High-pressure processing of fish and fish products

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High hydrostatic pressure has recently been applied in food processing, and several commercial fruit and vegetable products have already been put on sale. High hydrostatic pressure results in protein denaturation, resulting in inhibition of some inherent enzymatic activities and of the biogenic activity of some microorganisms. However, high pressure also accelerates lipid oxidation in muscle tissues. Recent intensive research on the effects of high hydrostatic pressure on fish tissues has gradually revealed the benefits and defects of this novel processing technology.

High-pressure technology has been used in non-food applications for many years[1], and high pressure was first applied to the processing of foods and foodstuffs in 1899, when it was used to increase the shelf life of milk[2]. Subsequent progress in evaluating the effectiveness of high-pressure treatment has led to use of the technology to prevent the growth of food microorganisms[3] and to reduce spoilage caused by enzymatic activities of microorganisms[4]. Food-processing applications of high-pressure technology are not restricted to sterilization; other uses include: improvement of the tenderness of pre-rigor beef muscle[5–7], selective removal of β-lactoglobulin from whey concentrates[8], and acceleration of bovine milk curdling by rennet[9]. In 1990, several fruit jams came onto the Japanese market, representing the first application of high pressure to food processing on a commercial basis[10]. In the next year, high-pressure grapefruit juice appeared on the market in Japan. High pressure has subsequently been applied to the processing of orange juice to prevent the development of a bitter taste[11], and for primary sterilization, resulting in a juice requiring a lower level of dimethylsulfide than heat-sterilized juice[11].

For marine products, however, most applications of high pressure have been carried out on only a laboratory scale (Table 1). There have been some proposals for the commercial utilization of high pressure in seafood processing. Kamaboko products are usually produced by adding 2–2.5% NaCl to surimi with subsequent thorough mixing, shaping, steaming and cooling. The heat-induced gel of surimi thus obtained has many small air spaces inside, and the surface appears dimpled. If, however, gelation is induced by pressure rather than heat, the gel appears glassy and smooth, and retains the aroma of raw surimi[12]. Further detailed discussion of surimi and kamaboko technology is given in our earlier review[13].

In surimi processing, large amounts of sarcoplasmic proteins are washed out into the waste water[14]. Unlike the myofibrillar proteins, sarcoplasmic proteins do not form gels on heating. In order to utilize the sarcoplasmic proteins that are normally lost in the waste water, they could be recovered by coagulation and then pressurized at over 300 MPa. Pressure-induced gels thus obtained have a springy texture like that of sausage, and could be used in surimi and related products[15]. Furthermore, high-pressure treatment (500 MPa at 0°C for 10 minutes) is effective in killing *Vibrio parahaemolyticus*, *Vibrio cholerae* non-01 and *Vibrio mimicus* cells in sea urchin eggs (Table 1), while retaining the original flavor and taste[16] (in Japan, sea urchin eggs are usually consumed raw or salted, without heating). High hydrostatic pressure has also been applied to the preparation of alginate gels that are enriched in calcium, have a unique texture, and can be used as carriers for immobilized enzymes[17]. However, to date no commercial seafood products have been produced using high-pressure treatment.

Effects on the appearance of fish meats

Raw cod muscle appears slightly transparent, whereas raw mackerel muscle appears reddish and fresh. When the muscles of both fish are treated by high hydrostatic pressure, they become opaque, as if the muscle had been grilled or boiled. In order to evaluate the color changes quantitatively, a digital color difference meter can be used. When cod muscle was subjected to high pressure (up to 608 MPa for 15 minutes) the L-value (which gives an effective index of visual lightness)[18] increased with increasing hydrostatic pressure, although an initial pressurizing period of up to 30 minutes had no effect on the L-value of the treated cod muscle. The L-value of the mackerel sample also increased with increasing hydrostatic pressure. These results showed clearly that the color of the fish meat becomes lighter with increasing hydrostatic pressure (Fig. 1). The a-value, which is normally used as an index of visual redness, decreased remarkably in mackerel muscle when it was subjected to increasing hydrostatic pressure up to 608 MPa for 15 minutes. The b-value, an effective index of yellow color when it is positive, showed no significant changes after the high-pressure treatment. In summary, changes in the measured L-, a- and b-values of cod and mackerel muscles coincide well with the visual changes in color. Similar changes in appearance occurred when Alaska pollock surimi was treated at up to 500 MPa (Ref. 19).

