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Development of lipid oxidation and flesh colour in frozen stored fillets of Norwegian spring-spawning herring (*Clupea harengus* L.). Effects of treatment with ascorbic acid

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Abstract

Frozen stored fillets from Norwegian spring spawning herring (*Clupea harengus* L.) become discoloured. Based on the hypothesis that lipid oxidation in the fillets caused discoloration, we designed an experiment where ascorbic acid was added to the water (2 g l^{-1}) in RSW-tanks on board a fishing vessel and/or by spray (20 g l^{-1}) after filleting but before freezing. Treatment with ascorbic acid on board and on the fillet line led to 8- and 17–24-fold increases, respectively, in the ascorbic acid concentration in the herring fillets. Ascorbic acid treatment on the fillet line, but not the treatment on board, protected the herring fillets against oxidation during freezing, an effect that could be detected until 9 weeks of storage at −30 °C. Between 9 and 14 weeks of storage there was a new burst of lipid oxidation where the treatments had no effect. There was a large variation in colour within each fillet and between fillets in the same group. During freezing, the amount of yellow colour increased substantially and the amount of red colour increased in the posterior end of the fillets, causing a visible change in appearance. Thereafter only minor changes occurred until week 10 but, in week 30, the fillets had become less red, causing a less fresh and more grey appearance. Compared to the within fillet and within group variations, there were only minor effects of ascorbic acid treatment on colour. It is suggested that shelf life of frozen herring fillets should be set at between 9 and 14 weeks at −30 °C. Treatment with ascorbic acid will not alleviate the discoloration of frozen herring fillets.

Keywords: Herring; Lipid oxidation; Flesh colour; Antioxidants

1. Introduction

The Norwegian spring-spawning herring (*Clupea harengus* L.) reached a spawning biomass of 9 million tonnes in 1997, after having recovered from a nearly extinct state in the late 1960s. The stock feeds in the Norwegian Sea from April to August and gathers in deep fjords in Northern Norway during the wintering period from September to January. Spawning migration commences in mid January to the spawning grounds southwards on the Norwegian coast (Slotte, 1999). During the wintering and spawning periods, the herring do not feed but rely on body energy stores accumulated in spring and summer. This leads to a cyclic change in body lipid and energy content, the lipid level being up to 30% of wet weight in May–September and sometimes below 10% in March–April (Slotte, 1999 and references therein; Hamre, Lie, & Sandnes, in press).

The herring stock is the basis of extensive fisheries on the Norwegian coast from September to March, and important products are frozen herring fillets, which are sold mainly on the Eastern European markets. Frozen herring fillets from fish caught late in the season are of inferior quality due to discoloration.

Herring fillets are rich in polyunsaturated fatty acids, which are very susceptible to peroxidation. Further, herring appears to have higher oxidation rates than other fish species (unpublished results), perhaps due to higher activity of enzymatic oxidation (Hultin, 1988). In lipid peroxidation (Frankel, 1998), free radicals abstract a hydrogen from a double bond of fatty acids to produce fatty acid radicals, which react further with oxygen to produce fatty acid peroxides. The fatty acid peroxides are free radicals, which can react with a new fatty acid,

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initiating a new cycle of auto-oxidation. The primary product of lipid oxidation is the fatty acid hydroperoxide, measured here as peroxide value (PV). The fatty acid hydroperoxide is unstable and decomposes readily to shorter chain hydrocarbons such as aldehydes. These compounds are volatile, and important for rancid odour and taste. In the present study we measure thiobarbituric acid reactive substances (TBARS), consisting mainly of malondialdehyde as a representative of aldehydes. Fish flesh is protected from lipid oxidation by nutrient and endogenous antioxidants, where vitamin E is an important lipid soluble antioxidant and vitamin C serves as antioxidant in the water phase. Vitamin E reacts with the fatty acid peroxide radical, preventing the radical from reacting with new fatty acids and thereby breaking the chain reaction of auto-oxidation. Vitamin C can regenerate the vitamin E radical and will also scavenge reactive oxygen species in the water phase. Of the endogenous antioxidants, gluthathione and ubiquinone are present in the water and lipid phases, respectively. The oxidative stability of the fillet is affected by antioxidant enzymes, neutralising reactive oxygen species, and by transition metals such as iron and copper, which have a prooxidant effects as free ions and haeme, and antioxidant effects as integral components of the antioxidant enzymes.

