Effects of High Pressure on the Myofibrillar Proteins of Cod and Turkey Muscle

K. Angsupanich, M. Edde, and D. A. Ledward*

Department of Food Science and Technology, The University of Reading, P.O. Box 226, Whiteknights, Reading RG6 6AP, U.K.

When turkey breast muscle and isolated myofibrillar protein and myosin of cod or turkey (pH ≈7) were subjected to pressures up to 800 MPa for 20 min, DSC and electrophoresis (SDS−PAGE) indicated that high pressure-induced denaturation of myosin led to the formation of structures that contained hydrogen bonds and were additionally stabilized by disulfide bonds. Disulfide bonds were also important in heat-induced myosin gels. Hardness of whole cod muscle, estimated by texture profile analysis, showed pressure-treated samples (400 MPa) to be harder than cooked (50 °C) or cooked and then pressure-treated or pressure-treated and then cooked samples, supporting the suggestion that pressure induces the formation of heat labile hydrogen-bonded structures while heat treatment gives rise to structures that are primarily stabilized by disulfide bonds and hydrophobic interactions. As expected, turkey myosin is more stable than that of cod; however, it seems their pressure-induced gelation mechanisms are similar.

Keywords: *High-pressure treatment; myosin gelation; cod myofibrillar proteins; turkey myofibrillar proteins; protein denaturation; DSC; TPA; electrophoresis*

INTRODUCTION

The use of high pressure to both preserve foods and modify their eating and functional properties is receiving considerable attention, and several high-pressure processed foods are now commercially available (Ohshima et al., 1993). Though work on both meat and fish has shown that high pressure may be a useful processing aid for such products (Cheftel and Culioli, 1997), our understanding of the effects of pressure on the structural components of muscle is limited. It is generally accepted that the connective tissue protein, collagen, is relatively unaffected by pressure (Gekko and Koga, 1983) since it is primarily stabilized by pressure insensitive hydrogen bonds (Heremans, 1995). However, the effects on the myofibrillar proteins is more complex. It has been shown that high pressures enhance denaturation and gelation of the structural proteins of muscle (myosin and actomyosin) (Cheftel and Culioli, 1997). Thus, depolymerization of actin and actomyosin and solubilization of myofibrillar proteins can be induced by high-pressure treatment. Cheftel and Culioli (1997) and Gilleland et al. (1997) showed that myosin denaturation and concomitant disulfide bond formation occurred during pressure-induced surimi gelation (300 MPa at 5 °C for 30 min). O'Shea et al. (1976) showed that pressure promoted myosin aggregation to form dimers, whereas F-actin was depolymerized: Ikkai and Ooi (1969) showed that although actomyosin disaggregates under pressure it does not dissociate into actin and myosin in the absence of ATP. Ikeuchi et al. (1992) concluded that F-actin denaturation plays an important role in the increased thermal gelation of pressurized actomyosin (to 200 MPa), and Sano et al. (1989) also showed that the pressure denaturation of actin increased the rigidity of gels formed by heating at 80 °C. Myosin in low ionic strength solution (0.1 M KCl and pH 6.0) formed a fine gel network at 280 MPa (Yamamoto et al., 1990), but Yamamoto et al. (1993) showed that pressures up to 210 MPa did not promote gelation of myosin in high ionic strength solution (0.5 M KCl, pH 6.0). Aggregation was similar to that induced by heat treatment in that a one-headed myosin molecule was formed by the two myosin heads interacting. Increasing pressure and time caused the formation of a clump with the myosin heads packed in the center and the tails extended radially. However, tail to tail interaction was not seen at these pressures (Yamamoto et al., 1993).

Our previous studies on whole cod muscle (Angsupanich and Ledward, 1998) have shown that pressure treatment produces very different textural characteristics to those seen in both raw and cooked fish. Moreover, DSC data showed that unlike thermally induced gels, which are mainly stabilized by hydrophobic interactions and disulfide bonds, pressure-induced gels appeared to be stabilized, at least partially, by hydrogen bonding.

Myosin is the major component of skeletal muscle and plays a significant role in gel formation. Thus, the aims of this study were to investigate the effect of pressure on isolated myofibrillar protein and myosin from cod and to compare the effects with those found on the myofibrillar proteins of turkey breast.

MATERIALS AND METHODS

All chemicals used were analytical grade, and fresh chilled cod (*Gadus morhua*) fillet and turkey breast were obtained from local retail outlets.

The fish and turkey muscles and their subsequent extracts were kept at 4 °C at all stages of preparation. All solutions and buffers were precooled to 4 °C before use.

Preparation of Myofibrillar Protein. Myofibrillar protein from cod was prepared by the method of MacDonald and

* Corresponding author. Tel: +44 1189318715. Fax: +44 118 9310080. E-mail: D.A.Ledward@afnovell.reading.ac.uk.

