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Effect of dietary α-tocopherol supplementation and gamma-irradiation on α-tocopherol retention and lipid oxidation in cooked minced chicken

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The effects of dietary α-tocopherol supplementation and gamma-irradiation on α-tocopherol retention and lipid oxidation in cooked minced chicken during refrigerated storage were studied. Minced breast and thigh meat from broilers fed diets supplemented with 100, 200 or 400 mg α-tocopheryl acetate/kg feed was irradiated at 2.5 or 4.0 kGy. Cooked irradiated and unirradiated meat was stored at 4°C for 5 days. α-Tocopherol concentrations increased with increasing dietary supplementation. Concentrations decreased during storage, but retention was not affected by irradiation. Lipid stability was determined by measuring the formation of thiobarbituric acid-reacting substances (TBARS) and cholesterol oxidation products (COPs) during storage. TBARS and COPs increased during storage and were reduced by increasing levels of dietary α-tocopheryl acetate supplementation. Irradiation accelerated TBARS formation during storage, but this was prevented by supplementation with 200 mg α-tocopheryl acetate/kg feed. Irradiation tended to increase COPs during storage, although no consistent effects were observed. In general supplementation with over 400 mg α-tocopheryl acetate/kg feed may be required to control cholesterol oxidation in minced chicken. The results suggest that, overall, irradiation had little effect on lipid stability in α-tocopherol-supplemented meat following cooking and storage.

INTRODUCTION

Irradiation extends the shelf life of foods and improves safety via the reduction of spoilage and pathogenic microorganisms (Monk *et al.*, 1995). Exposure of foods to radiation initially involves the ionisation of water, followed by the generation of hydroxyl radicals and the superoxide anion (Glidewell *et al.*, 1993), and may result in the oxidation of proteins, lipids, carbohydrates and vitamins (Simic, 1983; Glidewell *et al.*, 1993; Kilcast, 1994). Irradiation does not adversely affect the overall nutritive value of foods (Stevenson, 1994), and the oxidative changes induced by irradiation are similar to those observed using conventional food processing techniques. However, a combination of irradiation and other processing conditions, such as storage and cooking, may result in accelerated oxidative deterioration in foods. Storage of irradiated fats in the presence of oxygen does accelerate the autoxidative process (Diehl, 1983), and irradiation has been shown to accelerate the oxidation of unsaturated fatty acids and cholesterol during the refrigerated storage of meats (Heath *et al.*, 1990; Hwang and Maerker, 1993).

Vitamin E is the most radiation-sensitive of the fat-soluble vitamins, and irradiation has been shown to decrease vitamin E (α-tocopherol) in meats (Lakritz *et al.*, 1995). From a nutritional perspective, the destruction of α-tocopherol in irradiated meats is not a problem, as meats are a poor source of the vitamin. However, α-tocopherol is a highly effective antioxidant in meats. Dietary supplementation with α-tocopheryl acetate results in α-tocopherol becoming an integral component of biological membranes, where it acts as a highly effective chain-breaking antioxidant and inhibits phospholipid and cholesterol oxidation (Buckley *et al.*, 1995). In addition, it inhibits loss of water-holding capacity, colour and flavour during storage. Irradiation may have a negative impact on α-tocopherol and may reduce the antioxidant capacity of muscle and promote oxidation. Little is known about the effects of irradiation on α-tocopherol and lipid stability in meats from

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α-tocopherol-supplemented animals, particularly after cooking. It was, therefore, decided to examine the effect of cooking and storage on lipid oxidation in irradiated meat from chickens fed α-tocopheryl acetate supplements.

MATERIALS AND METHODS

Reagents

The α-tocopheryl acetate used in the diets was obtained from Roche Products Ltd, Welwyn Garden City, Hertfordshire, UK. Cholesterol oxide standards were purchased from Sigma Chemical Co. Ltd, Poole, Dorset, UK and Steraloids (UK) Ltd, New Barnet, Hertfordshire, UK. All other chemicals were of 'AnalaR' grade, obtained from British Drug House, Poole, Dorset, UK and Rathburn Chemicals Ltd, Walkerburn, Scotland.

