limits referred to in the law as "tolerance limitations."

A significant feature of the Food Additives Amendment is the Delaney Clause which is intended to protect the consumer from the potential hazards of ingesting carcinogenic chemicals. FDA is prohibited by this clause from approving the use of any food additive which has been found to induce cancer when consumed by man or animal. FDA may approve a proposed use of an additive after evaluating the data submitted to it and finding that there is no doubt that the proposed use would accomplish the "intended physical or other technical effect," that is, the effect the additive may have on the sensory or other aspects of the food. The task of the FDA, in its consideration of whether the additive produces the desired effect and how much of the additive is required to produce the intended effect, does not include judging whether the effect increases the value of the product to the consumer or improves its marketability. Significantly, the amendment does not delegate to the FDA the authority to balance the benefits of a food additive against the risks associated with its use in deciding whether to approve the use. The FDA is expected only to consider whether the additive produces the intended effect and whether it is safe for human consumption.

In January 1971, the Intergovernmental Relations Subcommittee began an investigation to determine the adequacy of the protection being afforded consumers by the FDA and the USDA against potentially harmful food additives. The investigation was undertaken because of widespread public concern about the potential dangers of food additives. In March of that year, hearings focused on additives of current scientific concern such as nitrites and nitrates because of their possible conversion in food or in the body to cancer-producing nitrosamines and nitrosamides making them a potentially serious health hazard. To date, there is no conclusive evidence that nitrosation, the formation of nitrosamines from their precursors, in the human stomach causes cancer; however, cancer has been produced in

test animals which had been simultaneously fed nitrites and certain secondary amines. Malignant tumors were produced in rats by a dose as low as 2 ppm of diethylnitrosamine (Committee on Government Operations, 1972). To date, a "no-effect level" has not been established for this compound. A German study (Sander and Seif, 1969), in which nitrate and the secondary amine diphenylamine were fed concurrently to human volunteers, demonstrated that both nitrosation and the reduction of nitrate to nitrite were occurring in the human body. Formation of nitrosamines in human gastric juices due to ingestion of precursors has also been demonstrated by other investigators (Sen, Smith, and Schwinghamer, 1969; and Magee, 1971). In no instance, however, was carcinogenesis established.

Nitrates and nitrites received USDA approval for use in the curing of meats years before passage of the Food Additives Amendment and were, therefore, exempt from its provisions because of "prior sanctions." However, prior sanctions may be revoked and regulatory action taken against the use of nitrates and nitrites. For food other than meats, no such prior sanction exists, and the use of nitrates and nitrites in such foods must be precleared and approved for use by FDA.

The Delaney Clause prohibits FDA from approving any food additive "if it is found to induce cancer when ingested by man or animal." Application of this clause to the nitrite situation is complicated by the fact that nitrites per se have not been found to produce cancer but rather when ingested simultaneously with certain secondary and tertiary amines have produced cancer in animals. It would appear then that the Delaney Clause is technically and legally not applicable to nitrites if a reaction takes place between nitrites and secondary amines in a man's stomach but does become applicable if it can be demonstrated that nitrosamines are produced in the meat or fish from the added nitrite and these nitrosamines are consumed.

The discovery prior to 1925 that nitrites would more efficiently produce the intended effect, namely color fixation and enhancement in meat, led to the petition of USDA by several

meat establishments to experiment with the use of nitrites. As a result of these experiments, the limit or tolerance of 200 ppm of nitrite was set. This limit was based on the maximum nitrite content found in the cured meats produced during these experiments, and it in no way pertains to the minimum quantity necessary to accomplish the intended effect. There is no justification whatsoever for this limit. A most important question to be answered then is how much nitrite is actually needed to accomplish its purpose. As little as 10 ppm of nitrite, for example, suffices to impart acceptable flavor and color to bacon; however, from a bacteriological viewpoint, this level may be inadequate.

In view of the findings of the subcommittee, the evidence that nitrites play an important role in preventing the occurrence of botulinum poisoning in meats is not exceptionally strong except in special cases such as the larger-sized canned hams. The 3- and 5-lb cans may require the addition of nitrite because the heat penetration necessary to destroy botulinum spores in meat may be difficult to achieve.

A summary (Center for Disease Control, 1974) of botulism outbreaks during the period 1899-1975 revealed only 11 (possibly 10) instances in which meat was the cause of botulinum poisoning. Beef was the vehicle in four outbreaks (in one outbreak, traced to beef stew, the actual source is unknown), pork in three, poultry in three, mutton in one, and venison in one. All of these were caused by types A and B toxins with the exception of the venison-caused outbreak which was traced to the rare type F. There has never been any reported instance of botulism attributable to fresh meat. Here the potential might seem to exist since beef is often aged at controlled temperatures for varying periods of time before cooking or curing. However, most meats are cooked prior to consumption destroying the botulium toxin and ensuring the safety of the food. The toxin is easily destroyed by heat, therefore any meat which must be cooked cannot present a hazard to the consumer. Bacon, then, would be considered a typical example.