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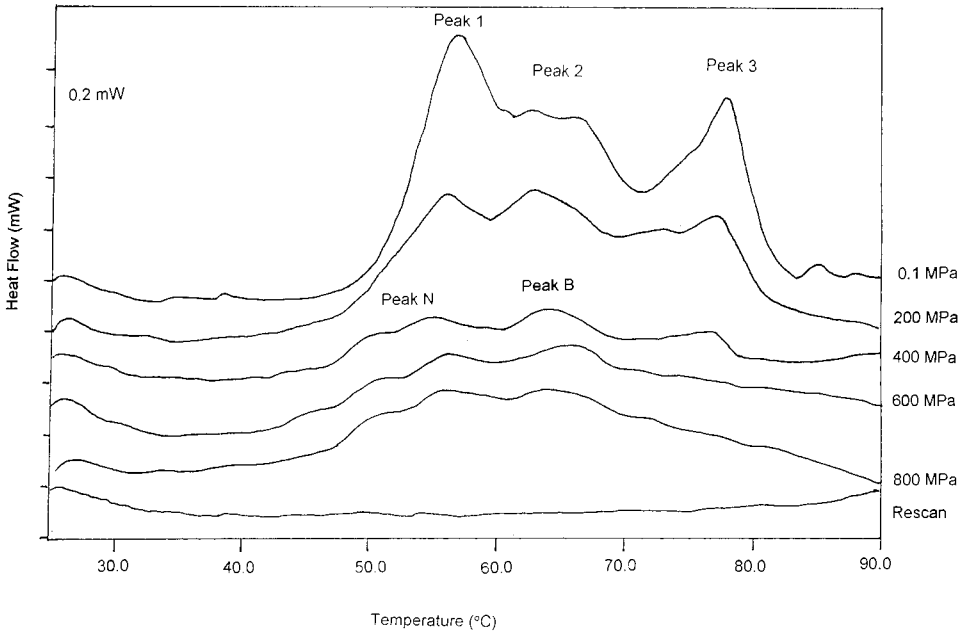

Figure 1. Thermograms of turkey breast muscle, heated at 10 °C min^{-1} after treatment at different pressures in air for 20 min at ambient temperature. Peaks 1 and 2 correspond to myosin, and peak 3 corresponds to actin. Peak N represents a structure produced after pressure treatment.

Lanier (1994). The concentration of extracted myofibrillar protein (pH ≈ 7, I = 0.3) estimated to be about 7.3 ± 0.9% was determined by drying to obtain the solid content (8.4%) and subtracting from this the estimated potassium chloride content (1.1%). Turkey breast myofibrillar protein was prepared by the method of Northcutt et al. (1993). The extracted sample had a solid content of 17.0%, and the concentration of salt (sodium chloride, magnesium chloride, ethylenediamine tetraacetic acid (EDTA), and sodium phosphate) was 3.1% giving an estimated protein concentration (pH ≈ 7.2, $I = 0.75$) of 13.9 ± 0.1%. Attempts to prepare turkey myofibrillar protein by the method of MacDonald and Lanier (1994) yielded samples of low native myosin content, as assessed by differential scanning calorimetry.

Preparation of Myosin. Cod and turkey breast myosin preparation was based on the method of Martone et al. (1986) as modified by Park and Lanier (1989). The cod myosin concentration (pH ≈ 7, $I = 0.24$) was 11.3 ± 3.1% (solid content 12.2%, concentration of potassium chloride and Tris 0.9%), and that of turkey breast (pH ≈ 7, $I = 0.24$) was 4.2 ± 0.9% (solid content 5.1%, concentration of salts 0.9%).

Pressure Treatment. Each protein or meat sample (10 g) was packed in Multivac bags (Bosley, International, NL) in the presence of air and subjected to pressures in the range of 200−800 MPa for 20 min at ambient temperature in a prototype Stansted 'Food-Lab' high-pressure rig (Stansted Fluid Power Ltd, Stansted, UK) as described previously (Angsupanich and Ledward, 1998). The pressure treated samples were analyzed immediately after treatment. All experiments were carried out in triplicate.

Heat Treatment of Cod Myosin Sample. Cod myosin samples (10 g) were sealed in Cryovac bags and heated in a water bath at 40−80 °C for 10 min and cooled with running water.

Differential Scanning Calorimetry (DSC). DSC was performed on a Perkin-Elmer DSC7 with TAC7/DX thermal analyzer controller. The instrument was calibrated using indium (mp 156.6 °C, ΔH fusion 6.8 cal/g). A 10−20-mg sample was weighed into a standard 40 μL aluminum pan, then hermetically sealed, and heated from 10 to 95 °C at 10 °C/min. An empty pan was used as reference. A rescan from 10 to 95 °C was carried out on all samples to check for reversibility.

Sodium Dodecyl Sulfate Polyacrylamide Gel Electrophoresis (SDS−PAGE). SDS−PAGE was performed on gels of 10% polyacrylamide containing 1% SDS (Laemmli, 1970). A 0.2-g sample of concentration indicated in the Materials and Methods was stirred with 10 mL of Tris buffer (pH 7.5) containing 8 M urea, 2% SDS, and 2% 2-mercaptoethanol for 24 h and filtered (Whatman No. 5). The filtrate was mixed with an equal volume of 0.125 M Tris buffer (pH 6.8) containing 10% SDS, 0.002% 2-mercaptoethanol, and 0.002% bromophenol blue, and 10 μL of protein solution was applied to each well. Separation used power at 150 V and 70 A, and fixation and staining by coomassie brillant blue was as described by Neuhoff et al. (1988). SDS−PAGE without 2-mercaptoethanol was performed similarly but omitting this compound from all solutions.

