Sodium Chloride-Mediated Lipid Oxidation in Beef Heart Surimi-like Material[†]

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Lipid oxidation in beef heart surimi prepared in the presence of salt (0.1 M NaCl) was significantly ($P < 0.05$) less than in its absence at all pH (5.5, 6.0, 6.5, and 7.0) values tested. Lipid oxidation in surimi during 48-h storage was inhibited at pH 7.0 even without 0.1 M NaCl but was stimulated at pH 5.5 regardless of salt treatments. In surimi prepared without salt at pH 6.0, the added salt concentrations of 0.025 and 0.1 M NaCl slightly inhibited lipid oxidation, but concentrations above 0.1 M NaCl promoted lipid oxidation throughout storage. Different salts (NaCl, KCl, LiCl, NaBr, Na_2SO_4) at ionic strength of 0.1 M inhibited lipid oxidation in a similar manner. However, at an ionic strength of 0.6 M, the effect of specific salts on stimulating lipid oxidation varied. Inhibition of lipid oxidation in beef heart surimi by salt washing may be related to the removal of heme proteins from minced muscle.

Keywords: *Beef heart; surimi; salt; lipid oxidation*

INTRODUCTION

Salt (NaCl) plays important roles in meat processing. Depending on the concentration used, the function of salt varies in meat products. In fish-based surimi (washed minced product) processing, salt is used at 0.1−0.2% levels to facilitate dewatering of the surimi. In cooked muscle foods, where texture and water binding are important quality traits, a relatively high concentration of salt (typically 2−3%) is used to solubilize myofibrillar proteins. Other useful functions of salt include imparting flavor to meat products and inhibiting growth of spoilage organisms. However, salt was shown to be capable of catalyzing lipid peroxidation in muscle foods (Salih *et al.*, 1989; Kanner *et al.*, 1991; Osinchak *et al.*, 1992), although the opposite was also reported (Chang and Watts, 1950; Nambudiry, 1980). There were also studies which showed no effect by salt on lipid oxidation in some food systems (Rhee *et al.*, 1983; Torres *et al.*, 1988). The conflicting observations on the role of salt in lipid oxidation may be due to differences in the meat systems or species examined (*e.g.*, beef, pork, chicken, or fish), complexity of the model system, and concentrations of salt used. Some contradictions also exist in reports on the mechanism of salt-catalyzed lipid oxidation in muscle foods. While some researchers (Castell *et al.*, 1965; Ellis *et al.*, 1968) claimed that the prooxidant activity of NaCl was the result of sodium ions, others (Osinchak *et al.*, 1992) characterized chloride as the active component of the prooxidative salt.

In recent years, there has been an increased emphasis on utilization of meat by-products in the manufacture of value-based muscle foods. This has led to several studies aimed at preparing surimi-like materials from beef hearts for further use in formulated meats (McKeith *et al.*, 1988; Kenney *et al.*, 1992; Wan *et al.*, 1993). However, the susceptibility of beef heart muscle to oxidative deterioration was well recognized in these studies. In our previous research, we had shown that inhibition of lipid oxidation in beef heart surimi-like materials can be achieved using combinations of pH, NaCl, and buffer type in the washing media (Srinivasan *et al.*, 1995). We found that a salt concentration of 0.1 M in final washing solution (pH 6.0) during the preparation of beef heart surimi curtailed lipid peroxidation. However, the role of salt in relation to variations in pH on lipid oxidation in the surimi was not understood. Furthermore, relatively high concentrations of salt (0.5−1.0 M) are routinely used in further processed meats, especially in those gel-based muscle foods. Thus, an improved understanding of lipid oxidation as related to salt treatment under various ionic conditions is important. The purpose of this study was to examine the role of NaCl in catalyzing or inhibiting lipid oxidation in beef heart surimi-like material and its mixture with salt (sol) during the preparation and storage as a function of pH. It was hoped that the study would also allow us to gain a further appreciation of the mechanism(s) by which salt mediated lipid oxidation in muscle foods.

MATERIALS AND METHODS

Materials. Fresh beef hearts (24−28 h postmortem) were obtained from cattle of age ranging from 16 to 20 months slaughtered at a local meat-packing company. Beef hearts were individually vacuum packaged in polyethylene bags and frozen in a blast freezer (−29 °C). All heart samples were stored at −29 °C and used within 4 months. Piperazine-*N*,*N*′-bis(2-ethanesulfonic acid) disodium salt (PIPES), cytochrome *c* (type V-A; bovine heart), myoglobin (type 1; horse skeletal muscle), and bovine serum albumin (fraction V) were purchased from Sigma Chemical Co. (St. Louis, MO). Standard protein molecular weight markers (broad range, Catalog No. 161−0317) were obtained from Bio-Rad Laboratories (Hercules, CA). All other chemicals used were of at least analytical or electrophoresis grade.