Animals and diets

Cobb 500 broiler chicks were purchased at 1-day-old from a commercial hatchery. Chicks were randomly divided into three groups and were fed diets supplemented with 100 (E-100), 200 (E-200) or 400 (E-400) mg α-tocopheryl acetate/kg feed. Diet composition is outlined in Table 1. Chicks were housed in raised wire cages (6 chicks per cage) in a controlled environment room, employing a 12 h normal light/dim cycle. Feed and water were provided *ad libitum*. After 8 weeks, birds were slaughtered by conventional methods. Carcasses were chilled for 24 h. Breast and thigh meat was removed, vacuum-packed and stored at −20°C.

Preparation of irradiated meat

Minced breast and thigh meat was irradiated to a dose of 2.5 or 4.0 kGy, at a temperature of 4°C, in a gamma source using ^{60}Co (Gamma Beam 650 irradiator, Nordion International Inc., Canada). Irradiation was carried out at the Food Science Division, Department of Agriculture, Belfast, Northern Ireland. Irradiated samples and unirradiated controls were stored at −20°C for approximately 2 months prior to the storage stability study.

Table 1. Composition of broiler diets (g/kg)

Maize	505.6
Soya bean meal	395.8
Sunflower oil	60.0
Dicalcium phosphate	20.0
Calcium carbonate	10.0
Sodium chloride	4.0
DL-Methionine	1.6
Mineral mix[a]	1.0
Vitamin mix[b]	2.0

[a]Supplying per kg feed: $MnSO_4 \cdot 4H_2O$ (333.3 mg); $ZnSO_4 \cdot 7H_2O$ (220.3 mg); $C_6H_3O_7Fe \cdot 3H_2O$ (450 mg); $CuSO_4 \cdot 5H_2O$ (35 mg); KIO_3 (2 mg); $CoSO_4 \cdot 5H_2O$ (1 mg); Na_2SeO_3 (0.35 mg).
[b]Supplying per kg feed: Retinol (900 μg); cholecalciferol (15 μg); menadione sodium bisulphite (2 mg); riboflavin (5 mg); calcium pantothenate (15 mg); niacin (30 mg); cyanocobalamine (0.015 mg); folic acid (0.6 mg); pyridoxine (3.5 mg); thiamine (2 mg); biotin (0.2 mg); choline chloride (1.2 g).

Storage study

Unirradiated and irradiated minced meat was cooked in a conventional oven at 160°C for 45 min. On cooling, samples were wrapped in an oxygen permeable PVC film (6000–8000 ml cm^{-3} m^{-2} 24 h^{-1}), and stored, under fluorescent light, at 4°C. Samples were assessed for α-tocopherol content and lipid oxidation (TBARS and COPs formation) immediately after cooking and after 5 days of storage.

Determination of α-tocopherol

α-Tocopherol was extracted from cooked minced meat and determined by a reverse-phase HPLC method as described by Sheehy *et al.* (1994).

Determination of thiobarbituric acid reacting substances (TBARS)

The extent of fatty acid oxidation was determined by measuring TBARS (Ke *et al.*, 1977). Results were expressed as mg malonaldehyde/kg meat.

Determination of cholesterol oxidation products (COPs)

Total lipids were extracted from samples by the method of Marmer and Maxwell (1981). Lipid extracts were evaporated to dryness by rotary evaporation and reconstituted in hexane:ethyl acetate (9:1). 6-Ketocholesterol (50 μg) was added as an internal standard. COPs were separated from cholesterol and other lipids by the sample clean-up procedure of Park and Addis (1987). Elution was allowed to proceed by gravity-flow. The COPs extract was evaporated to dryness and reconstituted in ethyl acetate (4 ml). An aliquot (1 ml) was evaporated to dryness and 100 μl of bis(trimethylsilyl)trifluoroacetamide (BSTFA) were added. The sample was derivatised at room temperature, in the dark, for 30 min. The derivatised sample was evaporated under nitrogen and reconstituted in ethyl acetate (100 μl). GC analysis was carried out using a Shimadzu Model GC-14A gas chromatograph with flame ionisation detection. A Shimadzu Model C-R6A Chromatopac integrator was used to quantify cholesterol oxides. The column used was a methylsilicone column (15 m, 0.32 mm i.d., film thickness 0.25 μm, S.A.C. Chromatography Ltd, Letchworth, Hertfordshire, UK). The carrier gas was helium at a pressure of 0.7 kg/cm^2. Oven temperature programming was as follows: 190–220°C at 10°C/min; 220–234°C at 0.4°C/min; 234–255°C at

1.5°C/min and hold at 255°C for 20 min; injector temperature, 300°C; detector temperature, 300°C.