William Lijinsky of the Carcinogenesis Program at Oak Ridge National Laboratory has done considerable research in the area of nitrosamine occurrence and detection. He is critical of the present use of nitrites stating that the permitted level of 200 ppm of nitrite leaves no margin of safety. Lijinsky has not called for a ban on the use of nitrites in the curing process but rather urges that the permitted levels of residual nitrite be brought into line by carefully determined needs rather than to be set by an arbitrarily chosen amount. If the amount of residual nitrite varies so considerably in a finished product and if the amount is so often found below 50 ppm and found to be effective at these low concentrations, why then can't lower limits be established and enforced?

More than 6,500 samples of nitrite-treated meat were tested by the USDA during the period 1 January 1970, to 31 January 1971. More than 79 percent of these samples were found to have 25 ppm or less, and 92 percent contained 50 ppm or less. Actually, the problem of how much nitrite is needed in the curing of a product and the determination of the residual nitrite that remains in the product after curing and storage are complicated, to say the least. Residual nitrite is only that amount of nitrite which can be detected by current analytical techniques. It is a superficial description and in no way explains what became of the nitrite which was incorporated into the product at the time of curing but which, during storage, "disappeared." A number of reaction pathways have been well established for the removal of free nitrite in meat products (Cassens et al., 1974); however, total losses still can't be accounted for. To be concerned with the free nitrite which might combine with amines to form nitrosamines is to be concerned with only a portion of the problem. What, for example, is the "Perigo inhibitor" (Perigo, Whiting, and Bashford, 1967)? This unknown inhibitor for the growth of several species of *Clostridium* has been found to form when certain culture media are heated in the presence of $NaNO_2$, and its inhibitory activity differs from that of nitrite alone.

If nitrites are being used with concomitant formation of nitrosamines when no danger of botulism exists or if it is found that the nitrites are being added not as a preservative but only to fix color, then it apears unquestionable that their use should be prohibited. If, on the other hand, levels of nitrite can be established which cause no nitrosamines to be formed in the product, then the use of nitrites should not be prohibited by a sweeping generalization; i.e., assuming it can be shown that there is no significant formation of nitrosamines in the stomach after ingestion of these nitrites. The FDA initiated formal action in November 1972, to ban the unnecessary and hazardous use of nitrites, an action which would seriously affect the smoked fish industry. If finalized, this action would put a stop to the use of sodium nitrite in smoked-cured sablefish, salmon, and shad, sodium nitrite in smoked tuna fish products, and potassium nitrite in cod roe. Allegedly, the additives in these instances are being added primarily as preservatives.

Following the passage of the Food Additives Amendment of 1958, the FDA imposed regulations pertaining to the use of nitrate and nitrite in smoked and cured fish products. The following regulations (Committee on Government Operations, 1972) were published.

1) Regulations 21 CFR 121.1063 and 121.1064 which permit the use of sodium nitrate and sodium nitrite in amounts not to exceed 500 parts per million of nitrate and 200 parts per million of nitrite in smoked and cured salmon, sablefish, and shad, and in home-cured meat, poultry and game, primarily for color fixation and secondarily for preservation.

2) Regulation 21 CFR 121.1132 permits the use of potassium nitrate as a curing agent in the processing of cod roe in an amount not to exceed 200 parts per million of the finished roe.

3) Regulation 21 CFR 121.223 permitting the use of sodium nitrite as a preservative and color fixative in canned pet food containing fish and meat provided that the level of sodium nitrite does not exceed 20 parts per million.

4) Regulation 21 CFR 121.1064 permitting the use of sodium nitrite as a preservative and color fixative in smoked and cured tuna fish products, provided that the level of sodium nitrite does not exceed ten parts per million in the finished product.

5) Regulation 21 CFR 121.1230 permitting the use of sodium nitrite in processing smoked chubs to aid in inhibiting the outgrowth of toxin formation from *Clostridium botulinum* type E, provided that the sodium nitrite content of the edible portion of the finished smoked product is not less than 100 parts per million and not greater than 200 parts per million as measured in the loin muscle.