Preparation of Surimi-like Material. All experiments were replicated with at least three different hearts using ground and washed muscle samples freshly prepared on different days. All samples for each experiment originated from the same hearts. For each parameter studied in the same experiment, 100 g of ground muscle was used. Beef hearts

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were thawed at 2 °C for 24 h. All sample preparations were carried out in a 5 °C walk-in cooler, unless otherwise specified. After the removal of caps, vessels, and external fat tissue, the lean muscle was diced into approximately 2-cm cubes, and ground through a 4.7-mm diameter orifice using a Kitchen Aid (Kitchen Aid Inc., St. Joseph, MI) mincer. The minced muscle was washed twice in 10 vol (v/w) of washing solution (specified below) by stirring with a spatula for 1 min. The mixture was allowed to stand for 10 min and then strained on a nylon screen mesh (0.25 cm^2). A third and final wash was done in 5 vol (v/w, based on original weight of mince) of a buffer (specified below) by blending in a Waring blender for 1 min at high speed. The pH of the homogenate was adjusted to desired values with 0.1 N HCl or 0.1 N NaOH, if necessary, and centrifuged at 2000*g* for 15 min. The supernatant was decanted and stored together with the pellet in an ice container (0 °C).

Washing Solutions. All washing solutions were prepared using PIPES as the buffering media since orthophosphate was found to have an inhibitory effect on lipid peroxidation in beef heart surimi (Srinivasan *et al.*, 1995). In the case of buffer-washed surimi (control), all three washes were done in 25 mM PIPES of the same pH (5.5, 6.0, 6.5, or 7.0) free of salt. For salt-washed surimi, all three washes were done in 25 mM PIPES of the same pH as for buffer-washed surimi except that 0.1 M NaCl was incorporated in the final wash. In a preliminary experiment, 25 mM PIPES, pH 6.0, buffer containing 0.1 M NaCl was used for all three washes (denoted "all salt-wash"). When various salts were used, all three washes were done in 25 mM PIPES (pH 6.0) with specific salts incorporated in the final wash at a concentration equivalent to 0.1 M NaCl in ionic strength.

Preparation of Salted Surimi. In experiments determining the effects of NaCl concentration, appropriate amounts of NaCl dissolved in 25 mM PIPES (pH adjusted to relevant value after the addition of salt) were added to buffer-washed surimi to final concentrations of 0.025−1.0 M NaCl. The final NaCl concentrations were based on the assumption of a negligible or no residual presence of NaCl in buffer-washed surimi (after a total dilution of ~725×). For salt-washed surimi, assuming a residual NaCl concentration of 0.1 M in the surimi pellet after the third (final) wash, appropriate amounts of NaCl in 25 mM PIPES were added to final concentrations of 0.1−1.0 M. In experiments determining the effects of specific salts, different salts were added to buffer-washed surimi to a final concentration of 0.1 or 0.6 M, except the sulfate salt which was 0.033 or 0.2 M, respectively (ionic strength equal to the monovalent salts). The final protein concentration of all samples was 50 mg/mL.

Protein Content. Protein concentration in samples was determined by the biuret method after diluting a 0.1-mL aliquot to 1.0 mL with distilled deionized water and reacting with 4.0 mL of biuret reagent (Torten and Whitaker, 1964). Bovine serum albumin was used as standard.

Thiobarbituric Acid-Reactive Substances (TBARS). The TBARS measurement was based on the method of MacDonald and Hultin (1987) using a molar extinction coefficient of 15 600 M^{-1} cm^{-1}. Two milliliters of 7.5% TCA, 0.1% PG, and 0.1% EDTA was added to 1.0 mL of sample while mixing. The mixture was centrifuged, and the supernatant was used for reaction with thiobarbituric acid.

Conjugated Dienes. Conjugated dienes were measured as increases in the absorbance at 233 nm (Esterbauer *et al.*, 1982) of a hexane, 2-propanol extract. A 0.5-mL sample was extracted with 5.0 mL of combined hexane and 2-propanol (3:2) for 1 min on a vortex mixer and centrifuged at 2000*g* for 5 min. A blank was run with distilled deionized water. The difference in absorbance at 233 nm was used for the determination of conjugated dienes, using a molar extinction coefficient of 25 200 M^{-1} cm^{-1} (Buege and Aust, 1978).