Statistical analysis

All data were subjected to analysis of variance (ANOVA). The statistical significance of the differences between means was determined by the method of least significant difference (Snedecor and Cochran, 1967).

RESULTS

Effect of irradiation and storage on α-tocopherol

α-Tocopherol concentrations in minced, irradiated and cooked breast and thigh meat before and after storage at 4°C are shown in Table 2. In breast and thigh, α-tocopherol concentrations increased significantly ($p < 0.001$) as supplementation increased from E-100 to E-200 and E-400. Irradiation at 2.5 or 4.0 kGy, followed by cooking, did not significantly reduce the concentration of α-tocopherol compared to the control samples. α-Tocopherol concentrations decreased significantly ($p < 0.01$) after storage, in breast and thigh. However, irradiation did not reduce the α-tocopherol concentration in stored meat compared to control samples.

Effect of irradiation on TBARS

TBARS in irradiated and cooked breast and thigh meat during refrigerated storage are shown in Table 3. Immediately after cooking, TBARS were significantly ($p < 0.05$) reduced as dietary α-tocopheryl acetate supplementation increased in breast, but not in thigh. TBARS in all irradiated breast and thigh samples were similar to their respective controls. TBARS increased in all treatments during storage, but were significantly reduced ($p < 0.025$) as supplementation increased from E-100 to E-200 and E-400. After storage, TBARS in irradiated E-100 breast samples were significantly ($p < 0.001$) higher than the unirradiated control, at both dose levels. At the E-200 and E-400 supplementation levels, the irradiated samples did not differ significantly from the controls. In thigh, TBARS in samples irradiated at 4.0 kGy were significantly ($p < 0.005$) higher than control values in meat from the E-100 group. As in the case of breast meat, irradiation did not increase TBARS in samples from the E-200 and E-400 groups.

Effect of irradiation on COPs

COPs were detected in some groups only, immediately after cooking (results not shown). After storage for 5 days, 7-ketocholesterol (7-Keto), 7β-hydroxycholesterol (7β-OH), 20α-hydroxycholesterol (20α-OH) and 25-hydroxycholesterol (25-OH) were detected in unirradiated and irradiated breast meat from the E-100 and E-200 groups (Table 4). In the E-400 group, 7-Keto, 7β-OH and 25-OH were detected in samples irradiated at 4.0 kGy, and only 25-OH was detected in the samples irradiated at 2.5 kGy. In general, significant ($p < 0.05$) decreases in COPs were observed as dietary α-tocopheryl acetate supplementation increased. This effect was greatest as supplementation increased from E-200 to E-400. In thigh, 7-Keto, cholestan-3β,5α,6β-triol (Triol) and 25-OH were detected in unirradiated and irradiated meat from all dietary treatments (Table 5), and values were consistently higher than those observed in breast meat. All COPs decreased significantly ($p < 0.001$) as dietary α-tocopheryl acetate supplementation increased. In breast and thigh, irradiation increased some COPs relative to control values, but the effect was not consistent. The data for total COPs indicate that, in breast, irradiation significantly increased COPs formation in E-200 and E-400 compared to controls. In thigh, a significant effect was observed in E-200 only.

Table 2. α-Tocopherol content of irradiated minced chicken meat immediately after cooking and after storage at 4°C for 5 days

Treatment[1]	α-Tocopherol (μg/g muscle)			
	Breast		Thigh	
	Day 0	Day 5	Day 0	Day 5
100 Control	17.5 ± 0.40^{a}	14.7 ± 0.21^{ad}	40.2 ± 0.18^{a}	20.4 ± 0.91^{ad}
2.5	16.4 ± 0.64^{a}	14.2 ± 0.09^{ad}	40.2 ± 0.49^{a}	18.6 ± 0.16^{ad}
4.0	16.6 ± 1.03^{a}	14.7 ± 0.21^{ad}	42.1 ± 0.24^{a}	19.7 ± 1.38^{ad}
200 Control	25.1 ± 0.58^{b}	22.6 ± 0.47^{bd}	50.3 ± 1.81^{b}	28.8 ± 1.13^{bd}
2.5	24.8 ± 0.14^{b}	23.7 ± 0.28^{b}	51.7 ± 0.43^{b}	27.0 ± 1.03^{bd}
4.0	24.6 ± 0.82^{b}	23.5 ± 0.33^{b}	50.6 ± 0.88^{b}	27.7 ± 0.11^{bd}
400 Control	35.3 ± 1.76^{c}	30.7 ± 1.26^{cd}	65.7 ± 2.26^{c}	59.0 ± 3.25^{c}
2.5	36.1 ± 0.58^{c}	30.7 ± 0.77^{cd}	63.4 ± 2.34^{c}	59.5 ± 0.80^{c}
4.0	33.7 ± 1.44^{c}	30.4 ± 0.27^{c}	65.3 ± 0.85^{c}	59.1 ± 1.69^{c}

