Effects of oxidised oil and vitamin E on performance and some blood traits of heat-stressed male broilers

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Abstract

The present study was conducted so that the possible effects of thermally-oxidised dietary oil and vitamin E supplementation could be determined on the performance, lipid peroxidation, antioxidant defence system and some blood and meat quality traits of broilers. Broilers were fed grower diets containing fresh or oxidised oil, supplemented with and without vitamin E (200 mg/kg) from four to six weeks of age. Neither oxidised dietary oil nor vitamin E supplementation had any adverse effect on weight gain, feed consumption or feed conversion ratio. However, the broilers that received oxidised oil had lower levels of plasma cholesterol and triglyceride compared to the control. The glucose level was neither influenced by oxidised oil nor by vitamin E supplementation. The plasma malondialdehyde level increased slightly in broilers fed oxidised oil, indicating increased lipid peroxidation. Higher glutathione peroxidise ($GSH–P_x$) activity observed in the broilers fed oxidised oil suggests greater oxidative stress. Vitamin E supplementation partly depressed $GSH–P_x$ activity in broilers fed oxidised oil. This depression in enzyme activity might be the result of homeostatic compensation. The uric acid concentration was not affected by oxidised oil and vitamin E supplementation. Feeding oxidised oil did not cause a marked discolouration in breast meat. Vitamin E supplementation had some beneficial effects on colour measurements.

Keywords: Lipid peroxidation, oxidative stress, poultry, performance, meat colour
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Introduction

Vegetable oils, rich in polyunsaturated fatty acids (PUFAs), are highly digestible in chickens and are used in relatively large quantities in broiler diets to increase growth rate (Engberg *et al.*, 1996). However, oils are susceptible to oxidation during storage and after addition to the diet, especially under the promoting effects of oxygen, high temperatures or metallic catalysts. Lipid oxidation is a serious problem to both the food industry and the consumer, because it shortens the shelf life of meat and meat products, and increases the development of rancid odours and flavours. In addition, free radicals formed during oxidation may be a potential health risk to consumers (Jakobsen, 1999).

Furthermore, the usage of oxidised oil in diets can lead to growth depression in poultry (Cabel *et al.*, 1988; Lin *et al.*, 1989; Sheehy *et al.*, 1994; Dibner *et al.*, 1996; Engberg *et al.*, 1996; Jankowski *et al.*, 2000) and in other animal species (Keller *et al.*, 2004). It has been suggested that growth depression might be due to a reduction in feed intake as a result of reduced palatability and off flavour of rancid feed, or a decrease in digestibility of the oxidised oil (Wideman, 1986; Zdunczyk *et al.*, 2000; 2002).

The ingestion of oxidised oils also reduces tocopherol levels of tissues and plasma (Sheehy, 1993; Fei, 1995; Eder, 1999a; Jankowski *et al.*, 2000) and increases the amount of thiobarbituric acid-reactive substances (TBARS), suggesting an elevation in lipid peroxidation and cellular fragility (Asghar, 1989; Sheehy *et al.*, 1993; 1994; Fei, 1995; Enberg *et al.*, 1996; Eder & Kirchgessner, 1999; Juskiewicz *et al.*, 2000). Similarly, Zdunczyk *et al.* (2000) and Keller *et al.* (2004) reported that oxidised dietary oils lead to a reduced antioxidant status of erythrocytes and make them more susceptible to haemolysis. It is also suggested that these detrimental effects of oxidised dietary oils can be alleviated by vitamin E supplementation (Jakobsen *et al.*, 1993; Sheehy *et al.*, 1993; Dibner *et al*., 1996; Keller *et al.*, 2004).

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Increasing the fat content of diets helps birds to cope with heat stress and can overcome growth depression in broilers (Gous & Morris, 2005). However, special care should be taken to prevent fat oxidation at high ambient temperatures. Jankowski *et al.* (2001) reported that short and long-term feeding of diets containing oxidised oils decreased the final body weight of turkeys kept at high environmental temperatures. Furthermore, raising the fat level in diets by using vegetable oils, can promote oxidation problems in meat and meat products.

Heat stress stimulates the release of corticosterone from the adrenal gland, initiates lipid peroxidation in the cell membrane and leads to the generation of free radicals (Etches *et al.*, 1995). These free radicals can damage cell membranes by inducing lipid peroxidation of PUFAs in the cell membrane (Halliwell *et al.*, 1992; Yu, 1994; Etches *et al.*, 1995; Altan *et al.*, 2000; 2003). However, the peroxidation induced by heat stress might disappear upon the stimulation of the antioxidation ability (Lin *et al.* 2000). Puthpongsiriporn *et al.* (2001) also reported that lipid peroxidation in yolk and plasma, induced by heat stress, can be ameliorated by vitamin E supplementation. Therefore, the requirements of vitamin E for stabilization of cell membrane oxidation, are higher during heat stress (Kirunda *et al.*, 2001).

Generally, feed producers do not use oil with a high peroxide value in animal diets. However, feedstuffs stored in silos or broiler diets stocked on farms, especially during high summer temperatures, can easily undergo thermal oxidation, resulting in potentially toxic lipid peroxidation products. There are a few studies in which the effects of oxidised oil and vitamin E supplementation have been evaluated on the performance of broilers exposed to heat stress. Therefore, lipid peroxidation induced by heat stress and oxidised dietary oils in broilers must be taken into consideration.