Effects on fish protcins

Since the properties of their constituent proteins have been characterized qualitatively and quantitatively,

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myofibrils of normal fish muscle, which essentially determine the nature of fish meat, are generally used to evaluate effects of high pressure on fish protein. Myofibrils are prepared for high-pressure treatment by removing sarcoplasmic (water-soluble) proteins (e.g. glycolytic enzymes and heme proteins) and stroma proteins (e.g. collagen) from minced fish muscle. Myofibrils occupy 65–80% of total muscle proteins and are composed of contractile proteins (e.g. myosin and actin), regulatory proteins (e.g. troponin and tropomyosin), elastic proteins (e.g. connectin) and some minor proteins[20].

When carp myofibrils were pressurized at 150 MPa for 30 minutes, electron microscope studies revealed that the arrangement of myofibrils was destroyed and that the filaments cohered to each other so that their striation pattern was lost. Suzuki *et al.*[21] reported that a marked progressive disarrangement of myofibrils occurred in beef meat subjected to increasing pressure. By contrast, myofibrils treated at 38°C for 2 hours still exhibited a striped appearance, although some unique structural changes had occurred[22]. These results suggest that the mechanism for denaturation of myofibrillar protein by high hydrostatic pressure treatment is different from that for denaturation induced by heat.

The mobilities of myosin heavy chain and actin in sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS–PAGE) were not altered by either of these treatments[22]. Similar results have been reported for Alaska pollock[19] and for beef myofibrils[21]. Therefore, high-pressure treatment of fish muscles causes neither degradation nor covalent cohesion of myofibrillar proteins, according to the SDS–PAGE analysis. However, it has been found that when normal muscle from cod and mackerel is subjected to high-pressure treatment, bands corresponding to certain extractable proteins disappear from SDS–PAGE gels without the expected appearance of low molecular weight bands (Fig. 2), which suggests that, rather than being degraded by high-pressure, certain sarcoplasmic proteins become covalently linked together and are thus resistant to extraction with SDS[18].

Myosin is a dominant component of fish myofibrils and has an ATP hydrolytic activity in the presence of 3–5 mM Ca^{2+} (Ca^{2+}–ATPase activity). Thermostability of the Ca^{2+}–ATPase activity of fish myofibrils, which is closely correlated with their normal habitation temperatures, is an indicator of the extent of denaturation of fish myofibrillar proteins[23]. When carp myofibrils were treated at 35°C or 40°C for up to 30 minutes, Ca^{2+}–ATPase activities exhibited a negative linear correlation with the treatment time[24]. Therefore, inactivation of Ca^{2+}–ATPase activity by heat treatment follows a first-order chemical reaction. On the other hand, carp myofibrils that were pressurized separately at 125 MPa and 150 MPa showed a double-linear relationship, which had break points at 60 minutes and 90 minutes, respectively (Fig. 3). This discrepancy again suggests that the mechanism of denaturation of fish myofibrils by heat is different from that of denaturation induced by high-pressure treatment, which might cause depolymerization of myofibrillar proteins[25–27] or aggregation of myosin[28] as reported for mammalian meats.

Table 1. The conditions currently used in high-pressure treatment of fish and related products

Sample	Conditions	Ref.
Alaska pollock surimi	0–700 MPa; room temperature; 10 minutes	12
Tuna and squid meats	0–450 MPa; 25°C; 15 minutes	22
Carp myofibrils	0–200 MPa; 0°C; 30 minutes	22
Carp muscle	0–500 MPa; 0°C; 30 minutes	22
Alaska pollock surimi	200–500 MPa; 0°C; 10 minutes	19
Alaska pollock surimi	0–600 MPa; 0°C; 15 and 30 minutes	18
Minced meats of tuna, flying fish, sardine and Alaska pollock	0–500 MPa; room temperature; 30 minutes; and heated at 70°C for 20 minutes	50, 51
Sardine meat	0–400 MPa; room temperature; 30 minutes	37
Dehydrated sardine meat	0–180 MPa; room temperature; 60 minutes	36
Mackerel meat	0–600 MPa; 0°C; 15 and 30 minutes	18
Water-soluble proteins	50–500 MPa; 5°C; 20 minutes	14
Salmon meat	0–500 MPa; 5°C; 20 minutes	53
Marine yeast	0.1–50 MPa; 0–30 minutes	54
Fish surimi	0–500 MPa; 10 minutes	55
Frozen tuna block	0.1–150 MPa; 0–20°C; 60 minutes	56
Marine fish (9 species) and carp muscle	0–200 MPa; room temperature; 13 hours	52
Sea urchin eggs	0–500 MPa; 0°C and 17°C; 10 minutes	16
Alginate	0–900 MPa; 25°C; 30 minutes	17