It is well known that lipid oxidation causes discoloration of fish fillets (Ruff, FitzGerald, Cross, Hamre, & Kerry, in press) and we hypothesised that this was the reason for the deterioration in quality of herring fillets. Ascorbic acid treatment of trout and herring fillets prior to freezing has been shown to prevent lipid oxidation (Bowernfeind, 1951). The purpose of the present experiment was to study the effect of treatment of herring fillets with ascorbic acid on the development of lipid oxidation and colour during frozen storage.

2. Materials and methods

2.1. Fish

A full scale experiment was performed in co-operation with a fish processor (Austevoll Fiskeindustri AS, Storebø, Norway) and a fishing vessel owner (PR Lafisk ANS, Storebø, Norway). In a catch of Norwegian spring spawning herring (*Clupea harengus*, L.) taken by the vessel MS Verdi on the 4 March 1996, two RSW tanks were treated with ascorbic acid (2 g l^{-1}) and two tanks served as controls. Before the ascorbic acid was added, the fish in all the tanks were flushed with seawater to remove blood. The catch was landed after 2 days at sea and filleted according to industrial procedures. Both groups of herring were divided in two, where half of each group was sprayed with ascorbic acid solution (20 g l^{-1}) after filleting and the other half was untreated. The herring were then packed in 20 kg plastic lined cardboard boxes, frozen in a tunnel to −30 °C and stored at this temperature for 30 weeks. The groups are named as follows: —: no treatment with ascorbic acid; –+: no ascorbic acid on board, ascorbic acid after filleting; +–: ascorbic acid on board but not after filleting; ++ ascorbic acid both on board and after filleting.

Samples were taken before filleting, before freezing and after 2, 6, 9, 14, 22 and 30 weeks of frozen storage at −30 °C. The samples taken before freezing consisted of 12 fish per group and were analysed as three pooled samples of four fish per sample. During frozen storage three boxes per group were sampled at each point. The herring were thawed over night and 10 double fillets per box were divided in two. One half was used for chemical analyses and the other for measurement of colour. The 10 single fillets per box taken for chemical analyses were pooled to one sample.

2.2. Chemical analyses

Dry matter was determined gravimetrically after freeze-drying. Lipid was determined gravimetrically after extraction with ethyl acetate and isopropanol (Lie, Waagbø, & Sandnes, 1988). Vitamin E was analysed as descibed by Lie, Sandvin, and Waagbø (1994), vitamin C according to Mæland, Rosenlund, Stoss, and Waagbø (1999), TBARS according to Hamre, Næss, Espe, Holm, and Lie (2001) and peroxide value according to Undeland, Härröd, and Lingert (1998).

2.3. Colorimetric measurement

Flesh colour was measured at three points in the fillet (anterior, middle, posterior), using a portable Hunterlab Miniscan/EX instrument (10* standard observer, illuminant D65, Hunter Associates Laboratory Inc, 11491 Sunset Hills Road, Reston, Virginia, USA) calibrated to a white and a black standard. The tristimulus $L^*a^*b^*$ measurement mode was used as it relates to the human eye response to colour. The L^* variable represents lightness ($L^*=0$ for black, $L^*=100$ for white), a^* scale represents the red/green, $+a^*$ intensity in red and $-a^*$ intensity in green. b* scale represents the yellow/blue, $+b^*$ intensity in yellow and $-b^*$ intensity in blue.

2.4. Statistics

The software Statistica (Statsoft Inc., Tulsa, USA) was used for statistical analyses. Two way ANOVA, for analysing effect of treatment during repeated measurements, was applied on chemical data from the storage experiment. Colour measurements were analysed using three way ANOVA on effects of treatment and two repeated measurement factors; measuring point on the fillet and time. Differences between means were ana-

lysed using the Scheffe test for post hoc comparison. Differences and effects were considered significant at $P < 0.05$.