Texture Profile Analysis (TPA). Untreated and treated (400 MPa for 20 min) samples of cod fillet (2 × 2 × 1.5 cm) were heated in a water bath at 50 °C for 10 min. In addition, samples of some of the untreated, cooked samples were pressure treated at 400 MPa for 20 min. All samples were dried with filter paper and kept at 4 °C for 1 h before analysis. Texture profile analysis was carried out with a Stable Microsystem type (version 3.7G) at ambient temperature (≈20 °C). All samples were compressed to 50% of their height at 2 mm/s using a cylindrical-shaped piston 38 mm in diameter. The hardness was determined from the area underneath the first curve (Bourne, 1982).

Two-way analysis of variance was carried out using the Statistical Analysis System (SAS), SAS Institute Inc., Cary, NC. Differences between means were analyzed by a LSD range test at a significance level of $p \leq 0.01$.

RESULTS AND DISCUSSION

Appearance of the Samples. The fresh turkey breast muscle was translucent and red in color, but on pressure treatment at 200 MPa it became slightly opaque. With increasing pressure it became more opaque, and the redness decreased so that it appeared similar to cooked muscle.

The untreated isolated cod myofibrillar protein was a soft, opaque white paste, which when pressurized at 200 MPa formed a soft white gel that became more rigid with increasing pressure. The isolated turkey myofibrillar protein was a light brown paste and on pressure treatment at 200 MPa formed a very weak gel. The