Values are means ± SE of four duplicate determinations.
[1]Feeds were supplemented with 100, 200 or 400 mg α-tocopheryl acetate/kg. Muscle was irradiated to doses of 0 (Control), 2.5 or 4.0 kGy.
[a–c]Means in the same column not sharing a common superscript letter are significantly different to at least $p < 0.001$.
[d]Significantly different from corresponding Day 0 group to at least $p < 0.01$.

Table 3. TBARS in irradiated minced chicken meat immediately after cooking and after storage at 4°C for 5 days

Treatment[1]	TBARS (mg malonaldehyde/kg meat)			
	Breast		Thigh	
	Day 0	Day 5	Day 0	Day 5
100 Control	0.33 ± 0.02^{a}	2.20 ± 0.05^{a}	0.41 ± 0.01^{ab}	6.50 ± 0.07^{a}
2.5	0.35 ± 0.01^{a}	2.62 ± 0.04^{b}	0.39 ± 0.01^{b}	6.52 ± 0.11^{a}
4.0	0.33 ± 0.01^{a}	2.70 ± 0.06^{b}	0.40 ± 0.03^{ab}	6.75 ± 0.03^{b}
200 Control	0.33 ± 0.01^{a}	1.31 ± 0.03^{c}	0.44 ± 0.01^{a}	6.28 ± 0.01^{c}
2.5	0.34 ± 0.03^{a}	1.28 ± 0.01^{c}	0.40 ± 0.05^{ab}	6.29 ± 0.01^{c}
4.0	0.31 ± 0.01^{a}	1.32 ± 0.03^{c}	0.43 ± 0.02^{ab}	6.27 ± 0.02^{c}
400 Control	0.27 ± 0.01^{b}	0.80 ± 0.02^{d}	0.37 ± 0.01^{b}	5.79 ± 0.05^{d}
2.5	0.25 ± 0.01^{b}	0.85 ± 0.04^{d}	0.39 ± 0.01^{b}	5.66 ± 0.03^{d}
4.0	0.26 ± 0.02^{b}	0.77 ± 0.04^{d}	0.39 ± 0.01^{b}	5.44 ± 0.08^{e}

Values are means ± SE of four duplicate determinations.
[1]See Table 2 for description of treatments.
[a–e]Means in the same column not sharing a common superscript are significantly different to at least $p < 0.005$.

Table 4. Cholesterol oxidation products (COPs) content of irradiated, minced and cooked chicken breast meat following storage at 4°C for 5 days

Treatment[1]	COPs (μg/g muscle)				
	7-Keto	7β-OH	20α-OH	25-OH	Total
100 Control	0.90 ± 0.07^{a}	0.57 ± 0.06^{ab}	0.23 ± 0.02^{a}	0.58 ± 0.05^{a}	2.26 ± 0.09^{a}
2.5	0.98 ± 0.10^{a}	0.47 ± 0.07^{ac}	0.48 ± 0.07^{bc}	0.58 ± 0.04^{a}	2.51 ± 0.12^{a}
4	1.09 ± 0.10^{a}	0.68 ± 0.06^{bd}	0.34 ± 0.05^{ab}	0.51 ± 0.05^{a}	2.62 ± 0.22^{a}
200 Control	1.11 ± 0.12^{a}	0.38 ± 0.07^{c}	0.45 ± 0.08^{bc}	0.34 ± 0.08^{b}	2.21 ± 0.25^{a}
2.5	1.89 ± 0.12^{b}	0.58 ± 0.07^{a}	0.58 ± 0.05^{c}	0.43 ± 0.08^{ab}	3.48 ± 0.25^{b}
4	1.70 ± 0.19^{b}	0.50 ± 0.05^{cd}	0.55 ± 0.05^{c}	0.37 ± 0.07^{b}	3.15 ± 0.17^{b}
400 Control	ND	ND	ND	ND	ND
2.5	ND	ND	ND	0.17 ± 0.03^{c}	0.17 ± 0.03^{c}
4	0.09 ± 0.02^{c}	0.14 ± 0.09^{e}	ND	0.63 ± 0.05^{a}	0.86 ± 0.17^{d}

Values are means ± SE of four duplicate determinations.
[1]See Table 2 for description of treatments.
[a–e]Means in the same column not sharing a common superscript are significantly different to at least $p < 0.05$.
ND, not detected.
Abbreviations: 7-Keto, 7-Ketocholesterol; 25-OH, 25-Hydroxycholesterol; 20α-OH, 20α-Hydroxycholesterol; 7β-OH, 7β-Hydroxycholesterol.