Electrophoresis. SDS−PAGE was performed according to the method described by Laemmli (1970) using an SE 250 Mighty Small II slab gel electrophoresis unit (Hoefer Scientific Instruments, San Francisco, CA). The stacking gel and the resolving gel were made of 3% and 15% acrylamide, respectively. Sample buffer contained 8 M urea, 2 M thiourea, 3% SDS, 75 mM dithiothreitol, 0.05 M Tris-HCl, and 0.05% bromophenol blue (pH 8.0) and was used at a 1:1 dilution of sample with the buffer to a final protein concentration of 1 mg/mL (Srinivasan and Hultin, 1995).

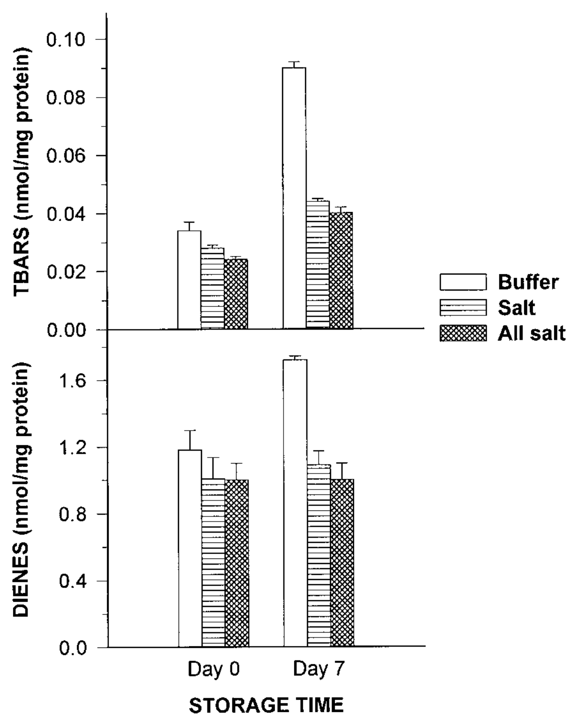

Figure 1. TBARS and conjugated dienes in beef heart surimi after preparation (day 0) and storage (day 7) at 0 °C. The surimi was prepared by washing in 25 mM PIPES with or without 0.1 M NaCl (final pH 6.0). Buffer: all three washes were done in buffer alone. Salt: two washes were carried out in buffer alone and a third wash in buffer plus salt. All salt: all three washes were performed in buffer plus salt.

Total Hematin Content. Total hematin extraction and calculation were based on the method of Hornsey (1956). After extraction of the pigment with acetone/HCl solvent in a screw-capped test tube for 1 h, the mixture was centrifuged at 2600*g* for 10 min, and the supernatant was measured for hematin by measuring absorbance at 640 nm.

Statistical Analysis. Data were analyzed using the General Linear Models procedure of the Statistix 3.5 software package (Analytical Software, Inc., St. Paul, MN) for microcomputers. Analysis of variance (AOV) was done to determine the significance of main effects (treatment, storage time). Significant ($P < 0.05$) differences between means were identified using the least significant difference procedure (Snedecor and Cochran, 1989).

RESULTS

Incorporation of salt (buffered at pH 6.0) in the final wash (salt-washed) or in all three washes (all salt-washed) resulted in a slightly lower ($P < 0.05$) TBARS content in surimi-like material (referred to as 'surimi' without inference to fish-based product) on the day of preparation than control surimi (buffer-washed) (Figure 1). After 7-day storage at 0 °C, the difference in TBARS in surimi with and without salt became very large ($P < 0.05$). Incorporation of salt in the final wash or in all three washes did not make a difference with respect to the TBARS content at either day 1 or 7. Conjugated dienes, as another index of lipid oxidation, showed no difference ($P > 0.05$) among surimi samples with or without salt on the day of preparation (Figure 1). However, after 7-day storage, there was an increase in diene content in buffer-washed surimi, while the dienes of salt-containing surimi samples remained the same.

Table 1. Effect of Salt (0.1 M NaCl) in Washing Solution on Protein and Hematin Concentrations in the Preparation of Beef Heart Surimi[a]

	protein		hematin	
surimi	SUP (mg/mL)	SED (mg/g)	SUP (μmol/mL)	SED (μmol/g)
buffer-washed	1.033	286	0.045	0.187
salt-washed	3.316	250	0.055	0.114

[a] Surimi was prepared by two washes in 10 vol of 25 mM PIPES at pH 6.0 followed by a final wash in 5 vol of the same buffer without (buffer-washed) or with 0.1 M NaCl (salt-washed); SUP (supernatant) and SED (sediment, *i.e.*, surimi) were from the final wash.