Effects on inherent enzyme activities

A decrease in the level of ATP triggers the onset of rigor mortis; ATP and its related compounds degrade as follows:

$$ATP \rightarrow ADP \rightarrow AMP \rightarrow IMP \rightarrow HxR \rightarrow Hx \rightarrow X \rightarrow U$$

where ATP denotes adenosine-5′-triphosphate, ADP is adenosine-5′-diphosphate, AMP is adenosine-5′-monophosphate, IMP is inosine-5′-monophosphate, HxR is inosine, Hx is hypoxanthine, X is xanthine, and U is uric acid. In most fish species, a rate-determining step exists between X and U in a series of such reactions; as a result, HxR and Hx accumulate in fish

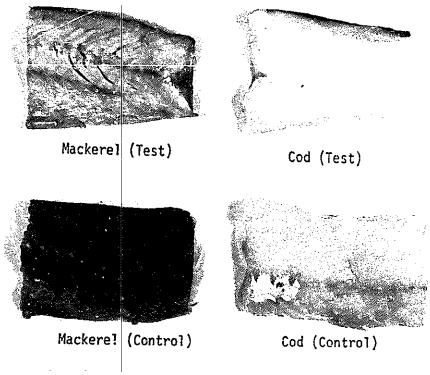

Fig. 1

Cod and mackerel fillets pressurized at 608 MPa for 15 minutes. The pressurized fillets ('Test') turned opaque, compared with the nonpressurized samples ('Control').

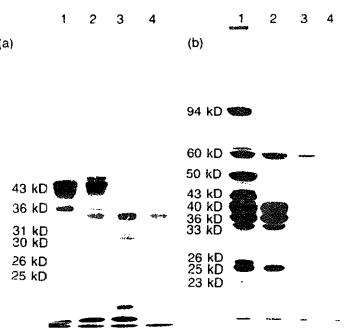

Fig. 2

SDS–polyacrylamide gel electrophoresis of the sarcoplasmic proteins from (a) cod and (b) mackerel muscles pressurized for 15 minutes. Lane 1, control; lanes 2, 3 and 4, protein profiles after hydrostatic pressure treatments of 202, 405 and 608 MPa, respectively. A specific loss of a major sarcoplasmic protein component with a subunit molecular mass of 43 kDa was noted in the pressurized samples from both cod and mackerel. Reprinted with permission from Ref. 18.

muscle. The ratio of HxR and Hx to the total amount of ATP and its decomposed compounds[29] is defined as the *k* value, which is an effective index of fish freshness. The breakdown of the ATP-related compounds listed above is usually catalysed by certain dephosphorylases inherent in fish muscles. ATP and its related compounds are generally heat-labile and heated fish muscle thus exhibits a relatively high *k* value, even though the raw fish sample is quite fresh and has a low *k* value. When carp muscle was treated with various high hydrostatic pressures of 200, 350 and 500 MPa, and subsequently stored at 5°C, suppression of the decrease in IMP level was observed in muscle tissue treated at 350 or 500 MPa[22]. These results strongly suggest that the enzymes involved in the degradation of ATP and its related compounds undergo protein denaturation and are deactivated during high-pressure treatment. IMP contributes to the development of umami taste in fish and fish products[30]; therefore, the degree of umami should increase with high hydrostatic pressure treatment because the enzymatic activities that catalyse the breakdown of IMP are suppressed under such conditions.

Another drastic change that occurs during the storage of fish muscle at low temperatures is the accumulation of free fatty acids that are released from glycerolipids by certain lipolytic enzymes[31–33]. When cod muscles were treated at 202 MPa for 15 minutes and subsequently stored at −2°C for 6 days, the level of free fatty acids increased as much as that in non-treated cod muscle. In muscle treated at ≥405 MPa, the fatty acid level did not increase and the phospholipid level did not decrease (Fig. 4). These observations suggest that the enzymatic degradation of phospholipids is successfully inhibited by pressurization at ≥405 MPa for more than 15 minutes.