Regulation (5) went into effect in August 1969. Before that date, chubs were prepared without the use of nitrites because it use was not permitted. However, after three outbreaks of botulism which were attributed to smoked chubs and which caused a number of deaths, the Bureau of Commercial Fisheries under the U.S. Department of the Interior petitioned FDA for a regulation to allow the use of $NaNO_2$ in chubs. A regulation was published which requires chubs to contain not less than 100 ppm of sodium nitrite for effective preservation against botulism and permits a maximum of 200 ppm. This regulation also carries requirements for heat processing and an adequate content of NaCl, both of which have a preservative effect. If such a level correctly reflects the minimum nitrite content necessary for protection against botulism, the effectiveness of the low levels of nitrite found by the USDA in meats as a prevention against botulism becomes questionable. Actually, the level 100 ppm is in itself open to question and closer examination may indicate a lower level of nitrite may be sufficiently effective.

Whereas FDA's survey of the meat industry revealed a preponderance of samples of low $NaNO_2$ content, i.e., much less than 200 ppm, this was not commonly the case with fish samples analyzed. In a 1969 preliminary survey conducted by FDA to determine the distribution of sodium nitrite in cold, smoked fish, six plants were inspected. Three of these firms were found to have produced smoked salmon with sodium nitrite contents in excess of 200 ppm. A 1970 survey also revealed some smoked chub samples with excessively high nitrite concentrations. This is not meant to imply that this situation is typical of most smoked fish producers. Rather, it is intended to illustrate that violations of the regulations do exist,

and it is these violators who are exacerbating the problems in the use of nitrite additives for the conscientious processor of smoked fish. Violations become more common with insufficient monitoring and laxity in regulatory action. Though the maximum permitted tolerance for sodium nitrite is 200 ppm in smoked and cured fish, FDA has established 260 ppm as the action level for smoked chub. That is, seizure or other action involving smoked chub containing excess sodium nitrite will not be authorized unless the level is at least 260 ppm. The 1970 survey also revealed smoked chub samples with nitrite levels far below the minimum level of 100 ppm. Though such small concentrations of nitrite were not likely to have any significant preservative effect in the control of botulism, FDA instituted no action against any of the producers though they were in direct violation of the regulations published by the FDA.

If a safe product is to be offered to the consumer, it may be that the use of nitrites is needed. The outbreaks of botulism due to smoked fishery products are not common, but also they are not rare. A review of botulism outbreaks during the period 1899-1973 revealed the occurrence of 688 outbreaks involving a total of 1,784 cases with 978 reported deaths. Four hundred and ninety-five (72 percent) of these outbreaks have been traced to home processed foods. Only 62 (9 percent) outbreaks have been caused by commercially processed foods. The source of food responsible for the remaining outbreaks is unknown. As a vehicle responsible for botulism outbreaks, fish is relatively more important than meat. During the period 1899-1973, fishery products have been responsible for 4.4 percent of the total outbreaks, beef 0.9 percent, pork 0.4 percent, and poultry 0.4 percent. It is generally assumed that if botulism is caused by a marine product, type E toxin is responsible; but of the 29 outbreaks caused by fish products up to 1973, 19 were due to type E, 7 were due to type A, and 3 to type B.

The first reported outbreak of botulism in the U.S. due to fishery products occurred in 1932 and was caused by salmon caught and smoked in Labrador. At that time, type E

strain had not yet been identified. However in 1935, type E was isolated from Russian sturgeon and was found to be identical to that strain isolated from the salmon of the 1932 outbreak and also from some imported German canned sprats which were the cause of a second outbreak in 1934.

It was in the early sixties that attention was focused on the smoked, freshwater fish of the Great Lakes region. The first known cases of botulism caused by fish caught in the Great Lakes were reported in 1960 when commercially-processed, vacuum-packed, smoked ciscoes were found to be the cause of an outbreak in Minnesota. Type E toxin was isolated. Since 1960, commercially-processed, smoked fish from the Great Lakes region has been responsible for four outbreaks of botulism which have involved 22 cases resulting in 10 deaths. In 1963, Michigan was the location of an additional outbreak of type E botulinum; but in this case, three persons were stricken after eating commercially packed, canned tuna from California. In 1963, this region was particularly hard hit. Smoked whitefish, vacuum-packed in Michigan and shipped to the adjoining states of Tennessee, Alabama, and Kentucky was the cause of 17 cases, in which 5 persons died. In this same year, two additional outbreaks occurred in Michigan and Minnesota, also due to commercially processed, smoked whitefish. In 1967, commercially packed whitefish was the cause of an outbreak in Illinois.

Type E toxin shows a widespread distribution along both the Atlantic and the Pacific coasts and in the Great Lakes Region. Alaska is the leading state (12 outbreaks) reporting type E outbreaks which is due to the popularity of certain foods such as cheese eggs or fermented salmon eggs with certain groups of native Alaskans and the widespread practice of home-processing. Up to the present, 32 outbreaks of botulism have occurred in the United States which can be attributed to fish and seafood.