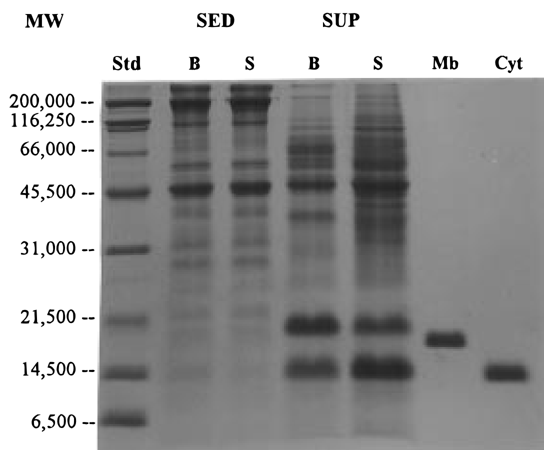

Figure 2. SDS−PAGE profile of supernatant (SUP) and sediment (SED) obtained from the final or third wash. Letters B and S represent buffer- and salt-washed beef heart surimi, respectively. The pH of the washing solutions was 6.0. Each lane contained 20 μg of protein. Standard molecular weight marker proteins (Std) were run simultaneously; Mb, myoglobin; Cyt, cytochrome *c*.

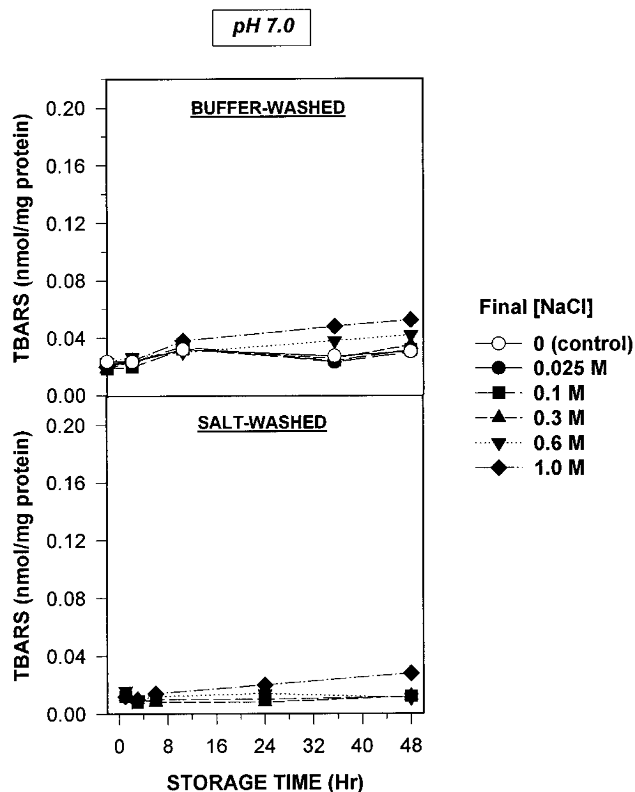

Figure 3. Changes in TBARS as a function of NaCl concentration in salted beef heart surimi at pH 7.0 during refrigerated storage (6 °C). The surimi was prepared either in the presence (salt-washed) or in the absence (buffer-washed) of salt (0.1 M NaCl) as described in Materials and Methods, and salt at various levels was added to the final surimi pellet before storage. The final salted samples contained 50 mg/mL protein.

Detectable changes in dienes were 1 order of magnitude higher than those of TBARS, and this may be related to sensitivity differences between the two methods employed. Only TBARS as an index of lipid oxidation was measured in all further experiments.

Protein contents in supernatants of the final washings (pH 6.0) were estimated (Table 1). There was 2-fold more protein in the supernatant of salt-washed (0.1 M NaCl) surimi than in the supernatant of buffer-washed surimi; protein content of salt-washed surimi (sediment) was slightly lower than that of buffer-washed surimi. Total hematin concentration in supernatant was higher than that of sediment and lower than that for salt-washed surimi as compared to the corresponding values of buffer-washed surimi (Table 1). SDS−PAGE analysis revealed some differences in peptide bands removed in supernatants between buffer- and salt-washing treatments (Figure 2). In general, a larger number of peptides were found in the supernatant of salt-washed surimi than in the supernatant of buffer-washed surimi. Two major peptide bands in both supernatants were particularly noticeable, one of which migrated the same distance in the gel as the cytochrome *c* standard and the other nearly as far as myoglobin. All peptide bands in both sediments were essentially the same.