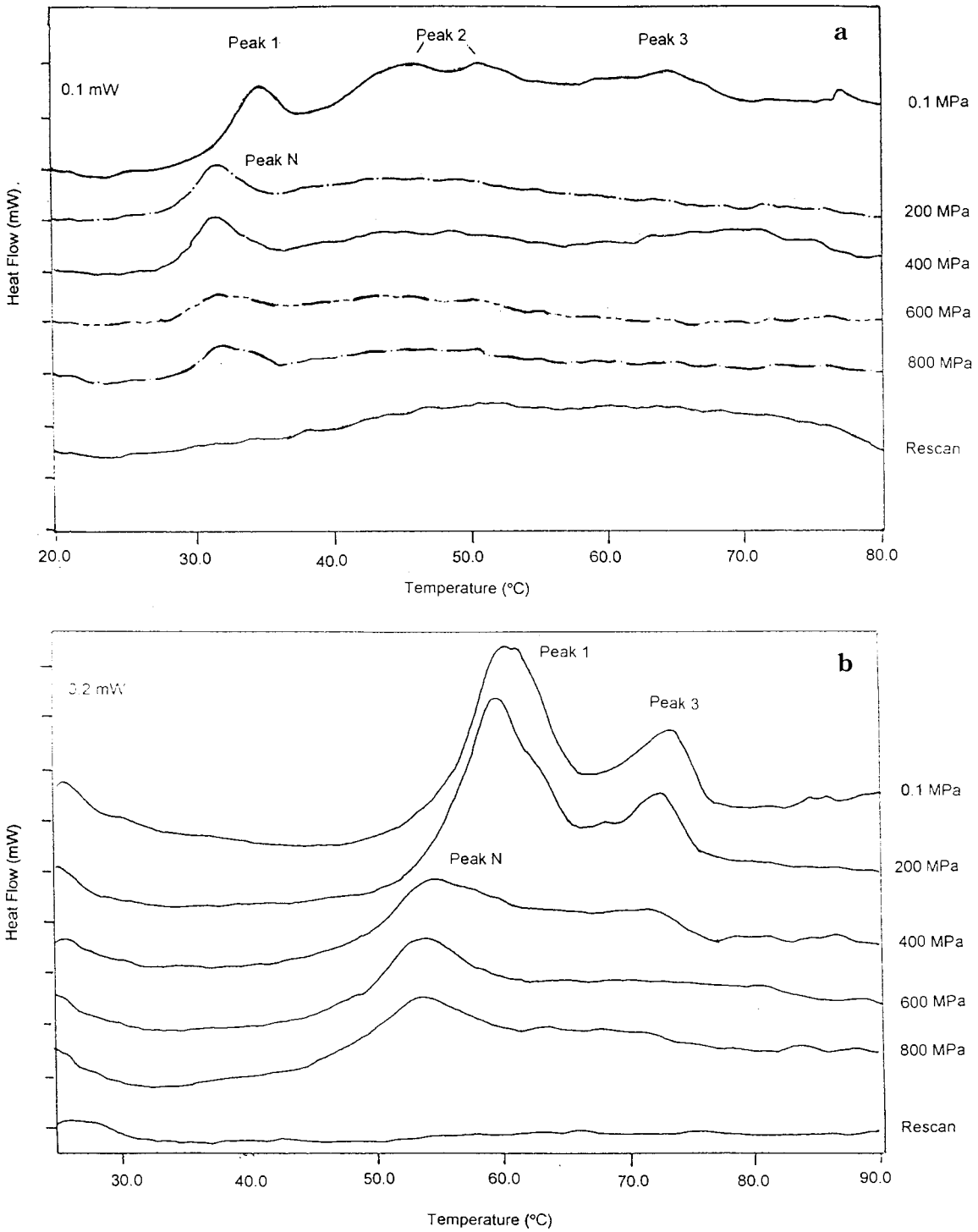

Figure 2. Thermograms of myofibrillar protein from cod (a) and turkey (b), heated at 10 °C min^{-1} after treatment at different pressures in air for 20 min at ambient temperature. Peaks 1 and 2 correspond to myosin, and peak 3 corresponds to actin. Peaks N represents a structure produced after pressure treatment.

isolated cod and turkey myosins were also soft white pastes but, they formed more transparent gels than the myofibrillar protein samples when treated at 200 and 300 MPa, respectively. These gels became harder with increasing pressure. When pressure treated at 800 MPa, all samples lost some moisture.

Differntial Scanning Calorimetry. *Whole Muscle.* Figure 1 shows the thermograms of intact turkey breast muscle when treated at pressures from 0.1 to 800 MPa at room temperature for 20 min. Three discernible peaks are seen at 58.5, 66.7 (doublet), and 77.8 °C. Peaks 1 and 2 correspond to myosin denaturation, and peak 3 is associated with actin denaturation. Mertens and Vold (1976) found three transition peaks for chicken at 57, 65, and 80 °C, while Dudziak et al. (1988) found three distinct peaks at 53, 61, and 69 °C. Treatment at 200 MPa decreased the size of all these peaks, and after treatment at 400 MPa they could not be seen (Figure 1) although peaks are seen of lower stability (N and B). We have previously shown (Angsupanich and Ledward, 1998) that, in intact cod muscle, pressure treatment causes myosin to denature at 100−200 MPa, although actin was slightly more resistant to pressure. The loss of the myosin peaks also gave rise to a hydrogen bonded

