Journal of Food Protection, Vol. 47, No. 2, Pages 119-121 (February 1984)

A Research Note

Residual Nitrite Concentration and Total Plate Counts in White and Dark Chicken Patties

ALFRED A. BUSHWAY* and KAI-WAN C. JEN

Department of Food Science, University of Maine, Orono, Maine 04469

(Received for publication June 13, 1983)

ABSTRACT

Effects of formulated nitrite (initial nitrite concentration added to patties) and muscle type on the residual nitrite concentration in raw and cooked chicken patties were studied. Microbiological determinations were done on raw chicken patties. Residual nitrite concentration in raw (104 ppm) and cooked (85 ppm) dark meat was higher than raw (90 ppm) and cooked (65 ppm) white meat after storage at 4 to 5°C. Cooking reduced the residual nitrite content of both white and dark meat. Formulated nitrite concentrations of 100 and 150 ppm lowered the total number of aerobic microorganisms developing in raw white meat patties by 10^2 and 10^4 colony-forming units/g, respectively, but were not effective in raw dark meat patties. A nitrite concentration of 400 ppm was required to repress the growth of aerobic microorganisms in raw dark meat for 6 d.

In recent years, there has been an increased consumption of convenience foods due to changing life styles and distribution patterns. In response to these changes, the poultry industry has developed a great variety of processed products such as 100% chicken frankfurters, bologna and salami. In many of these products, only one muscle type (white or dark) may be used.

Considerable research has been done on the fate of formulated nitrite in red meats. Goutefongea et al. (*4*) demonstrated that adipose tissue did not react with nitrite. Many investigators have shown that muscle pH will affect residual nitrite levels (*2,3,6,13*). The effect of processing, storage temperature and time on residual nitrite levels in red meat has also been studied.

In this study, we examined the effect of formulated nitrite and muscle type on the residual nitrite concentration and the total number of aerobic microorganisms in chicken patties.

MATERIALS AND METHODS

Sample preparation

Number 3 broilers (Maplewood Poultry Co., Belfast, ME) used in this study were obtained within 24 h of slaughter. Broilers were kept on ice before deboning which was done at 4°C.

Chicken breasts, drumsticks and thighs were hand deboned to produce white and dark meat separately. Skin, subcutaneous fat and bones were removed. White meat and dark meat were ground twice (1.27-cm plate followed by a 0.318-cm plate) using a Hobart grinder. Ground chicken (10 g) was homogenized in 30 ml of distilled water and the pH determined using an Orion Ionalyzer model 404. The remaining white and dark meat was divided into two equal parts and treated with 150 ppm nitrite (sodium salt, Sigma, St. Louis, MO). The nitrite was added in 3 ml of distilled water. Samples were mixed for 5 min in a Hobart mixer to obtain even distribution, and formed into 30-g patties. One-half of the white and dark meat patties was separated with patty papers, packed ten to a plastic bag to protect against dehydration, and stored at 4°C. The remaining patties were fried at 160°C for 2 min on each side, then packaged and stored as described for the raw patties. Fat and moisture loss were determined by weighing patties after cooling.

Chemical analysis

Determination of residual nitrite was done on days 0, 3 and 6 for raw patties and on days 0, 7 and 14 for cooked patties. Residual nitrite was determined colorimetrically (1).

Iron content of chicken white and dark meat was analyzed by inductively coupled plasma (ICP) spectroscopy using a Jarrell-Ash plasma comp 975. Procedures used were described in Jarrell-Ash bulletin 96-975. Six patties were analyzed at each sampling period.

Microbiological analysis

Total plate counts were determined on days 0, 2, 4, 6, 8, 10 and 12 on raw patties stored at 4°C. Three patties were randomly selected from each treatment (0, 50, 100, 150, 400 and 2,500 ppm nitrite). The higher concentrations (400 and 2500 ppm) of nitrite were used because 150 ppm were not effective in preventing aerobic bacterial growth in dark meat patties. Each patty was homogenized in sterile water in a sterilized Waring Blendor. Appropriate serial dilutions were prepared with 0.1% peptone (*9*) and used for inoculating triplicate pour plates of plate count agar (Difco). All plates were incubated at room temperature (22°C) for 5 d and then counted.

