EFFECT OF LACTIC ACID TREATMENT ON BIND AND COOKING LOSS IN RESTRUCTURED BEEF

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BACKGROUND

Traditionally, restructured meat products are made by extracting myofibrillar proteins, using salt, phosphates and mechanical action, forming a heat set protein gel between meat pieces after cooking. However, these products do not bind in the raw state, limiting their marketing potential. Alternatively, alginate gelation can be used for restructuring, i.e. meat pieces are mixed with alginate, calcium carbonate and an acidulant, to augment calcium carbonate solubility (Means & Schmidt, 1986; Ensor *et al.*, 1990). Binding is then a result of a chemically set calcium alginate gel, not dependent on heat treatment, allowing these products to be marketed in the raw, refrigerated state. Restructured meat products are normally manufactured from less expensive cuts. The resulting products may lack tenderness due to high levels of connective tissue. Previous results have shown a tenderising effect of lactic acid treatment on less tender beef cuts (Eilers *et al.*, 1994, Ertbjerg *et al.*, 1995), while limited information are available on binding when lactic acid treated cuts are used for restructuring.

OBJECTIVES

The objective of this study was to evaluate bind and cooking loss in salt/phosphate and alginate/calcium restructured beef when lactic acid injection was performed prior to the restructuring process.

METHODS

Experimental design

The effect of lactic acid injection on properties of restructured beef products was investigated in two experiments. In Exp. 1, three binder types were studied: sodium alginate/calcium carbonate (AC), sodium alginate/calcium carbonate/glucono-delta-lactone (ACG) and sodium chloride/sodium tripolyphosphate (SP) and three lactic acid injection levels were studied: 0, 0.25 and 0.50 M lactic acid injected to 10% of the muscle weight. AC treatments included 0.75% sodium alginate, 0.15% calcium carbonate and 99.10% injected meat; ACG treatments included 0.75% sodium alginate, 0.15% calcium carbonate, 0.60% glucono-delta-lactone and 98.50% injected meat; SP treatments included 1.00% sodium chloride, 0.50% sodium tripolyphosphate and 98.50% injected meat. In Exp. 2, three alginate levels (0.50, 0.75 and 1.00%) along with the three lactic acid levels were examined. The ALG:$CaCO_3$ ratio was constant 5:1 in all AC and ACG treatments. A complete randomized factorial arrangement of treatments (3 x 3) replicated three times was used in both experiments. Results were analysed by analysis of variance using a significance level of $P<0.05$.

Materials

Meat source was *M. semitendinosus* from 3-4 year old Black Pied Danish cows slaughtered at The Danish Meat Trade School, Roskilde. The muscles were removed from carcasses 48h postmortem, trimmed of visible fat and connective tissue and cut lengthwise in halves. After randomizing, meat pieces were injected to obtain 10% weight increases as follows: One third were injected with pure distilled water, one third with 0.25M lactic acid in distilled water and one third with 0.50M lactic acid in distilled water. Injected muscles were then cut in pieces (approx. 350g), vacuumized and conditioned for 24h at 5 °C prior to storage at -18 °C until use. Additives were sodium alginate (Sobalg Fd 176, Danisco Ingredients, Brabrand, Denmark); calcium carbonate, glucono-delta-lactone, sodium chloride, sodium tripolyphosphate (all: Merck, Darmstadt, Germany).

Processing

Frozen samples were thawed at 5 °C for 24h and ground through a 35 mm x 14 mm kidney plate. Formulations (300.00 g w/w) were made by mixing manually ground meat with additives. In AC and ACG treatments the mixing sequence was: sodium alginate (60s), calcium carbonate (30s) and without or with GDL (60s). In SP treatments, salt and phosphate were added to the meat simultaneously, and mixed for 120s. The blend was hand-stuffed into six dishes (Diameter = 70 mm, Height = 40 mm), 50 g in each dish, covered by flexible polyethylene foil and allowed to set at 5 °C for 20-24h. Following setting, three of six 50 g-samples were weighed, vacuum-packed, heated (80 °C/30 min) in a waterbath and cooled in running tap water. Cooked samples were then used for cooking loss and cooked bind measurements. The remaining three samples were used for raw bind and pH measurements.

Analysis

Raw and cooked bind was measured using an Instron Universal Testing Machine (Model 4301, Instron Corp., High Wycombe Bucks, Great Britain). Raw samples were analysed immediately after removal from the refrigerator. Cooked samples were equilibrated at room temperature for 1h prior to evaluations. Raw and cooked bind was measured by a modification of Field *et al.*, 1984. Briefly, samples were placed over a 52 mm diameter opening, and penetrated by a 12.75 mm diameter cylindrical probe mounted to the Instron crosshead (Load cell 100N, crosshead speed 100 mm/min). Data were expressed as force at peak (Newton). Cooking loss was estimated after reweighing the cooked samples just before cooked bind measurements. Raw pH was measured with a pH meter (Model PHM 62, Radiometer Copenhagen, Denmark) by inserting a combined electrode four times into the raw samples after raw bind estimations.