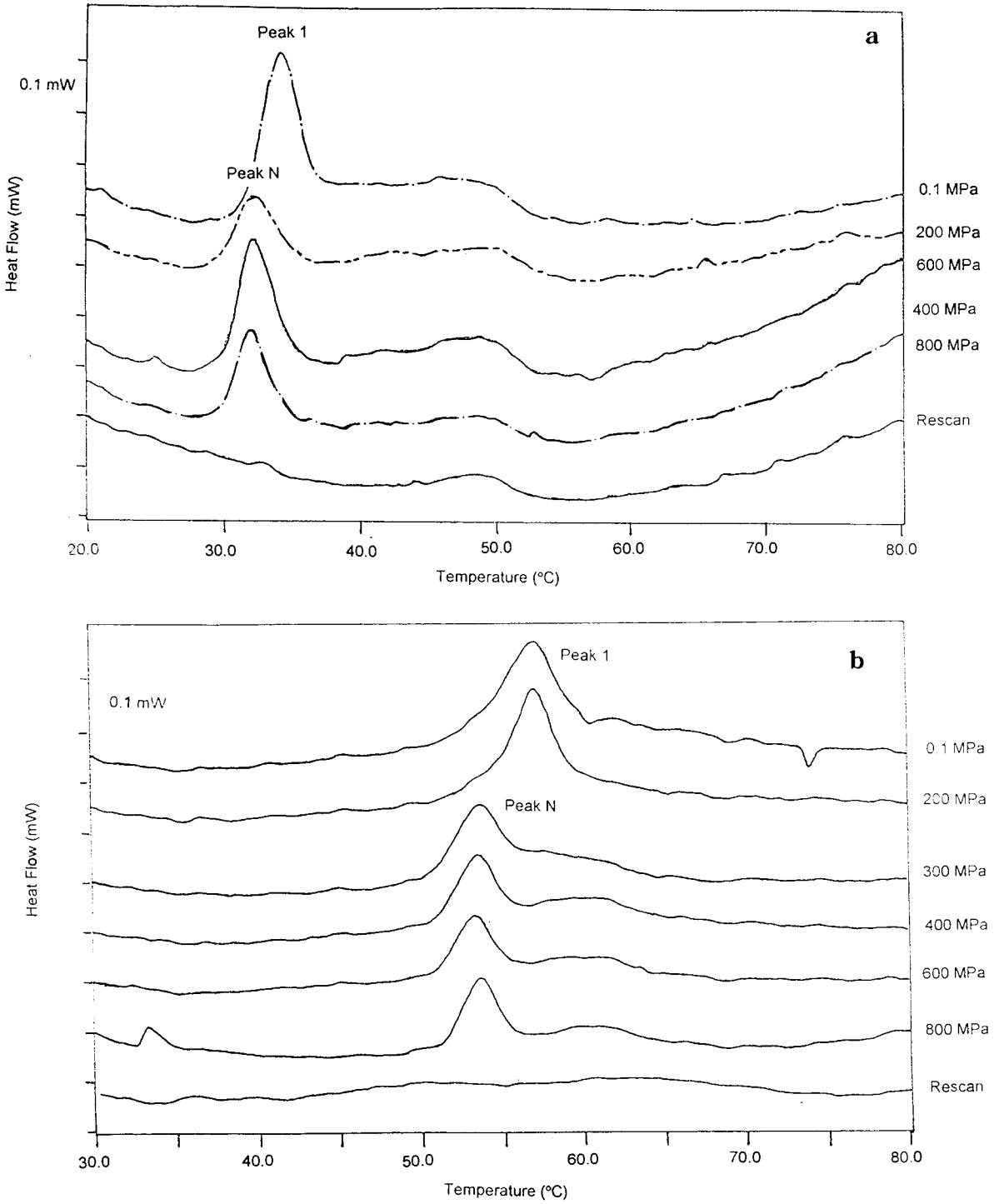

Figure 3. Thermograms of myosin from cod (a) and turkey (b), heated at 10 °C min^{-1} after treatment at different pressures in air for 20 min at ambient temperature. Peak 1 corresponds to myosin. Peak N represents a structure produced after pressure treatment.

network that had a lower thermal stability than the native myosin; it was also suggested that, in cod, a further hydrogen bonded structure was formed at still higher pressures (>400 MPa). It is seen from Figure 1 that in many respects turkey muscle responds similarly to cod, although the individual proteins are more stable to pressure and their barosensitivity is similar to those of pork muscle (Cheah and Ledward, 1996). Thus peaks appear (peaks N and B) after treatment at 400 MPa that are not markedly affected by further increases in pressure (Figure 1). Peak N is less heat stable than the native myosin and may represent a new or modified structure or a residual pressure-resistant structure. It is possible that peak B corresponds to collagen in the tissue, which is known to be relatively pressure insensitive (Gekko and Koga, 1983).

Isolated Proteins. *Myofibrillar Protein.* The thermograms of the untreated and pressure-treated cod and turkey myofibrillar proteins are shown in Figure 2. Four endothermic transitions at 34.2, 43.8, 50.6, and 64.9 °C are observed in the cod sample. Previous workers have attributed peaks 1 and 2 (doublet) in the thermogram for cod to myosin denaturation and peak 3 to actin (Hasting et al., 1985; Wu et al., 1985; Davies et al., 1988;

Table 1. Enthalpy Changes (ΔH, J/g Dry Sample) of New Structure and Myosin Denaturation in Intact Cod (pH ≈ 6.6) and Turkey (pH ≈ 6.0), Isolated Myofibrillar Protein from Cod (pH ≈ 7, I = 0.3) and Turkey (pH ≈ 7.2, I = 0.75), and Isolated Myosin (pH ≈ 7, I = 0.24) following Treatment at the Pressures Shown for 20 min[a]

sample	treatment (MPa)	ΔH_N	ΔH_M
intact cod[b]	0.1	0	8.08 ± 0.73
	100	0.78 ± 0.31	3.46 ± 0.72
	200	0.67 ± 0.05	3.14 ± 0.31*
	300	0.75 ± 0.05	1.79 ± 0.41*
	400	1.08 ± 0.05	2.15 ± 0.36*
	600	0.70 ± 0.15	1.86 ± 0.30*
	800	0.87 ± 0.10	1.55 ± 0.49*
isolated cod myofibrillar protein	0.1	0	4.86 ± 0.50
	200	1.40 ± 0.13	indistinct
	400	1.39 ± 0.12	indistinct
	600	0.74 ± 0.12	indistinct
	800	0.87 ± 0.12	indistinct
isolated cod myosin	0.1	0	2.15 ± 0.14
	200	2.13 ± 0.25	indistinct
	400	2.23 ± 0.30	indistinct
	600	2.29 ± 0.53	indistinct
	800	2.26 ± 0.19	indistinct
turkey breast	0.1	0	3.15 ± 0.22
	200	0	1.36 ± 0.16
	400	0.52 ± 0.10	0.48 ± 0.16*
	600	0.83 ± 0.04	0.26 ± 0.08*
	800	0.69 ± 0.17	0.31 ± 0.18*
isolated turkey myofibrillar protein	0.1	0	1.35 ± 0.41
	200	0	1.29 ± 0.35
	400	0.84 ± 0.22	indistinct
	600	0.69 ± 0.19	indistinct
	800	0.75 ± 0.06	indistinct
isolated turkey myosin	0.1	0	4.17 ± 0.74
	200	0	6.22 ± 1.24
	300	2.94 ± 0.00	indistinct
	400	2.58 ± 0.20	indistinct
	600	2.17 ± 0.02	indistinct
	800	2.43 ± 0.56	indistinct

[a] All values are the means ± standard deviation of three or four determinations. ΔH_N is the enthalpy change of denaturation for new peak (peak N). ΔH_M is the enthalpy change of denaturation for myosin (* includes peak B, Figure 1). [b] From Angsupanich and Ledward (1998).