Lipid oxidation as a function of added salt was monitored in both buffer- and salt-washed surimi at various pH values. The final concentration of salt added to buffer-washed surimi was varied from 0.025 to 1.0 M. For salt-washed surimi, assuming a residual salt of 0.1 M NaCl after the final (*i.e.*, third) wash, salt was added to the surimi to final concentrations ranging from 0.1 to 1.0 M. At pH 7.0, the effect of salt in the range of 0.1−0.6 M in salt-washed surimi caused very little lipid oxidation as measured by TBARS during 24-h incubation at 6 °C (Figure 3). A slight increase ($P < 0.05$) in TBARS was observed at 1.0 M NaCl. In buffer-washed surimi at the same pH, TBARS slightly increased ($P < 0.05$) during the 24-h incubation at high salt concentrations, *i.e.*, 0.6 and 1.0 M NaCl. When the pH of the washing solutions was adjusted to 6.5, the changes in TBARS due both to storage and to added salt became prominent (Figure 4). In buffer-washed surimi, salt at a 0.3 M concentration and above resulted in an increase in TBARS, while there were no significant ($P > 0.05$) changes in TBARS below 0.3 M salt. The largest increase in TBARS occurred at 1.0 M NaCl. In salt-washed surimi at pH 6.5, the changes in TBARS were relatively small. There were small increases in TBARS in salt-washed surimi at elevated salt concentrations of 0.6 and 1.0 M NaCl.

A further reduction of pH to 6.0 resulted in even larger changes in lipid oxidation. In salt-washed surimi, a salt concentration of 0.1 M did not significantly stimulate lipid oxidation during storage (Figure 5). However, increasing the salt concentration to 0.3 M or above promoted lipid oxidation. In buffer-washed surimi, lipid oxidation appeared to be slightly inhibited (first 24 h at least) by 0.025 and 0.1 M salt. However, salt concentrations of 0.3 M and above greatly accelerated lipid oxidation. As the pH of the samples was further reduced to 5.5, lipid oxidation was remarkably high both

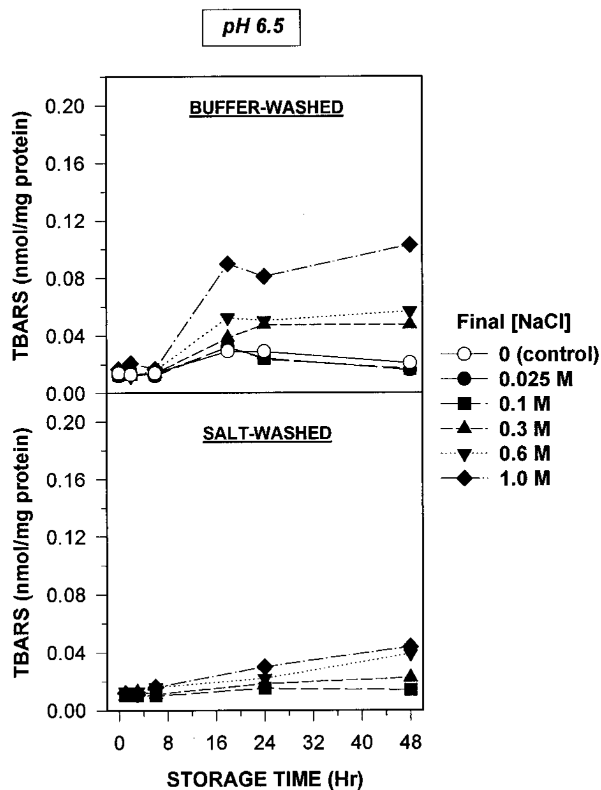

Figure 4. Changes in TBARS as a function of NaCl concentration in salted beef heart surimi at pH 6.5 during refrigerated storage (6 °C). Figure legends are the same as in Figure 3.

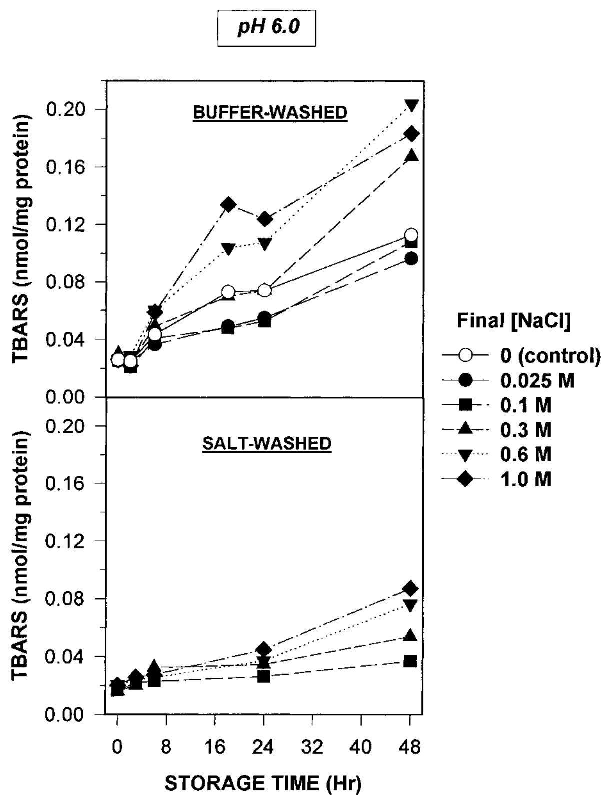

Figure 5. Changes in TBARS as a function of NaCl concentration in salted beef heart surimi at pH 6.0 during refrigerated storage (6 °C). Figure legends are the same as in Figure 3.