RESULTS AND DISCUSSION

The effect of muscle type on the concentration of residual nitrite in chicken patties stored at 4°C is shown in Tables 1 and 2. Both the raw and cooked white meat patties contained less residual nitrite than the dark meat patties. The fact that cooked chicken patties, both white and dark, contained less residual nitrite than raw patties reflects the loss of nitrite during the conversion of myoglobin to nitrosylhemochrome on cooking. Two factors may contribute to the difference in residual nitrite content in chicken white and dark meat patties.

TABLE 1. *Effect of muscle type on residual nitrite levels in raw chicken patties formulated with 150 ppm nitrite and stored at 4 to 5°C for 6 d.*

Treatment	Day	Nitrite[a] (ppm)
White meat	0	104 ± 2.4
	3	99 ± 1.9
	6	90 ± 1.6
Dark meat	0	115 = 2.2
	3	114 ± 2.9
	6	104 ± 1.8

[a]All values represent the means of six analyses.

TABLE 2. *Effect of muscle type on residual nitrite levels in cooked chicken patties formulated with 150 ppm nitrite and stored at 4 to 5°C for 14 d.*

Treatment	Day	Nitrite[a] (ppm)
White meat	0	80 ± 3.1
	7	75 ± 3.5
	14	65 ± 0.9
Dark meat	0	94 ± 3.4
	7	90 ± 2.7
	14	85 ± 2.0

[a]All values represent the means of six analyses and are based on the uncooked weight of the patties.

First, the raw and cooked dark meat had a higher pH than raw and cooked white meat, i.e., 6.4 and 6.6 vs. 5.6 and 6.0, respectively. At lower pH, reactions converting nitrite to other products are enhanced. Lee et al. (*6,7*) showed that pale muscle retains less residual ntirite than red muscle. These researchers attributed the difference in residual nitrite to the lower pH of white muscle. Secondly, dark meat contains higher concentrations of iron than white meat (7.0 vs. 3.0 ppm). Tompkin et al. (*10,11*) have demonstrated that nitrite and/or nitric oxide may bind iron thus preventing the use of nitrite as an antibotulinal agent and also as a participant in color fixation. Hence for nitrite to be effective in chicken dark meat, it is necessary to increase the residual nitrite concentration. Analysis of the moisture expelled from the patties during cooking demonstrated that no detectable nitrite was lost during cooking. White meat and dark meat patties decreased by 12 to 18% in weight during cooking.

The results obtained from bacteriological analysis of raw chicken patties treated with different levels of nitrite (0, 50, 100, 150, 400 and 2500 ppm) and held at 4°C are shown in Figures 1 and 2. The total plate counts for fresh chicken patties both treated and untreated ranged from 4×10^3 to 3.0×10^4 CFU/g.

Bacterial growth in meat formulated with 50 ppm nitrite was not effectively inhibited during storage at 4°C. Total aerobic counts for chicken meat with 100 and 150 ppm nitrite were 2 to 4 $\log_{10}$ colony-forming units (CFU)/g lower than that of the control made without nitrite (Fig. 1). Nitrite at concentrations of 100 and 150 ppm was not effective in lowering the total number of aerobic microorganisms in dark meat patties (Fig. 2). The 400-ppm level of added nitrite decreased bacterial counts in white meat (Fig. 1), but was only effective in dark meat for the first 6 d of storage (Fig. 2).