Although the need for nitrite in smoke-cured fish as a means of preventing botulism has been stressed, the bacteriostatic action of nitrite is not firmly established. It is not mandatory

that processors of cured, smoked fish use nitrites in their product to prevent botulism. Sodium chloride in the concentration range 2.5 percent-5.8 percent has been reported to inhibit outgrowth and toxin production by *C. botulinum* type E (Abrahamsson, Gullmar, and Molin, 1966; Segner, Schmidt, and Boltz, 1966; Christiansen et al., 1968; Emodi and Lechowich, 1969; Lechowich, 1970). However, inhibition of outgrowth of types A and B spores requires a sodium chloride concentration of 8.2 percent-10.5 percent (Lechowich, 1970). It is generally agreed if the 180°F for 30 minutes requirement is met and the fish is subsequently distributed under adequate refrigeration with proper sanitation, there would be no need for the use of $NaNO_2$. The complaint of industry is, however, that they are not equipped to use that alternative and, moreover, use of this alternative results in a product showing excessive thermal damage. Also, the possibility that heat resistant mutants may evolve must not be overlooked. In 1953, a team of investigators (Dolman and Chang, 1953) reported survivors among type E spores heated at 212°F (100°C) for 30 minutes. A second alternative would be to heat the product to an internal temperature of 150°F for 30 minutes and have a minimum of 5 percent salt in the water phase. It is generally felt, however, that this produces a product that is too salty. (This is the problem which now confronts the meat and fish industries.)

The controversial status of nitrites during recent years has resulted in extensive testing of a variety of meat and fishery products by a number of institutions, government and academic, and private industry for nitrosamine occurrence. Analyses of various cured meat products have disclosed the presence of nitrosamines in many. A concentration of dimethylnitrosamine (DMNA) has been detected in some ham samples at the 5 parts per billion (ppb) level (Fazio, White, and Howard, 1971). In other samples, it was nonexistent.

Investigators of the Food Research Division in Ottawa have made a study of numerous cured meat products with regard to their nitrite and nitrosamine contents. In one study (Panalaks, Iyengar, and Sen, 1973), 197 samples of various kinds of meat products were tested. Of these, 57 samples indicated trace amounts (2-12 ppb) of DMNA. These included such products as ham spread, luncheon meat, ox tongue, wieners, bologna, Polish and Ukranian sausage, salami, pastrami, and bacon. Others were negative. No mass spectrometric confirmation of the identity of DMNA was carried out so results should be considered as only tentative. In only a few types of meat products did the concentrations of nitrate and nitrite correlate with that of DMNA detected in the samples. In a later study by this same group (Panalaks et al., 1974), of a total of 80 samples of different kinds of cured meat products, 17 contained 13-105 ppb nitrosopyrrolidine (NP_y); 29 samples contained 2-35 ppb DMNA; and 9 samples contained 2-25 ppb diethylnitrosamine (DENA). In a few cases, identities were confirmed by gas-liquid chromotography (GLC) and mass spectrometry.

Scientists with the USDA and FDA have investigated the volatile nitrosamine content of bacon. High *N*-nitrosopyrrolidine concentrations in this product have been reported. The FDA investigators (Fazio et al., 1973) reported levels isolated from 8 commercial brands ranging from 10-108 ppb. This potent carcinogen was found in the cooked product but not in the raw form. Fat remaining in the pan after frying also contained nitrosopyrrolidine at levels ranging from 45-207 ppb. Since approximately 1.5 billion pounds of bacon are consumed each year, the presence of this nitrosamine in such a popular product deserves much consideration. Studies (Pensabene et al., 1974) investigating the effect of levels of sodium nitrite and cooking on the formation of NP_y indicate that the concentration of this nitrosamine increases with increasing nitrite levels. Samples which were pan fried showed the highest concentration of NP_y, while microwave-cooked samples showed the least. Concentrations of NP_y appeared to be dependent upon the frying temperature.