3. Results

Dry matter content of the herring fillets was 28 ± 1% and lipid constituted 11 ± 2% of wet weight. Ascorbic acid concentration in the water of the RSW tanks on board was 1580 mg kg^{-1} just after addition and was gradually reduced to 281 mg kg^{-1} at landing of the fish (Fig. 1). There was a significant effect of treatment on ascorbic acid concentration of the herring fillets; group — < group +– < group–+ = group + + (Table 1). Fillet vitamin C concentration was significantly reduced with time and most of the reduction was associated with freezing. Fillet vitamin E concentration was higher in the two groups where vitamin C was added after filleting (groups –+ and + +) than in the group not treated with ascorbic acid (group —). The group treated with ascorbic acid on board only (group +–), had intermediate vitamin E concentration. Fillet vitamin E content was significantly and gradually reduced with storage time. Treatment with ascorbic acid had no effect on PV when the whole storage experiment was subjected to ANOVA. When testing the first 9 weeks only, the fish treated with ascorbic acid after filleting (group –+), had lower PV than all other groups. PV was not detectable before freezing and was low and similar at 2, 6 and 9 weeks of frozen storage. From 14 weeks, the PV increased and became more variable. TBARS was low and similar in the two groups treated with ascorbic acid after filleting (groups –+ and + +), significantly higher in the group treated with ascorbic acid on board only (group +–) and highest in the untreated group (—). The group differences were clear in weeks 2-9 of frozen storage. Thereafter, the picture became more complex. A mean TBARS-level of 4 μmol kg^{-1} was found in the fillets before freezing, increasing to 22–25 μmol kg^{-1} in weeks 2–9 and to 34–37 μmol kg^{-1} in weeks 14–30 of frozen storage. The three levels were all significantly different from each other.

The fillets were lighter and more yellow at the anterior end compared to the posterior end (Tables 2 and 3). Before freezing, more red colour was found at the anterior than at the posterior end of the fillets but, after freezing, the tail area became more red and the picture was reversed. L^* was not affected by treatment with ascorbic acid. There was a slight, but significant effect of treatment on b^*, where herring treated with ascorbic acid only on the fillet line (group –+), was less yellow than both groups treated with ascorbic acid on board (groups +– and + +, difference in means 0.6 units) Group –+ was intermediate. Untreated fish (group —) had a slightly lower a^* value than fish treated both on board and after filleting (group + +, difference between means 0.67 units), while the other groups were intermediate. The fillets became gradually darker from before freezing to 10 weeks of frozen storage. By week 30, the fillets had become slightly lighter again. The intensity of red colour increased gradually until week 10 and decreased thereafter to less than the level found before freezing. The intensity of yellow increased dramatically from before freezing until week 2 and stayed constant thereafter.

4. Discussion

The levels of dry matter and lipid in the fillets of 28 and 11%, respectively, were in line with previous results