Table 5. Cholesterol oxidation products (COPs) content of irradiated, minced and cooked chicken thigh meat following storage at 4°C for 5 days

Treatment[1]	COPs (μg/g muscle)			
	7-Keto	25-OH	Triol	Total
100 Control	2.05 ± 0.06^{a}	1.70 ± 0.12^{a}	1.61 ± 0.08^{a}	5.36 ± 0.10^{ab}
2.5	2.19 ± 0.12^{ab}	1.70 ± 0.03^{a}	1.37 ± 0.08^{b}	5.26 ± 0.16^{a}
4.0	2.44 ± 0.10^{b}	1.79 ± 0.06^{a}	1.56 ± 0.21^{a}	5.79 ± 0.14^{b}
200 Control	0.76 ± 0.04^{c}	1.16 ± 0.03^{b}	0.81 ± 0.03^{cd}	2.73 ± 0.02^{c}
2.5	2.15 ± 0.30^{ab}	1.58 ± 0.13^{a}	1.00 ± 0.11^{c}	4.79 ± 0.28^{d}
4.0	2.11 ± 0.05^{ab}	1.60 ± 0.06^{a}	0.94 ± 0.04^{ce}	4.65 ± 0.13^{d}
400 Control	1.31 ± 0.09^{d}	0.73 ± 0.11^{c}	0.73 ± 0.10^{de}	2.77 ± 0.15^{c}
2.5	1.17 ± 0.04^{d}	0.70 ± 0.10^{c}	0.62 ± 0.03^{d}	2.49 ± 0.11^{c}
4.0	1.46 ± 0.09^{d}	0.79 ± 0.08^{c}	0.70 ± 0.05^{de}	2.95 ± 0.18^{c}

Values are mean ± SE of four duplicate determinations.
[1]See Table 2 for description of treatments.
[a–e]Means in the same column not sharing a common superscript are significantly different to at least $p < 0.05$.
ND, not detected.
Abbreviations: 7-Keto, 7-Ketocholesterol; Triol, Cholestan-3β,5α,6β-triol; 25-OH, 25-Hydroxycholesterol.

DISCUSSION

The results of the present experiment showed that, at supplementary dietary α-tocopherol intakes, α-tocopherol concentrations in meat were not affected by irradiation after subsequent cooking and storage. These results also indicate that α-tocopherol was not reduced by irradiation during the 2-month period of frozen storage prior to cooking and refrigerated storage. Significant losses of α-tocopherol have been reported in irradiated fish (Al-Kahtani *et al.*, 1996). In addition, irradiation has been shown to reduce α-tocopherol levels in beef, pork, lamb and turkey (Lakritz *et al.*, 1995). However, Lakritz and Thayer (1994) reported only minimal losses of α-tocopherol in chicken irradiated at a dose of 3 kGy.

TBARS increased after cooking and storage, but were decreased by dietary α-tocopheryl acetate supplementation. Irradiation did not affect TBARS immediately after cooking, but did significantly increase TBARS after storage, in breast and thigh meat, at the E-100 supplementation level. Other researchers have also found that irradiation increases lipid oxidation in stored meats. Heath *et al.* (1990) reported that a dose equivalent to 3 kGy increased TBARS in cooked chicken thigh following storage at 4°C for 4 days. Lefebvre *et al.* (1994) observed higher peroxide values in irradiated ground beef than in unirradiated meat, particularly after refrigerated storage. In the present study, irradiation did not significantly increase TBARS in cooked meat from the E-200 and E-400 groups, indicating that the higher α-tocopherol concentrations in these groups protected meat from the oxidising effects of irradiation. Previously, Patterson and Stevenson (1995) showed that dietary supplementation with α-tocopherol and ascorbic acid (800 mg/kg feed) reduced the levels of volatile lipid oxidation products formed in irradiated chicken breast and thigh meat. The results of the present study suggest that supplementation with levels of at least 200 mg α-tocopheryl acetate/kg feed are required to stabilise irradiated breast and thigh meat following cooking and during storage. This agrees with earlier observations that the optimum dietary level of α-tocopherol required to adequately stabilise meat exposed to prooxidising conditions, such as mincing and cooking, lies between 100 and 200 mg α-tocopheryl acetate/kg feed (Sheehy *et al.*, 1995).