in buffer-washed and in salt-washed surimi during storage (Figure 6) and was more so for buffer-washed surimi samples. The stimulating effect of pH 5.5 on

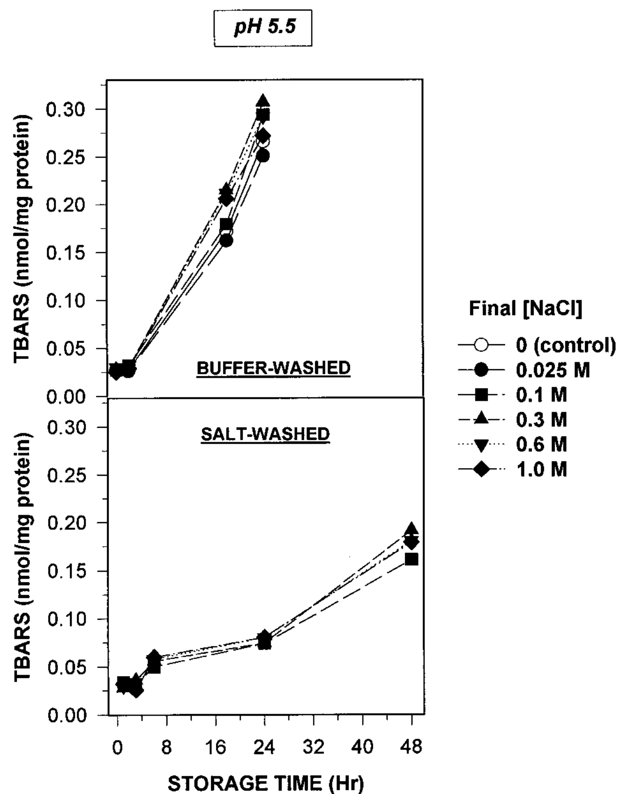

Figure 6. Changes in TBARS as a function of NaCl concentration in salted beef heart surimi at pH 5.5 during refrigerated storage (6 °C). Figure legends are the same as in Figure 3.

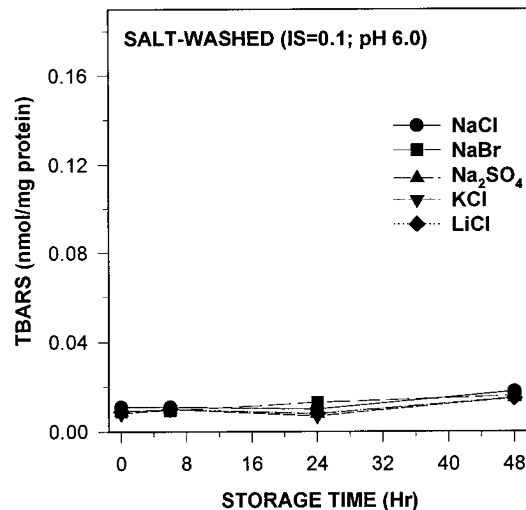

Figure 7. Effect of specific salt in the washing solution (pH 6.0) on TBARS in salted beef heart surimi during refrigerated storage (6 °C). Different salts of equal ionic strength of 0.1 M (0.1 M for monovalent salts and 0.033 M for Na_2SO_4) were used in the final wash in the preparation of the surimi.

lipid oxidation in both samples was independent of the concentration of added salt.

Sodium salts of some anions (Cl^-, Br^-, SO_4^{2-}) and chloride salts of some cations (Na^+, K^+, Li^+) were used at an equal ionic strength of 0.1 M in final washing solution, and their effects on lipid oxidation were compared (Figure 7). No significant ($P > 0.05$) differences were observed between various salts. The same specific salts at two different ionic strengths (0.1 and 0.6 M) were also added to buffer-washed surimi (after final wash), and changes in lipid oxidation induced by these salt treatments were monitored over a 48-h