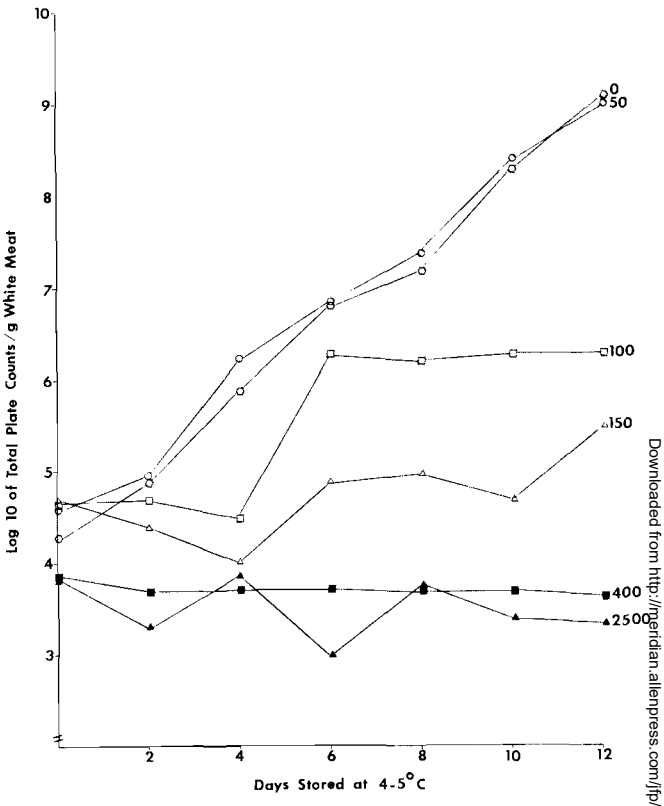

Figure 1. *Total plate counts per gram of chicken white meat vs. days of storage at 4 to 5°C. Patties were formulated with 0, 50, 100, 150, 400 or 2500 ppm nitrite. All points represent the average of 36 plates (Six plates for each dilution for each of 3 samples × 2 experiments).*

Two factors may contribute to these results. First, raw dark meat has a higher pH than raw white meat (6.4 vs. 5.6). It is well-established that nitrite is most inhibitory to bacteria at acid pH values of 4.5 to 5.5 (*5*). The inhibitory concentration decreases 10-fold for each pH unit that the pH rises from 5.0. Thus, it is not surprising that more nitrite is needed in dark meat since the pH is much higher. Secondly, dark meat contains a higher concentration of iron than white meat (7.0 vs. 3.0 ppm). Tompkin et al. (*10,11*) have deomonstrated a relationship between increased iron content and the decreased antibotulinal activity of nitrite. These researchers have suggested that iron reacts with nitric oxide produced from nitrite and consequently reduces the concentration of nitric oxide available to react with an iron-containing compound within the vegetative clostridial cell (*10,11*). Perhaps a similar reaction accounts for their inhibition of aerobic bacteria by nitrite, and the higher iron content of chicken dark meat may explain these results.

Results of these experiments demonstrate that the residual nitrite content of chicken dark meat is always higher than in chicken white meat, but the protection from bacterial spoilage is greatly reduced in dark meat patties compared to white meat patties when formulated nitrite levels are equivalent.

ACKNOWLEDGMENT

This research was supported in part by a grant from the Faculty Research Fund, University of Maine at Orono, and was performed as part of NC-133.