RESULTS AND DISCUSSION

Raw bind in Exp. 1 was, as expected, higher ($P<0.05$) in AC and ACG treatments when compared to SP formulations (2.43, 2.96 and 0.94, respectively), since binding in salt/phosphate-restructured meat is a result of heat gelation of extracted myofibrillar proteins. ACG treatments had significantly higher raw bind values than AC treatments, presumably because of the presence of GDL causing more calcium ions to be released for reaction with alginate. When alginate levels were elevated (Exp.2), only the highest level of 1.00% increased raw bind ($P<0.05$) (Table 1). In neither of the experiments was raw bind significantly affected by increasing the lactic acid levels from 0 M to 0.50 M. Regarding alginate treatments, the positive effect of reducing pH when introducing lactic acid, leading to more released calcium, may be counteracted by calcium ion binding of lactate present in the medium (pK_A for lactic acid is 3.08 in aqueous solution).

Cooked bind was affected by interaction ($P<0.05$) between binder type and levels of lactic acid in Exp. 1. This was due to a marked cooked bind reduction for SP treatments when lactic acid levels were elevated (Fig.1), suggesting that salt/phosphate binding was hampered by decreased pH-values presumably as a result of reduced solubility of myofibrillar proteins. As shown in Fig. 1, cooked bind in Exp. 1 was considerably higher in ACG treatments when compared to AC treatments, again indicating the calcium releasing effect of GDL. Apparently, lactic acid injection had no influence on cooked bind in AC and ACG treatments (Fig. 1). In Exp. 2, cooked bind was not affected by increased alginate levels ($P>0.05$) (Table 1), but was enhanced in treatments containing 0.50 M lactic acid.

Cooking loss was, as for cooked bind, affected by interaction ($P<0.05$) between binder type and lactic acid levels in Exp. 1 (data not shown). SP treatments showed a pronounced cooking loss increase from 27% to 40% at 0 and 0.5 M lactic acid, respectively. Seemingly, cooked bind and cooking loss were inversely related when lactic acid levels increased in SP treatments, i.e. a weakening of product bind resulted in more released water after cooking. Part of the cooking loss increase probably originated from poorer water binding of myofibrillar proteins when pH decreased, either due to elevated lactic acid levels or the presence of GDL. In Exp. 2, cooking loss was markedly decreased when alginate levels increased from 0.50% to 1.00% (Table 1), due to excellent water binding properties of the hydrocolloid.

CONCLUSIONS

Post rigor injection of lactic acid (up to 0.50M) did not affect raw bind in alginate or salt/phosphate restructured beef products. Lactic acid markedly reduced cooked bind in salt/phosphate treatments, but had no effect or slightly enhanced cooked bind of alginate formulations. Generally, cooking loss increased as a function of lactic acid content, particularly in salt/phosphate treatments. In alginate restructuring, lactic acid injected meat could be used, especially if cooking loss increases could be compensated for by adding more alginate to the products. In restructured products, further studies are necessary for optimising the lactic acid treatment and sensory evaluation should be included.

Table 1. Raw bind, cooked bind, cooking loss and pH in Exp. 2 as affected by alginate (A) and lactic acid levels (B)[1].

(A)

Alginate (%)	Raw bind (N)	Cooked bind (N)	Cooking loss (%)	pH
0.50	2.09[a]	7.76[a]	32.93[c]	5.11[a]
0.75	2.22[a]	7.04[a]	28.49[b]	5.21[b]
1.00	3.23[b]	6.60[a]	23.92[a]	5.24[b]

(B)

Lactic acid (M)	Raw bind (N)	Cooked bind (N)	Cooking loss (%)	pH
0	2.64[a]	6.17[a]	26.36[a]	5.41[c]
0.25	2.55[a]	7.20[ab]	28.80[b]	5.14[b]
0.50	2.36[a]	8.03[b]	30.19[c]	5.01[a]

Means in a column with unlike superscripts differ ($P<0.05$)
[1]Values averaged across lactic acid levels (A) and alginate levels (B)

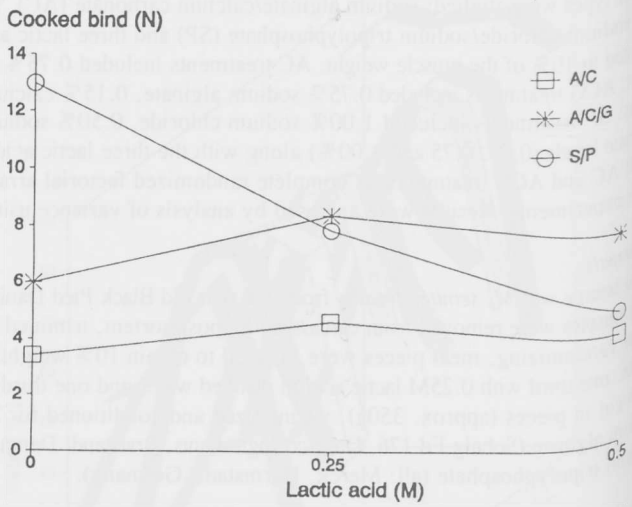

Fig. 1. Cooked bind in Exp. 1 as affected by binder type and lactic acid levels.

LITERATURE

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