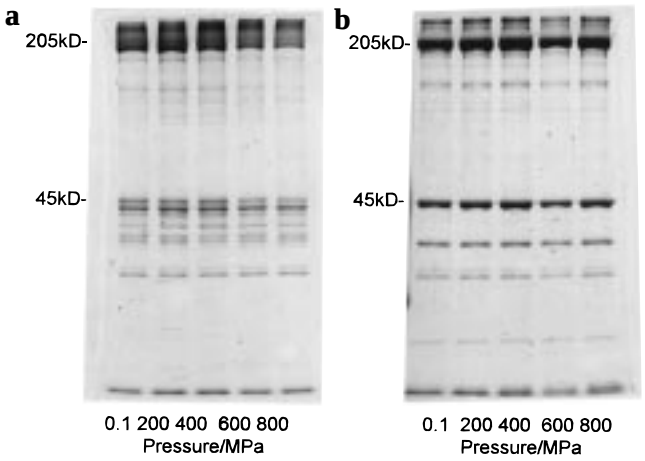

Figure 4. SDS−PAGE analysis of solubilized protein in the absence (a) and in the presence (b) of 2-mercaptoethanol for cod myofibrillar protein after treatment at different pressures for 20 min. Lane 1, untreated; lane 2, 200 MPa; lane 3, 400 MPa; lane 4, 600 MPa; and lane 5, 800 MPa.

Howell et al., 1991). However, the extracted myofibrillar protein from cod has peaks at lower temperatures than those of whole cod muscle (cf. 34 and 42 °C), indicating purification decreases the heat stability of the proteins (Wu et al., 1985). Comparison of Figures 1 and 2b show that in the turkey breast myofibrillar system no marked decrease in stability is apparent in myosin as a distinct peak is seen at 59.2 °C, corresponding to myosin denaturation. There is some loss of actin stability as compared to the intact muscle (Figure 1), and peak 2 is less distinct in the isolated myofibrillar systems suggesting that sarcoplasmic proteins may contribute to this transition.

In cod myofibrillar protein, pressure treatment at 200 MPa and above for 20 min led to a dramatic loss of all original peaks and the appearance of a low melting transition at 31.6 °C (peak N). These results agree with the previous study of whole cod muscle (Angsupanich and Ledward, 1998). However, the new structure (peak B) melting in the range of 40−60 °C found in pressure-treated (300 MPa and higher) whole cod muscle (Angsupanich and Ledward, 1998) or turkey breast (Figure 1) was not seen in pressurized myofibrillar protein. This suggests that peak B does not relate to actomyosin denaturation but is related to other proteins such as collagen in cod and turkey muscle.

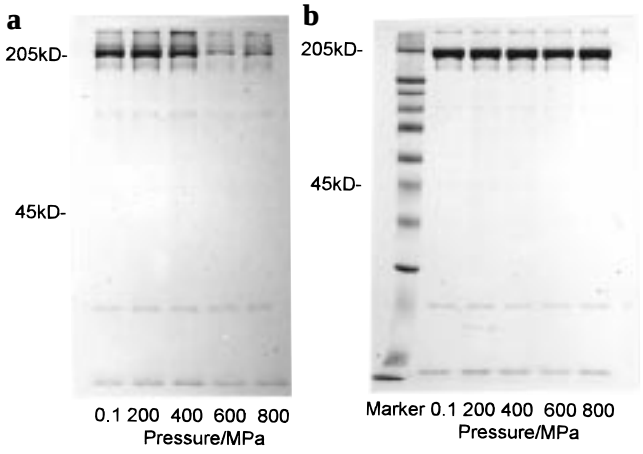

Figure 5. SDS−PAGE analysis of solubilized protein in the absence (a) and in the presence (b) of 2-mercaptoethanol for turkey myosin after treatment at different pressures for 20 min. Lane 1, molecular weight marker; lane 2, untreated; lane 3, 200 MPa; lane 4, 400 MPa; lane 5, 600 MPa; and lane 6, 800 MPa.

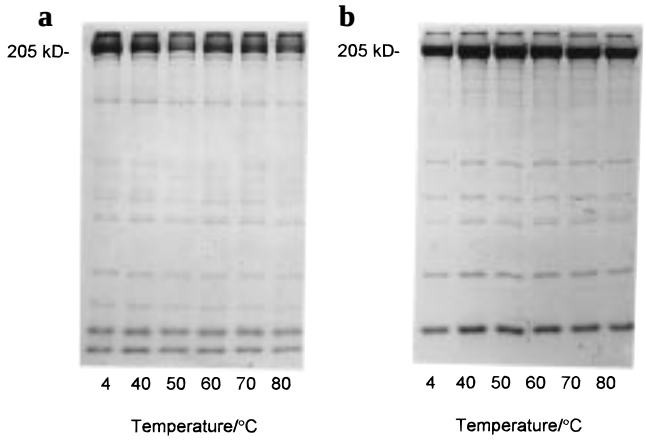

Figure 6. SDS−PAGE analysis of solubilized protein in the absence (a) and in the presence (b) of 2-mercaptoethanol for cod myosin after treatment at different temperatures for 10 min. Lane 1, untreated (4 °C); lane 2, 40 °C; lane 3, 50 °C; lane 4, 60 °C; lane 5, 70 °C, and lane 6, 80 °C.