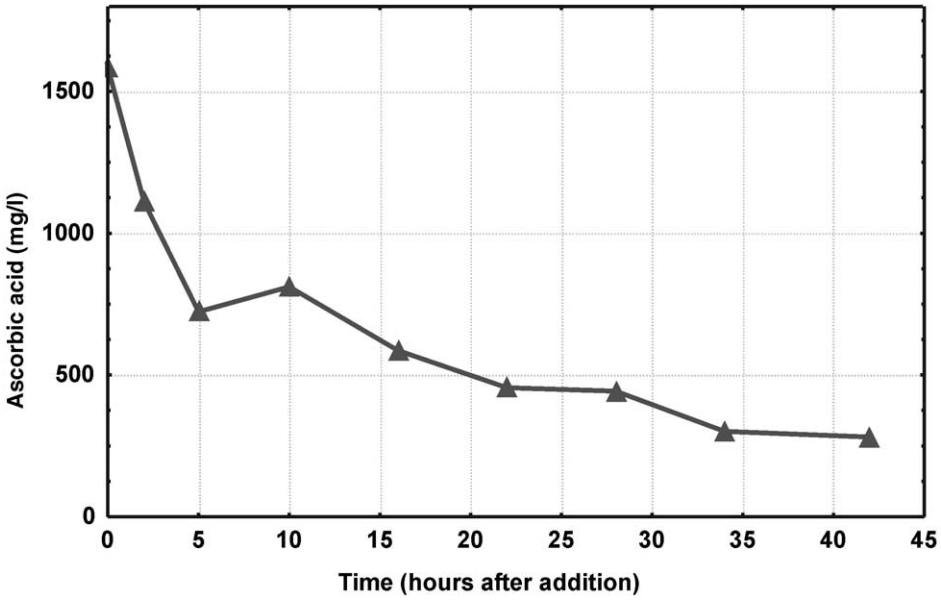

Fig. 1. Ascorbic acid concentration in RSW tank water.

Table 1
Concentration of vitamin E, vitamin C, peroxide value (PV) and thiobarbituric acid reactive substances (TBARS) in herring fillet treated with ascorbic acid and stored at −30 °C

Weeks	0 (Before freezing)	2	6	9	14	22	30	ANOVA *P*
Vitamin E (mg kg^{-1} wet wt.)								
—	35±5	30±1	27±2	25±2	25±3	25±1	22±3	Treatment: 0.003
−+	28±4	30±6	32±4	28±1	31±3	25±3	25±2	Time: $<10^{-5}$
+−	33±3	33±7	24±1	31±3	30±5	28±4	28±2	Treatment×time: NS
++	41±1	32±4	30±3	32±8	32±2	32±2	28±4	
Vitamin C (mg kg^{-1} wet wt.)								
—	4.2±0.8	1.7±0.1	2.1±0.2	1.2±0.1	1.8±1.0	2.3±0.2	2.5±0.3	Treatment: 10^{-6}
−+	96±35	43±6	70±10	27±6	51±19	44±29	59±14	Time: $<10^{-6}$
+−	32±1	15±3	13±2	11±2	11±1	11±8	7.6±1.1	Treatment×time: $<10^{-5}$
++	70±11	55±6	50±6	38±7	37±1	85±26	39±9	
PV (mmol kg^{-1} lipid)								
—	n.d.	1.2±0.1	1.2±0.1	1.8±0.4	5.8±0.4	4.2±0.1	1.7±0.4	Treatment: NS
−+	n.d.	0.4±0.3	0.4±0.1	0.8±0.3	7.0±5.1	3.7±1.4	1.6±0.6	Time: $<10^{-6}$
+−	n.d.	1.0±0.2	1.2±0.1	1.4±0.3	5.4±2.3	3.6±0.9	4.9±1.9	Treatment×time: NS
++	n.d.	0.5±0.1	0.8±0.3	2.1±0.8	3.5±0.8	3.7±0.2	2.2±0.5	
TBARS (μmol kg^{-1} wet wt.)								
—	2.7±0.8	41±8	27±3	36±3	39±5	47±5	36±6	Treatment: 10^{-4}
−+	2.3±2.5	15±2	11±4	18±4	39±5	36±10	29±5	Time: $<10^{-6}$
+−	6.9±1.7	29±5	33±9	26±2	27±5	28±2	34±8	Treatment×time: 10^{-6}
++	2.9±0.6	13±1	19±3	20±5	42±6	24±3	38±4	

Codes: —: no treatment with ascorbic acid; −+: no ascorbic acid on board, ascorbic acid after filleting; +−: ascorbic acid on board but not after filleting; ++ ascorbic acid both on board and after filleting. Mean±SD; $n=3$ pooled samples of 4 fish at 0 weeks, otherwise 10 fish sample-1; n.d., not detected.