Effects on the oxidation of fats, oils and tissue lipids

Marine lipids are characterized by a high level of polyunsaturated fatty acids (PUFAs) such as eicosapentaenoic acid and docosahexaenoic acid[34]. PUFAs are generally susceptible to autoxidation, and oxidative degradation of lipids in foods and foodstuffs during processing and subsequent storage directly affects the quality of the products, including their flavor, color, texture and nutritional value[35]. There have been relatively few studies of the effects of high hydrostatic pressure on fish oils. When extracted sardine oils were treated by high hydrostatic pressure of up to 506 MPa for 60 minutes, the peroxide value (POV) and thiobarbituric acid (TBA) number (both indicators of the degree of oxidation) of the oils did not change[36,37]. On the other hand, when cod muscles were exposed separately to high hydrostatic pressures of 202, 404 and 608 MPa for 15 and 30 minutes, the POV of the extracted oils increased with increasing hydrostatic pressure and processing time; even more pronounced effects of high-pressure treatment have been observed for mackerel muscle lipids[18]. Based on these results, it is concluded that isolated extracted marine lipids are relatively stable against autoxidation under high hydrostatic pressure up to 608 MPa. By contrast, the oxidation of lipids existing in the muscle is accelerated by high-pressure treatment. The POV levels of the oils extracted from the pressurized and subsequently refrigerated cod muscle were remarkably higher than that of non-pressurized and refrigerated cod muscle. Similarly, increased POV levels induced by high-pressure treatment have also been observed in mackerel muscle[18].

The results obtained from the studies described above have shown that fish meats may contain certain factors that accelerate lipid oxidation during high-pressure treatment. A model system consisting of extracted sardine oils (20%) thoroughly mixed with defatted sardine meats was treated at up to 108 MPa for 30 and 60 minutes, and subsequently stored at 5°C for several days. Both POV and TBA numbers were higher in the samples treated for the longer time period[36]. When the extracted lipids were treated in the absence of defatted sardine meat they were oxidized to only a slight extent after storage for the same length of time. By using water-washed sardine meat instead of defatted meat, the effect of water-soluble components existing in the sardine meat on the oxidation of sardine lipids could be evaluated. The first water washing of sardine meat removed ~10% of the total iron from the sardine meats; non-heme iron was eliminated faster than heme iron[36]. Therefore, certain metal ions may play an important role in promoting autoxidation of lipids in pressurized fish meat.

Effects on bacteria

The effect of high pressure on the microorganisms in food systems is of great interest, because it directly affects the utility of this treatment in food preservation. Many studies have been made of the effect of high pressure on microorganisms in milk[38], meats[39], fruits[40] and citrus juices[41]. To study similar effects in marine products, total plate counts of microorganisms in tuna meat and squid mantle flesh samples were taken before and after high-pressure treatment at 450 MPa and 25°C for 15 minutes[22]. The initial total plate counts for the tuna meat and squid mantle flesh used were 5.2×10^3 and 1.9×10^4 colonies per gram of tissue, respectively. The total plate counts of both samples decreased with increasing hydrostatic pressure and had fallen to below 300 colonies per gram of tissue in both samples by the end of the pressurization period. These results suggest that high-pressure treatment reduces microorganism activity. However, satisfactory sterilization could not be achieved using high-pressure treatment alone – the combination of high-pressure treatment with another sterilization method was required for efficient sterilization.

In order to inhibit or inactivate vegetative microbes, a combination treatment of moderate hydrostatic pressure and pasteurization has been proposed[42]. The sterilizing effects of high pressure (up to 400 MPa) on *Lactobacillus casei* and *Escherichia coli* were more effective at the lower temperatures of 0°C and 60°C (Ref. 43). The effects on *E. coli* cells and *Bacillus subtilis* spores of combining

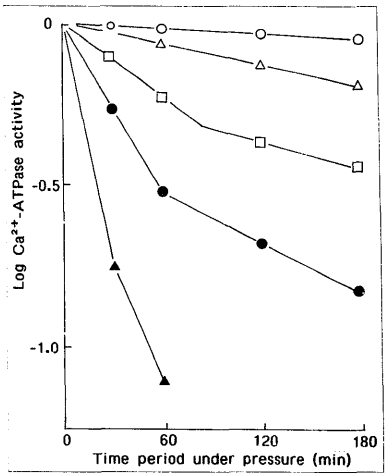

Fig. 3

Decrease in relative Ca^{2+}-ATPase activity of carp myofibrils during separate pressure treatments at 50 (○), 100 (△), 125 (□), 150 (●), and 200 (▲) MPa. The Ca^{2+}-ATPase activities exhibited a negative linear correlation with treatment time, while those exposed to high pressure for long periods showed a double-linear relationship (see text for details). Reprinted with permission from Ref. 22.