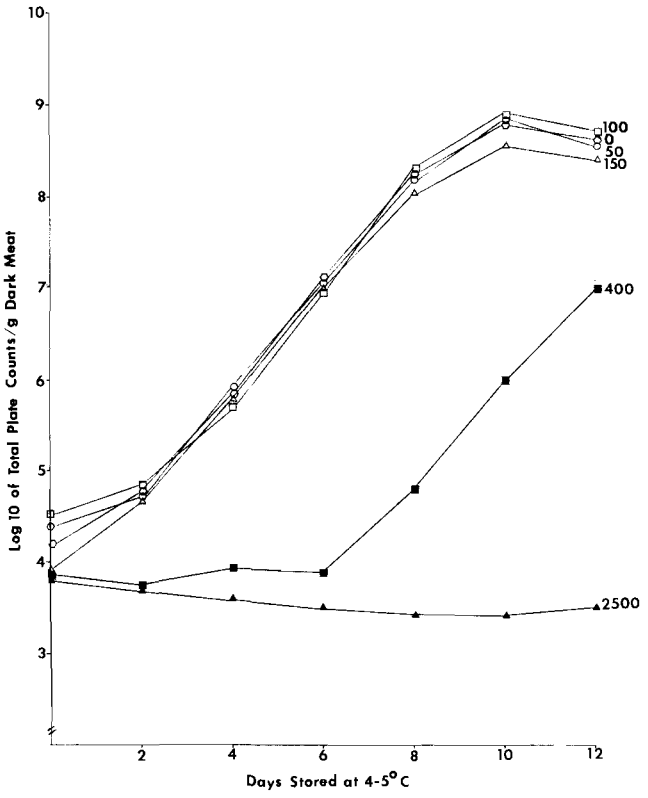

Figure 2. *Total plate counts per gram of chicken dark meat vs. days of storage at 4 to 5°C. Patties were formulated with 0, 50, 100, 150, 400 or 2500 ppm nitrite. All points represent the average of 36 plates (Six plates for each dilution for each of 3 samples × 2 experiments).*

REFERENCES

1. Association of Official Analytical Chemists. 1980. Official methods of analysis, 13th ed. AOAC, Washington, DC.
2. Fox, J. B., Jr., and R. A. Nicholas. 1974. Nitrite in meat: effect of various compounds on loss of nitrite. J. Agric. Food Chem. 22:302-306.
3. Fox, J. B., Jr., W. E. Townsend, S. A. Ackerman, and C. E. Swift. 1967. Cured color development during frankfurter processing. Food Technol. 21:68.
4. Goutefongea, R., K. Cassens, and G. Woolford. 1977. Distribution of sodium nitrite in adipose tissue during curing. J. Food Sci. 42:1637-1641.
5. Holley, R. A. 1981. Review of the potential hazard from botulism in cured meats. Can. Inst. Food Sci. Technol. J. 14:183-195.
6. Lee, S. H., R. A. Cassens, and O. R. Fennema. 1976. Effect of muscle type on residual nitrite in cured meat. J. Food Sci. 41:100-101.
7. Lee, S. H., R. A. Cassens, and H. Sugiyama. 1978. Factors affecting inhibition of *C. botulinum* in cured meats. J. Food Sci. 43:1371-1374.
8. Nordin, H. R. 1969. Depletion of added sodium nitrite in ham. Can. Inst. Food Technol. J. 2:79-85.
9. Straka, R. P., and J. L. Stokes. 1951. Rapid destruction of bacteria in commonly used diluents and its elimination. Appl. Microbiol. 5:21-27.
10. Tompkin, R. B., L. N. Christiansen, and A. B. Shaparis. 1978. The effect iron on botulinal inhibition in perishable canned cured meat. J. Food Technol. 13:521-527.
11. Tompkin, R. B., L. N. Christiansen, and A. B. Shaparis. 1979. Iron and the anti-botulinal efficacy of nitrite. Appl. Environ. Microbiol. 37:351-353.
12. Waldman, R. C., D. O. Westerberg, and C. Simin. 1974. Influence of curing ingredients and storage time on the quality of preblended sausage meats and frankfurters. J. Food Sci. 39:718-722.
13. Zaika, L. L., T. E. Zell, J. L. Smith, S. A. Palumbo, and J. C. Kissinger. 1976. The role of nitrite and nitrate in Lebanon bologna: a fermented sausage. J. Food Sci. 41:1457-1460.

Newsome, et al., *con't. from p. 118*

28. Sutherland, J. P., J. T. Patterson, and J. G. Murray. 1975. Changes in the microbiology of vacuum packaged beef. J. Appl. Bacteriol. 39:227-237.
29. Thornley, M. J. 1960. The differentiation of *Pseudomonas* from other Gram-negative bacteria on the basis of arginine metabolism. J. Appl. Bacteriol. 23:37-52.
30. Vanderzant, C., and R. Nickelson. 1969. Microbiological examination of muscle tissue of beef, pork and lamb carcasses. J. Milk Food Technol. 32:357-361.