Table 2
Colour measurements of herring fillets using Hunterlab miniscanner in tristimulus *L*a*b** measurement mode

Week	0			2			10			30		
	Anterior	Middle	Posterior	Anterior	Middle	Posterior	Anterior	Middle	Posterior	Anterior	Middle	Posterior
*L**												
—	57±4	59±3	51±3	58±4	54±10	46±3	56±3	56±4	45±4	59±4	57±4	50±4
−+	57±4	59±5	51±3	59±5	57±5	46±4	55±4	54±3	42±2	58±5	58±6	49±5
+−	59±3	61±5	54±4	59±3	57±4	44±3	57±4	55±5	42±3	58±4	55±4	48±4
++	57±2	58±5	52±2	57±4	56±4	44±2	59±4	55±4	41±3	60±3	59±4	48±4
*a**												
—	4.9±1.8	4.3±1.4	3.2±1.1	4.0±1.3	4.1±1.1	4.8±1.1	4.0±1.3	3.9±1.2	5.2±2.0	2.9±1.2	3.9±1.5	4.1±1.4
−+	5.6±1.2	3.9±1.7	3.0±1.5	4.4±1.3	4.2±1.2	5.2±1.1	4.5±1.4	5.0±1.3	7.2±1.7	3.3±1.0	3.2±1.2	3.5±1.5
+−	4.6±1.6	4.2±1.7	2.8±1.3	3.9±1.1	4.4±1.4	6.2±1.2	4.2±1.7	4.4±1.4	6.5±1.6	2.8±1.4	4.0±1.6	4.7±1.4
++	5.4±1.7	5.0±3.5	3.3±1.3	4.8±1.3	4.7±1.6	5.3±0.9	4.5±1.6	4.7±1.6	7.6±2.2	3.6±1.2	3.8±1.4	4.6±1.3
*b**												
—	15.1±1.1	14.7±1.4	11.7±1.1	19.1±1.2	18.4±1.3	14.7±1.5	21.0±1.9	20.2±1.5	16.1±1.4	17.7±1.3	18.5±1.2	15.2±1.6
−+	14.6±1.3	13.5±1.6	10.9±1.3	19.1±1.4	18.3±1.4	15.1±2.1	19.6±1.6	19.4±1.5	16.1±1.6	17.9±1.7	18.3±1.7	15.0±2.1
+−	14.7±1.2	14.3±1.2	11.7±1.2	20.2±1.3	19.8±1.8	15.0±1.5	21.0±1.7	20.2±1.7	15.7±1.8	17.6±1.3	18.6±1.3	16.8±5.6
++	14.6±1.2	14.1±0.9	11.7±1.4	20.0±1.0	19.0±1.4	14.8±1.6	21.4±1.2	20.3±1.2	15.9±1.9	19.6±1.6	19.5±1.2	14.7±1.3

Codes: —: no treatment with ascorbic acid; −+: no ascorbic acid on board, ascorbic acid after filleting; +−: ascorbic acid on board but not after filleting; ++ ascorbic acid both on board and after filleting. Mean±SD. Week 0 (before freezing): $n=12$. During frozen storage: $n=30$.

from herring caught during spawning migration in February/March (Slotte, 1999 and references therein, Hamre et al., in press). Vitamin C concentration in frozen herring fillets was reduced during wintering and spawning migration from about 3.0 to 1.0 mg kg^{-1} (Hamre et al., submitted for publication). The results of the present study show that vitamin C concentration is reduced during freezing, and that the concentration in fresh fillets is higher than indications in this previous study. The vitamin E concentration in fresh untreated

Table 3
Results of ANOVA tests on colour measurements in herring fillets

	L^*		a^*		b^*	
	F	P	F	P	F	P
Group	1	NS	7	0.0002	10	7×10^{-6}
Time	52	$<10^{-6}$	39	$<10^{-6}$	428	$<10^{-6}$
Measuring point on fillet	1941	$<10^{-6}$	40	$<10^{-6}$	999	$<10^{-6}$
Group×Time	3	0.004	2	0.02	2	NS
Group×Point	4	0.0005	4	0.0006	4	0.001
Time×Point	56	$<10^{-6}$	73	$<10^{-6}$	21	$<10^{-6}$
Group×Time×Point	3	2×10^{-6}	3	0.0001	5	$<10^{-6}$

fillets was also considerably higher than in Hamre et al. (in press), who found values between 8 and 28 mg kg^{-1}, not dependent on season.