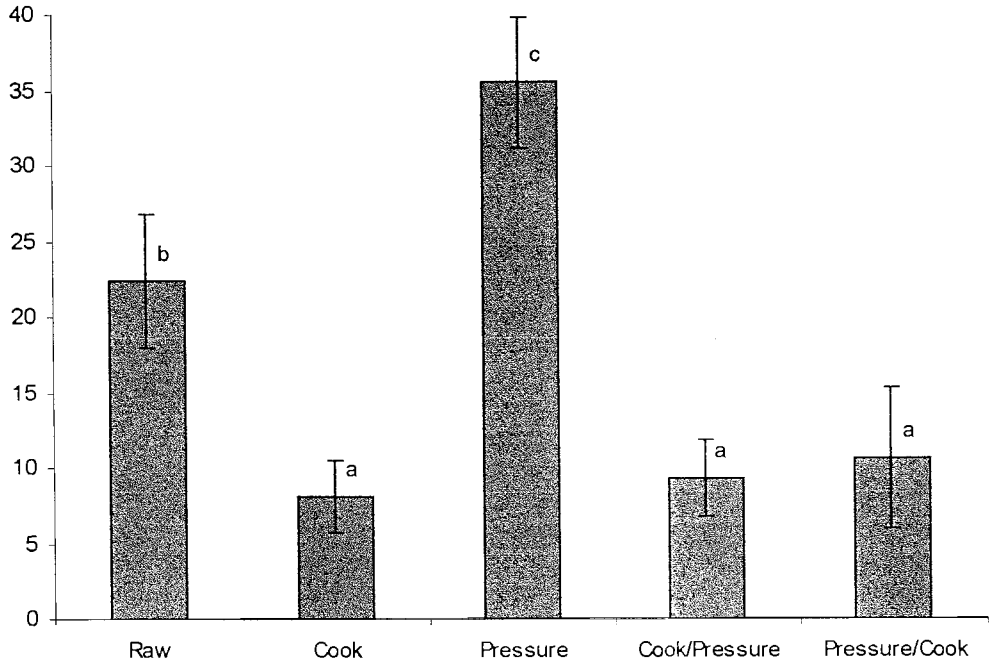

Figure 7. Effect of pressure (400 MPa) or temperature (50 °C) or a sequential combination of both treatments on the hardness of cod muscle. All values are the means ± standard deviations of 15 determinations (five determinations on each of three fish). Bars with the same subscripts are not significantly different ($p \leq 0.01$).

When isolated myofibrillar protein from turkey was pressure treated at 200 MPa, there was no change in any of the peaks (Figure 2). However, pressures of 400 MPa and above caused loss of the myosin peak and major loss of actin structure and a 'new' peak (peak N $\approx$ 53−54 °C) is seen, similar to that reported for cod muscle (Angsupanich and Ledward, 1998) and found in this study for cod myofibrillar protein (Figure 2a).

Myosin. For the extracted cod myosin, pressure treatment (at and above 200 MPa at room temperature for 20 min) led to a loss of the only major peak and the formation of a new or residual pressure-resistant structure that displayed an endothermic peak (peak N) at lower temperature (≈32 °C) (Figure 3a) in the same position as found in pressure-treated cod myofibrillar protein (Figure 2a) and whole cod muscle (Angsupanich and Ledward, 1998). This structure was relatively insensitive to further pressure treatment up to 800 MPa.

As with the myofibrillar protein, the purified cod myosin peaks have lower transition temperatures than those found in the intact muscle (42 °C). This may be due to the added salt solubilizing the myofibrillar protein and thus making the protein less thermally stable (Park and Lanier, 1989). No such effect was seen in turkey, although both myosins were in the same salt and pH environments.

The thermogram for the turkey myosin sample (Figure 3b) showed that denaturation occurred at 300 MPa. Cheah and Ledward (1996) found that in pork both myosin and actin were denatured after pressure treatment at 300−400 MPa. Again, as with the cod myosin, a structure was formed on denaturation of the myosin (peak N in Figure 3) of lower thermal stability than the native protein.

On rescanning whole muscle, isolated myofibrillar protein, and myosin, no peaks were seen indicating that the new or residual structure could be destroyed by heat, suggesting that it is primarily stabilized by hydrogen bonds, the only weak thermally labile linkages to break endothermally (Finch and Ledward, 1972; Finch et al., 1974).