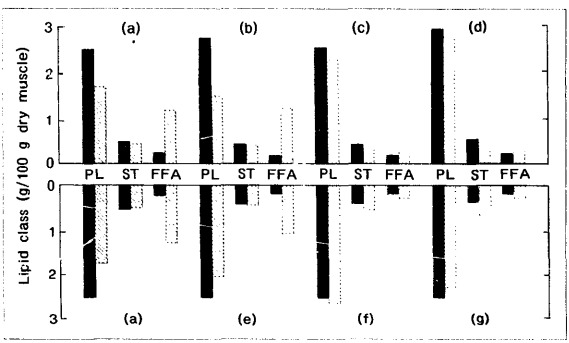

Fig. 4

The lipid class content of untreated cod muscle (a) and the changes in lipid class content after high-pressure treatment for 15 minutes (top) and 30 minutes (bottom) at a hydrostatic pressure of 202 MPa (b and e), 405 MPa (c and f) or 608 MPa (d and g). Closed bars and shaded bars represent the contents before and after storage at –2°C for 6 days, respectively. FFA, free fatty acids; PL, phospholipids; ST, free sterols. Levels of FFA in the samples pressurized at ≥405 MPa (c–g) were much lower than those of the control (a). Reprinted with permission from Ref. 18.

high pressure with exposure to alternating currents and surfactant treatment were also investigated in order to overcome the high pressure tolerance of these microorganisms[44]. However, the treatment of food and foodstuffs with such combination methods should be carried out under carefully controlled temperature conditions, since *Saccharomyces cerevisiae* cells became tolerant to a normally lethal hydrostatic pressure treatment of 150 MPa for 60 minutes when they were pre-incubated at 43°C for 30–60 minutes[45].

Future possibilities in food processing

In order to exploit the advantages of high-pressure treatment in the field of food processing, the following problems need to be solved.

- It is difficult to destroy most bacterial spores by high-pressure treatment alone. Some of the microorganisms in fish meat regenerate after the pressure is released, even when the meat has been pressurized at 450 MPa (Ref. 46). To achieve complete sterilization, the combination of a pressure treatment and another treatment, such as heating as well as cooling to below –20°C, seems to be required[47].

- To decrease the cost of pressurizing equipment, the upper limit of pressure that can be produced by the equipment should be lowered to the level that is sufficient for effective high-pressure treatment. In order to compensate for the reduced pressure ability of such equipment, other processing methods would be required in conjunction with high pressure, as already discussed.

On the other hand, certain superior characteristics of pressure treatment should be promoted; for example, high-pressure treatment is very effective in producing kamaboko with a very fine surface. Also, the flavor of raw oyster can be enhanced by pressure treatment[48]. Furthermore, most inherent enzymatic activities, which are undesirable for food quality, can be successfully inhibited by high-pressure treatment at over 405 MPa (Refs 18, 49). Because of this, the results obtained by high-pressure treatment differ remarkably depending on the particular characteristics of the treated foods being considered.

It is necessary to accumulate and consolidate the results of basic research studies to facilitate further development of this novel fish-processing technology.

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EHEDG Update

Hygienic design of closed equipment for the processing of liquid food

The European Hygienic Equipment Design Group (EHEDG) is an independent consortium formed to develop guidelines and test methods for the safe and hygienic processing of food, and includes representatives from research institutes, the food industry, equipment manufacturers and government organizations in Europe*. This is the 11th in a series of articles featuring the EHEDG to be published in *Trends in Food Science & Technology*. In a previous paper[1] the general criteria for hygienic equipment design were explained. The Design Principles subgroup of the EHEDG has subsequently produced further guidelines, summarized here, giving examples of how to apply the design criteria to equipment intended for use in closed plants.

This paper describes methods of construction and fabrication illustrating how hygienic design criteria can be met in closed process equipment. Examples are given to show how to avoid crevices, shadow zones and areas with stagnating product, and how to connect and position equipment in a process line to ensure unhampered cleaning in-place and draining. Attention is drawn to ways of preventing problems with joints, which might otherwise cause leakage or contamination of product with microorganisms or even with pieces of degraded elastomeric material.

*Readers requiring further information on the EHEDG are referred to *Trends in Food Science & Technology* (1992) Vol. 3(11), p. 277.

Joints and seals

It is strongly recommended that joints are avoided where possible. For piping, bending of the pipe is highly preferable over the use of prefabricated bends with couplings. If pipe bending is not possible, welding is the preferred method, provided that the welding is done correctly, to ensure a smooth and continuous weld[2]. Where detachable joints are necessary, they should be sealed by elastomers.

Compression of elastomers

Overcompression of elastomers may affect the hygienic characteristics of equipment in two ways.

Firstly, overcompression may lead to destruction of the elastomer, particularly if the overcompressed elastomer is heated (such as during pasteurization or sterilization). The elastomer may become brittle and fail to