COPs were detected in cooked, refrigerated meat, but dietary α-tocopheryl acetate supplementation inhibited their formation. Previous studies have also shown that dietary α-tocopherol significantly reduced the levels of COPs formed in cooked pork and veal during refrigerated storage (Monahan *et al.*, 1992; Engeseth *et al.*, 1993). In breast, no COPs were detected in meat from control groups supplemented with E-400 (Table 4). In thigh, COPs were detected at this supplementation level (Table 5). In general, COPs in all groups were higher in thigh than in breast meat. A previous experiment also showed that higher levels of COPs accumulate in thigh meat during storage (unpublished results). Thigh meat is more susceptible to lipid oxidation than breast meat. This is probably due to a higher phospholipid and polyunsaturated fatty acid content and a higher iron content. Cholesterol may also be expected to be more susceptible to oxidative attack in thigh due to the presence of higher levels of free radical species.

Hwang and Maerker (1993) found that irradiation increased COPs in beef, pork and veal, and accelerated their formation during storage. In the present study, although irradiation did not consistently produce significant increases in COPs, total COPs values, which indicate the overall progression of cholesterol oxidation, tended to increase in irradiated samples. In the case of breast meat this is most clearly seen in the E-400 group, where COPs were present in irradiated meat only. Irradiation accelerated COPs formation in some samples where TBARS values indicated that fatty acid oxidation was not affected. Similarly, Sevanian and McLeod (1987) reported that low levels of COPs were formed in an irradiated model system, in the absence of fatty acid oxidation. Cholesterol oxidation in meats is believed to be initiated by fatty acyl radicals arising from the oxidation of unsaturated membranal phospholipids (Osada *et al.*, 1993). The higher levels formed in irradiated samples may be due to the interaction of other prooxidants with cholesterol. Cholesterol is susceptible to attack by active oxygen species, including singlet oxygen and the hydroxyl radical (Gumulka *et al.*, 1982; Luby *et al.*, 1986). During mincing and cooking of meats, cellular membranes are ruptured, exposing the lipid-soluble membrane components to water-soluble prooxidants. In irradiated meat, exposure to higher levels of oxygen free radicals formed during the irradiation process, may have resulted in the formation of higher levels of COPs following storage.

Dietary α-tocopherol supplementation reduced the levels of COPs formed in irradiated and cooked meat. Total COPs values indicate that the antioxidant effect was greatest on increasing from the E-200 to the E-400 supplementation level. In breast, no decrease was observed as supplementation increased from E-100 to E-200, while levels decreased significantly when supplementation was increased to E-400. In thigh, a 1.2-fold decrease in total COPs was observed on increasing the supplementation level from E-100 to E-200. By comparison, supplementation with E-400 resulted in approximately a 2-fold reduction in total COPs values compared to supplementation with E-200. This is in contrast to the results of TBARS analysis, which indicated that supplementation with 200 mg α-tocopheryl acetate/kg feed was adequate to prevent the acceleration of fatty acid oxidation by irradiation.

Overall, the results show that irradiation following by cooking and storage had little effect on α-tocopherol stability in α-tocopherol-supplemented meat. While irradiation did accelerate fatty acid oxidation following

cooking and storage, the results suggest that supplementation with 200 mg α-tocopheryl acetate/kg feed is adequate to prevent this. Cholesterol oxidation in cooked meat was influenced by irradiation, although the effect was small. However, feeding 400 mg α-tocopheryl acetate/kg feed was necessary to practically inhibit cholesterol oxidation in breast meat. In the case of thigh, the optimum dietary concentration required to inhibit cholesterol oxidation in cooked irradiated and unirradiated meat would appear to be in the range of 400–600 mg/kg feed. In view of the current concerns regarding the potentially harmful effects of COPs on human health, further investigation of the occurrence of COPs in irradiated meats, and the effects of dietary α-tocopherol supplementation on their formation, is required.

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