Recently, Gilleland et al. (1997) found that pressure treatment at 300 MPa, 5 °C, for 30 min caused partial denaturation of myosin and complete denaturation of actin in a surimi sample prepared from Alaska pollock (*Theragra chalcogramma*), and they suggested that the high pressure-induced surimi gel was stabilized by intermolecular hydrophobic interactions. In contrast, we have suggested that hydrogen bonds are major stabilizing forces in pressurized myofibrillar protein samples, since such bonds break endothermally (Angsupanich and Ledward, 1998). The present results support this latter view. The enthalpies for the myosin peaks in the untreated and pressure-treated samples are summarized in Table 1. It is seen that although turkey myosin is, as expected, more stable than that of cod after pressure treatment both form or retain pressure-stable structures that melt several degrees lower than the temperature at which the native myosin denatures. It is possible that the 'new' structure found at the higher pressures in both cod and turkey myosin is due to the myosin heads aggregating leaving the hydrogen bonded tails or rods intact. It is interesting to note that the pressure induced or resistant structure 'melts' at the same temperature in the intact muscle, myofibrillar protein, and isolated myosin although there are inevitable differences in myosin concentration and ionic environment (cf. Figures 1−3). The volume of the denatured state for most proteins is less than that of the native (Isaacs, 1981), and thus, even if hydrogen bonded, there will be a tendency to unfold although the process might well in many circumstances be reversible (Cross et al., 1983). However, if the rods remained intact, one would expect the relative enthalpies of the denatured states to be a direct and constant function of that of the native myosin. This is clearly not so, as it can be seen in Table 1 that in the intact muscle and isolated myofibrillar protein the enthalpy change associated with peak N is less than half that of the native

myosin while in the isolated myosin they are a considerably higher percentage, suggesting that in the absence of other proteins more hydrogen bonds can form. There is also some evidence from Table 1 that the enthalpy (in the intact muscle and isolated myofibrillar protein) associated with peak N decreases with increasing pressure treatment. This is not surprising if, at these higher pressures, other structures are modified that inhibit the formation of the structure giving rise to peak N. In the purified myosins, no such pressure dependence is seen.

Electrophoretic Studies. On preparation of the samples for SDS−PAGE, it was noted that the untreated isolated cod and turkey myofibrillar proteins and myosins were soluble in the SDS buffer without 2-mercaptoethanol while in the more severely heat- or pressure-treated samples some precipitate was found that was removed by filtration. Electrophoretic analysis (SDS−PAGE) in the presence and absence of 2-mercaptoethanol for all samples (cod and turkey) studied in this work show that myosin aggregated by disulfide bond formation after treatment at high pressure (600 MPa and above). In the absence of 2-mercaptoethanol treatment at 600 and 800 MPa for 20 min complete solubilization was not achieved, and the concentration of myosin heavy chain, with molecular mass of 205 kDa, decreased. In the presence of 2-mercaptoethanol complete solubilization was achieved, and no difference in electrophoretic pattern was seen. Typical results for cod myofibrillar protein and turkey myosin are shown in Figures 4 and 5, respectively. The results from cod myosin show a pattern similar to that of turkey (data not shown). These results suggest that myosin heavy chain forms disulfide bonds on pressure treatment. However, for turkey myofibrillar protein in the absence of 2-mercaptoethanol, no significant difference in electrophoretic pattern was seen, although some loss of solubility occurred. This may be due to the addition of DL-dithiothreitol (DTT) during extraction or the higher salt content of the extract, although in whole cod muscle there was also little evidence of disulfide bond formation (Angsupanich and Ledward, 1998)

Electrophoretic analysis of heated cod myosin (40−80 °C for 10 min) in the presence and the absence of 2-mercaptoethanol are shown in Figure 6. The myosin heavy chain concentration at 205 kDa decreased significantly, in the absence of 2-mercaptothanol, when heated at 50 °C and higher. These results agree with those reported by Lee and Lanier (1995) and confirm that both pressure and heat treatment give rise to disulfide bond formation. It is thus likely that the differences in texture between heat- and pressure-treated cod samples are mainly due to the relative number of hydrophobic interactions and hydrogen bonds (Angsupanich and Ledward, 1998).

Texture Profile Analysis. Hardness as estimated by texture profile analysis on whole cod samples cooked, pressure-treated, cooked and then pressure-treated, and pressure-treated and then cooked are shown in Figure 7. It is seen there is no significant difference ($p < 0.01$) between the cooked and the cooked and then pressure-treated samples, while, as found previously (Angsupanich and Ledward, 1998), the pressure-treated sample is much harder than the others. However, when the pressure-treated sample was heated to 50 °C, the hardness decreased to a value similar to that found for the cooked. These observations further suggest that thermally labile hydrogen bonds help stabilize the pressure set gel. It is our view that on pressure treatment at 400 MPa a predominantly hydrogen bonded network is set up that readily melts at 50 °C, allowing some disulfide bonds and numerous hydrophobic interactions to form in a heat set gel. However, the gel network formed by heat at 50 °C will be stabilized by disulfide bonds and hydrophobic interactions. Although the hydrophobic interactions will break on pressure treatment and reform on pressure release, there will be little, if any, scope for additional hydrogen bond stabilization.

CONCLUSION

These experiments support the previous work on whole cod muscle (Angsupanich and Ledward, 1998) that suggested that pressure-induced myosin denaturation leads to very different gels to those produced by heat due to the formation of structures involving hydrogen bonds, though disulfide bonds make a contribution at higher pressures. The involvement of hydrogen bonds is indicated because destruction of this heat labile structure leads to a hardness value similar to that obtained by heat alone. A heat set myosin gel is primarily stabilized by disulfide bonds and hydrophobic interactions. Although cod and turkey myosin differ in both pressure and temperature stability, it seems likely their pressure-induced gelation mechanisms are similar.

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