In a German study (Nagata and Mirna, 1974) which was carried out to determine the effects of processing conditions on the concentration of nitrosamines in meat products, it was concluded that nitrosamine formation was influenced mainly by heat treatment (sterilization, roasting, frying), but not by pH. No effects of bacterial fermentation, increased salting, or increased addition of pepper or paprika was observed. No increase in nitrosamine concentration attributable to dry salting was noted except in bacon which contained 45 ppm nitrosamines—mainly dimethyl-, diethyl-, piperidine, and pyrrolidine derivatives. No relationship was observed between residual nitrate or nitrite concentration and nitrosamine content. Addition of ascorbic acid or its sodium salt resulted in increased concentrations of nitrosopyrrolidine. This is in contrast to other studies which have shown that the addition of ascorbic acid has acted to decrease the amount of nitrosamines formed in nitrite-treated meat products (Mirvish et al., 1972; Kawabata et al., 1973; Fan and Tannenbaum, 1973; Fiddler et al., 1973; and Mottram et al., 1975).

The quantity of cured meat consumed in this country is high. Approximately 70 percent of all pork produced annually in the United States is cured. The per capita consumption of pork in 1974 was 66.5 pounds which means an average of 46.5 pounds of cured pork was consumed by each person. This is more than 90 times the amount of cured fish which is consumed. As a nation, we consumed about 2 billion pounds of wieners and bologna in 1973, plus 66 million pounds of smoked or dried beef, and 230 million pounds of cured beef.

The quantity of cured fish consumed per capita annually in this country is small compared to that of meat. Of the total per capita consumption of 12.0 pounds of all types of fish, only 0.5 pound of cured fish is consumed. This has not fluctuated greatly over the last 25 years, ranging between 0.4 and 0.7 pounds. This does not lessen the responsibility of the fish processor to insure a safe product to the consumer; however, it would seem to indicate that he could expect the same or equal judgment on his product as that which the processor of cured meat products receives.

Although cured fish has come under investigation the same as has cured

meat, the nitrosamine concentrations found in any species have been relatively low compared to those isolated and measured in some meat products. Investigators at FDA have isolated and confirmed by mass spectrometry levels of DMNA ranging from 4 to 26 ppb from samples of raw, smoked, and smoked nitrite- and/or nitrate-treated sable, salmon, and shad (Fazio, Damico, et al., 1971). Though no nitrosamine was detected in the raw salmon and shad, in the raw sable a level of 4 ppb was measured. In a separate study (Howard, Fazio, and Watts, 1970) conducted by this same group on smoked, nitrite-treated chub, such very low levels of 1-2 ppb detected (demonstrated sensitivity of method 10 ppb) led to the conclusion that DMNA was not formed under processing conditions in the nitrite-treated fish. Even fortification of the fish with large amounts of trimethylamine hydrochloride (1,500 ppm) and treatment with nitrite at high levels (2,500 ppm) prior to smoking did not cause an increase in the apparent amount of DMNA. Nitrosamine studies conducted at the Northeast Utilization Research Center of the National Marine Fisheries Service have also revealed the presence of only trace amounts (<10 ppb) of DMNA in cold-smoked sablefish to which had been added nitrite at levels ranging from 0 to 550 ppm (Gadbois et al., 1975). Concentrations of DMNA in these samples showed no apparent increase when samples were stored for two weeks at refrigerated temperatures (40°F). Twenty-four samples of various salmon roe products packed by Japanese producers were also investigated by this group. The presence of DMNA in all samples analyzed was too low for confirmation. Twenty-three commercially prepared salted Alaska pollock roe samples analyzed for nitrosamines by a Japanese team of scientists showed almost no DMNA (Kawabata et al., 1973). In most cases, it was below or around the detection limit of 1 ppb, except in one sample from which 6.7 ppb DMNA was detected.

The FDA has placed the use of nitrite in both hot-smoked fish and cold-smoked fish processes under interim food additive regulation until tests are completed to establish the safety and the need for the preser-

vative. The agency had been expected to ban use of nitrite in the hot-smoked fish process, but FDA officials granted an extension after concern was expressed that present temperature and other requirements may not be effective to control *C. botulinum* types A and B without the use of nitrites. The FDA believes that type E can be controlled without nitrites, but is not as certain about the more heat-resistant types A and B. Studies on the effect of nitrite on outgrowth and toxin production by *C. botulinum* in hot- and cold-smoked fishery products are being conducted by the National Marine Fisheries Service.

The formation of *N*-nitroso compounds in food products and in biological systems is not completely understood. A problem of nitrosamine research is the minute occurrence of these substances in food. Not only are these compounds elusive in behavior, but their detection is most tedious. Occurrences at the parts per billion level make their extraction and isolation necessarily exacting, and techniques currently being used could benefit from improvements. No doubt, many erroneous results have been reported, and initial findings should be confirmed. The problem of nitrites and nitrosamines today calls for vast amounts of additional research in all areas—detection, formation, and the establishment of tolerance limits.

Nitrite additives—harmful or necessary? They may be both, but a cooperative effort must be made by private industry and federal officials before a more comprehensive answer can be given.

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