Ascorbic acid in the RSW tanks on board the fishing vessel was broken down with time to about 20% of the initial level. Nevertheless ascorbic acid concentration in the herring fillets was increased from 4 to 32 mg kg^{-1} by this treatment. The ascorbic acid must have penetrated the skin, since analyses were performed on skinned fillets, but it is uncertain how far into the fillet the ascorbic acid had diffused. The ascorbic acid spray on the fillet line was probably mainly a surface treatment, since the fillets were frozen shortly afterwards. Still, the ascorbic acid treatment would protect against lipid oxidation, since this is mainly a surface process, limited by the diffusion of oxygen (Undeland, Stading, & Lingert, 1998). The ascorbic acid analyses of these fillets show large variations, the increase in mean ascorbic acid concentration was 17–24-fold that of the untreated fillets. The reduction in ascorbic acid concentration from before freezing to week 2 of frozen storage (of about 50%) is probably mainly due to a reduction during freezing. Further storage led to a minor reduction in the group treated with ascorbic acid on board only, while the other groups seemed to have unchanged vitamin C concentrations.

The overall statistical analyses showed that the untreated herring fillets had less vitamin E than the fillets treated with ascorbic acid after filleting. This may be due to the regeneration of vitamin E by vitamin C as shown by Packer, Slater, and Wilson (1979). The effect was most evident from week 9 of frozen storage. In contrast to vitamin C, vitamin E concentration showed a gradual reduction throughout the storage period, especially in the group not treated with ascorbic acid. However, the vitamin E concentration stayed quite high until the end of the storage period. Fillet vitamin E levels have been measured as 20 mg kg^{-1} in Atlantic salmon fed 300 mg kg^{-1} α- tocopheryl acetate (Hamre & Lie, 1995), 9 mg kg^{-1} in turbot fed 500 mg kg^{-1} α-tocopheryl acetate (Ruff, FitzGerald, Cross, Hamre et al., in press) and 11 mg kg^{-1} in Atlantic halibut fed 200 mg kg^{-1} α-tocopheryl acetate (Ruff, FitzGerald, Cross, & Kerry, 2002).

With respect to lipid oxidation, the experiment can be divided into three periods: freezing, storage for 2–9 weeks and storage from 14 to 30 weeks. Before freezing, PV was not detectable and TBARS was 2–7 μmol kg^{-1}, which is almost as low as what is found in fresh fish fillets from other species (Hamre, Berge, & Lie, 1998; Ruff, FitzGerald, Cross, Hamre et al., in press; Ruff et al., 2002). From before freezing until 2 weeks of storage there was an abrupt increase in both PV and TBARS, which was most probably connected with the freezing process. Ascorbic acid, added after filleting, clearly protected the fillets against lipid oxidation during freezing and the mechanism behind the protection was probably scavenging of reactive oxygen species, since there was no simultaneous sparing of vitamin E. Treatment with ascorbic acid on board only, did not protect against lipid oxidation during freezing. In the period from 2 to 9 weeks of frozen storage, the levels of lipid oxidation products in the herring fillets stayed quite stable. After 9 weeks, both PV and TBARS increased, the variability became larger and there was no effect, any more, of the treatment with ascorbic acid. What initiated this second burst of lipid oxidation is obscure, but we experienced a similar result in another storage experiment with herring (unpublished results).