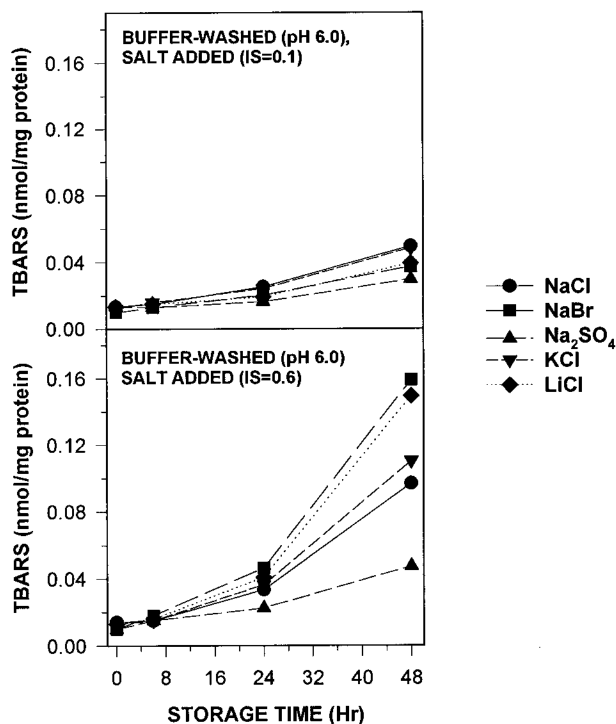

Figure 8. Effect of specific salt added to buffer-washed beef heart surimi on TBARS during storage (6 °C). Different salts were added to buffer-washed surimi to equal a final ionic strength of 0.1 or 0.6 M (*i.e.*, 0.1 or 0.6 M for monovalent salts and 0.033 or 0.2 M for Na_2SO_4).

storage period. At the lower ionic strength (0.1 M), the effects of all salts were small with Na_2SO_4 being the least among them (Figure 8). At the higher ionic strength (0.6 M), all salts were obviously much more stimulatory of lipid oxidation than they were at the lower ionic strength (0.1 M). However, prooxidant activities of these salts varied at higher ionic strength. Among the salts, NaBr and LiCl exhibited the highest potency and Na_2SO_4 the least, with NaCl and KCl being in the intermediate range.

DISCUSSION

The prooxidant activity of salt in catalyzing lipid oxidation in muscle foods under various conditions is well recognized. However, some reports showed that salt, depending on the concentration used, can inhibit lipid oxidation. Inhibition of lipid oxidation in beef heart surimi during storage by the incorporation of 0.1 M NaCl in washing solution was clearly demonstrated in this study. Incorporation of salt in the final washing step alone resulted in inhibition of the oxidation, and it was not necessary to add salt in all three washes during the preparation of surimi to achieve the inhibitory effect. An early study by Chang and Watts (1950) showed that 5% NaCl slightly inhibited oxidation of lard by hemoglobin or a muscle extract, the mechanism for which was unknown. Nambudiry (1980) also claimed that lipid oxidation was curtailed in deheaded and gutted sardine dipped in salt solutions to final concentrations of 2.3% and higher. The mechanism for the inhibition was not elucidated by the author. In both these preceding studies, inhibition of lipid oxidation was observed at high salt concentrations, *i.e.*, 2.3% and above, unlike the present study in which the inhibitory effect was seen at much lower salt concentrations (0.29% and 0.58%). Wimmer *et al.* (1993) reported suppression of lipid oxidation in frankfurters containing washed (water or 1.5% salt) mechanically separated pork during storage. The suppression was attributed to removal of unknown catalysts or initiators of lipid oxidation through washing.

Analysis of supernatant and sediment (surimi) for protein and total hematin concentrations revealed distinct patterns. Washing with salt removed 2-fold more protein from the muscle mince than washing without salt. The amount of residual hematin in salt-washed sediment was much lower than in buffer-washed sediment. Proteins that were removed by salt consisted mostly of non-myofibrillar proteins as revealed by SDS−PAGE analysis. The solubility of myofibrillar proteins in 0.1 M NaCl is known to be nearly minimal. The non-myofibrillar proteins removed by the dilute salt solution would consist of sarcoplasmic proteins including enzymes involved in glycolytic and other metabolic processes, heme proteins, and perhaps some prooxidative enzymes. Two of the major polypeptides extracted into the supernatant were presumably heme proteins, based on their mobilities (MW) relative to those of the protein standards (Figure 2). One of the major polypeptides extracted into the supernatant was likely cytochrome *c*, based on its similar electrophoretic mobility to that of the cytochrome *c* standard (MW around 12 300) (Figure 2). The polypeptide that migrated behind the presumed cytochrome *c* was another major component in the supernatant. It would be speculative to suggest that this was a myoglobin band or its derivative on the basis of the band intensity (relative abundance) and its location in the gel. However, the identity of this protein is not known since its mobility in the SDS−PAGE gel did not exactly match the myoglobin standard's. An accurate identification of this protein band would actually require more complicated analysis, such as immunological assay or peptide mapping. Since the supernatant of salt-washed surimi contained about 3 times as much protein as the supernatant of buffer-washed surimi and that the hematin content of salt-washed sediment was lower than that of buffer-washed sediment, it is evident that salt washing removed a substantially larger amount of heme proteins. Yang and Froning (1992) demonstrated a 70% decrease in redness, as measured by a colorimeter, by washing mechanically deboned chicken meat in 0.1 M NaCl solution. Cytochrome *c* is part of the electron transport system found in mitochondria (Pearson and Young, 1989). The concentration of cytochrome *c* in cardiac muscle (371 μg/g of tissue) is nearly 4 times as much as in skeletal muscle (97 μg/g of tissue) of rats (Potter and Dubois, 1942). Cytochrome c, an iron-containing heme protein, is a known biological catalyst of lipid peroxidation (Kanner *et al.*, 1994). Mitochondria account for nearly 33.8% of the total cell volume in cardiac muscle (Anversa *et al.*, 1978), and the mitochondrial protein comprises about 15% of the total protein in cardiac muscle compared to only about 5% in skeletal muscle (Zak and Galhotra, 1983). Mitochondria produce deleterious reactive oxygen species such as $O_2^{\bullet-}$ and H_2O_2 (Sohal and Brunk, 1992) and thus could be an important causal factor in mediating lipid oxidation in the present beef heart surimi study.