Atlantic salmon fillets stored at −30 °C for 48 weeks developed a maximum TBARS of 25 nmol g^{-1} (Hamre et al., 1998) and, until after week 24, TBARS did not exceed 10 nmol g^{-1}. Norwegian spring spawning herring, caught between September and March and stored at −30 °C for 3–67 days, had TBARS values between 43 and 122 nmol g^{-1} (Hamre et al., in press). Atlantic halibut and turbot stored on ice for 9 days had maximum TBARS values of 8.5 and 36 nmol g^{-1}, respectively (Ruffet et al., 2002), while Atlantic salmon stored on ice for 15 days had a maximum TBARS levels of 25 nmol g^{-1} (Hamre et al., in press). Lipid oxidation during storage is dependent on the vitamin E concentration in the fillet (Frigg, Prabucki, & Ruhdel, 1990; Hamre et al., 1998; Ruff, FiztGerald, Cross, Hamre et al., in

press), but vitamin E in herring fillets in the present study was higher than vitamin E levels reported in the studies with Atlantic salmon, Atlantic halibut and turbot (Hamre et al., 1998; Ruff, FitzGerald, Cross, Hamre et al., in press; Ruff et al., 2002). It seems that the susceptibility to lipid oxidation is species dependent, herring being more susceptible than Atlantic salmon and turbot more than Atlantic halibut and Atlantic salmon. Hultin (1988) found that enzymatic lipid oxidation of microsomes from light muscle of herring was 4–5 times higher than that from light muscle of winter flounder or red hake. The enzymatic lipid oxidation in dark muscle of herring was 3–4 times that in light muscle (Hultin, 1988). Differences in enzymatic lipid oxidation may explain the apparent species differences in susceptibility to lipid oxidation.

Even though the TBARS values were high in the present study, PV did not exceed the maximum recommended value for human consumption, which is 10 mmol kg^{-1} lipid. This is in accordance with routine PV values in frozen herring fillets (Hamre et al., in press). Undeland, Ekstrand, and Lingert (1998) stored herring fillets at −18 °C for 18 weeks and found a gradual increase in PV to 40 and 110 mmol kg^{-1} in light and dark muscle, respectively. Thus, temperature seems to be of great importance for preventing lipid oxidation in frozen stored herring fillets.

The largest variation in colour was found within the fillets, where the anterior end was dramatically lighter and more yellow than the posterior end. In frozen fillets, the anterior end was also less red than the posterior end, probably relating to the red muscle, which was visible in the tail area in frozen fish. There was also a large variation in colour between fillets from the same group. The data on colour were in accordance with results from Hamre et al. (in press) from fish caught late in the season, which showed that frozen stored fillets from herring caught in February/March were darker, more red and more yellow than fillets from herring caught in September. This was linked to changes in lipid and dry matter content of the fillets. Red colour was higher in the anterior part than in the posterior part of the fillet before freezing, but this relation changed with freezing. Moreover, yellow colour increased substantially with freezing; thus the appearance of the fillet changed dramatically during freezing. Yellow colour is often associated with lipid oxidation (Ruff, FitzGerald, Cross, Hamre et al., in press), supported by the increase in TBARS and PV found simultaneously with the colour changes during freezing in the present study. With prolonged frozen storage the fillets appeared less fresh and more grey, possibly related to the reduction in red colour seen in week 30. This may be caused by oxidation of haemoglobin to methaemoglobin, resulting in a change from red to brown colour. Compared to the large variation in colour within each fillet and between fillets of the same group, treatment with ascorbic acid had very little effect on colour, and the measured differences could not be observed with the eye. Ascorbic acid, added on the fillet line led to higher levels of red colour in weeks 2 and 10, possibly preventing oxidation of haemoglobin to methaemoglobin. Also there was a minor reduction in yellow colour in week 2 as a result of ascorbic acid treatment, which may be related to decreased lipid oxidation.

5. Conclusion

Freezing of herring fillets causes large changes in colour simultaneously with an increase in lipid oxidation. Ascorbic acid treatment had little effect on the colour changes, but ascorbic acid spray after filleting protected the fillets against lipid oxidation, although this effect only lasted for 9 weeks of frozen storage. After 9 weeks of storage there was an increase in lipid oxidation products, concomitant with further colour deterioration, especially related to loss of red colour resulting in less fresh and more grey appearance. The problems with discoloration can be met by setting the shelf life of frozen herring at between 9 and 14 weeks but not with ascorbic acid treatment. One should also consider excluding herring, caught late in the season, from the market for frozen fillets.

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