A combination of observations, namely, proteins washed out in the supernatant, hematin content reduced in the sediment, and correspondingly lipid oxidation curtailed, suggested that heme- and enzyme-catalyzed systems were present in beef heart muscle. The pH dependency of lipid oxidation indicated that a

heme-catalyzed mechanism may be predominant in the system. When the pH of the system was 7.0, lipid oxidation was inhibited substantially even in the absence of 0.1 M NaCl. Evidence of lipid oxidation at pH 7.0 was obtained when the concentration of added salt was high (0.6−1.0 M). In contrast, lipid oxidation was strongly promoted at pH 5.5 irrespective of the concentration of added salt. If an enzyme system were predominant, a variation in pH by 2.0 units might not produce such a drastic alteration, for example, inhibition of lipid oxidation at pH 7.0 and catalysis of the oxidation at pH 5.5, irrespective of salt treatments. The pH of the washing solution may affect not only the rate of prooxidant-catalyzed lipid oxidation but also solubilization of proteins by NaCl. Catalysis of lipid oxidation at pH 5.5 in the presence and absence of salt in the washing solution may indicate that salt at this low pH removes less catalysts during washing than at higher pH (*e.g.*, 6.0 and above). Inhibition of lipid oxidation in salt-washed surimi at pH 6.0 and above might be due to the ability of NaCl to displace iron from binding sites (macromolecules and membranous fatty acids) as suggested by Kanner *et al.* (1991). The displaced iron might have been removed in the supernatant thereby reducing iron-dependent lipid oxidation in the surimi sediment. At pH 5.5, the capacity of NaCl to displace iron from the binding sites might be reduced and, thus, unable to inhibit lipid oxidation.

All salts used in the present study, incorporated in washing solution at a 0.1 M ionic strength, inhibited lipid oxidation in a similar manner. This indicates that the nature of salt in the washing solution was not a critical factor in preventing lipid oxidation in beef heart surimi at this ionic strength. When these salts were added at a low concentration (0.1 M) to buffer-washed surimi, they exhibited only a minimal effect. When added at a much higher concentration (0.6 M), these salts stimulated lipid oxidation to various extents. The known binding abilities of various anions to ferric iron (Osinchak *et al.*, 1992) were not consistent with the hypothesis that redox (non-heme) iron was primarily responsible for lipid oxidation in our study. For example, chloride is a good binder of ferric iron, while bromide is not. Hence, one would expect different catalytic patterns of lipid oxidation by NaCl and NaBr. However, the extent of stimulation of lipid oxidation was similar for both anions.

In summary, incorporation of 0.1 M salt in the washing solution during the preparation of surimi-like material from beef hearts improved surimi stability against oxidative deterioration during storage on ice. This effect was greatly dependent on pH of the washing solution. Similar antioxidative effects by salt were achieved by adding low concentrations of salt (≤0.1 M) to washed surimi and adjusting the surimi pH to near neutrality. Salt acted as a prooxidant in washed beef heart surimi when its concentration was high (≥0.3 M) or the pH was reduced to the acidic range. One of the most feasible applications of beef heart-derived surimi is to use it as a functional ingredient in processed meats such as restructured meat loaves and frankfurters (Kenney *et al.*, 1992) where the ability of surimi to gel and to emulsify lipids is important. Therefore, it appears that by controlling and manipulating the pH and salt concentration in the washing media, as well as the storage time, one can manufacture beef heart surimi with specific functionalities suitable for use as a texture modifier in comminuted and restructured meat products.

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