The aim of the present study was to determine the possible effects of oxidised dietary oils on lipid peroxidation, some biochemical and production parameters of broilers reared under a high ambient temperature, and to determine whether vitamin E supplementation is able to alleviate the possible adverse effects of oxidised oil on lipid peroxidation. Colour measurements of breast muscle were also evaluated to determine whether oxidised dietary oil resulted in colour deterioration.

Materials and Methods

Three hundred and eighty-four day-old male chicks (Ross 308) were obtained from a commercial hatchery and randomly assigned to three dietary treatments with four replications (32 chicks per pen). The birds were maintained in an open-sided broiler house and received feed and water *ad libitum*. A lighting programme of 1 h dark : 23 h light was provided during the experimental period. The average daily temperature and relative humidity inside the house ranged from 30 to 35 °C and 50 to 70%, respectively. Individual body weights and feed intake for each pen were measured at four and six weeks of age, and mortality was recorded daily. The feed conversion ratio (FCR) was adjusted for mortality.

All birds were fed a commercial starter diet (235 g crude protein/kg and 12.68 MJ metabolizable energy/kg) for the first three weeks. From four to six weeks of age the experimental birds received one of three grower diets, all containing 13.3 MJ metabolizable energy/kg and 219.1 g crude protein/kg (Table 1). The basal diet (control), prepared with fresh sunflower oil, was supplemented with a vitamin premix containing 75 mg α-tocopherol acetate/kg. The second group received the basal diet containing oxidised sunflower oil (100 meq O_2/kg oil). The third group was fed the same diet as the second, but was supplemented with α-tocopherol acetate at a level of 200 mg/kg. The chemical composition of the diets was determined according to Verband Deutscher Landwirtschaftlicher Untersuchungs und Forschungsanstalten, VDLUFA (Naumann & Bassler, 1993). The metabolizable energy content of the diets was calculated from the chemical composition (TS, 1991). Sunflower oil was saturated with oxygen and intensively aerated during a heating process at 70 - 80 °C until the peroxide value reached 100 meq O_2/kg oil (Zduńczyk *et al.*, 2000). The peroxide analysis was performed in accordance with the procedures recommended by TS (2006).

At the end of the study, 12 broilers from each group were slaughtered and their *p. majors* were immediately removed from the right side of the carcasses, wrapped in film and stored at 4 °C for 24 h for pH and colour measurements. The pH values were measured in each breast muscle at three different locations, using a digital pH meter (Hanna Instruments H1 8314). Objective colour was measured at the surface of skinless breast samples using a reflectance spectrophotometer (Minolta CM 508d). Each colour parameter (L*= brightness, a*=redness and b*=yellowness) was taken at three different locations across the *p. major* (CIE, 1986).

A sample of 5 mL of blood was obtained from the wing vein of 13 birds per treatment and kept in an ice bath until the laboratory analysis could be performed. Blood samples were divided into two. The one subsample was used immediately after collection for antioxidant enzyme analysis. The second subsample was centrifuged at 4000 rpm for 10 min at 4 °C to separate plasma and stored at -70 °C, for later cholesterol, triglyceride and glucose analysis. Randox cholesterol, triglycerides and glucose Liquid Enzymatic Colorimetric Method kits (Randox Laboratories, Ardmore, Crumlin, UK) were used which are based on the CHOD-PAP, Glycerol-Phosphate Oxidase and GOD-PAP methods, respectively. Glutathione peroxidise ($GSH–P_x$) activity was determined using a commercially available enzyme kit (Ransel, RANDOX/RS-504) supplied by Randox Laboratories, Ardmore, Crumlin, UK. The Randox Uric Acid Enzymatic Colorimetric method kit was used for uric acid analysis. Uric acid is converted by uricase to allantoin and hydrogen

Table 1 The ingredient and chemical composition of starter and grower diets

Ingredients (g/kg)	Starter diet (0 to 3 weeks)	Grower diet (4 to 6 weeks)
Maize	504	494
Sunflower meal	56	64
Soybean meal	209	211
Full-fat soybean	120	129
Maize gluten meal	40	4.5
Dicalcium phosphate	16.7	13.4
Limestone	9.8	10.5
Sunflower oil	31.5	60
Vitamin mixture [1]	2	2
Mineral mixture [2]	1	1
Choline chloride	0.65	0.65
L-Lysine	3.9	3.5
DL-Methionine	1.45	1.7
L-Threonine	-	0.25
Salt	4.0	4.0
Coccidiostat[3]	0.5	0.5
Chemical composition (g/kg)		
Dry matter	918	928
Crude protein	235	219
Crude fibre	33.7	35.0
Crude ash	52.1	51.6
Ether extract	74.3	113
Total calcium	10.7	10.7
Total phosphorus	7.1	7.1
ME (MJ/kg)	12.68	13.30

[1] Provides per kg of diet: retinol acetate, 4.3 mg; cholecalciferol, 0.125 mg; tocopheryl, 75 mg; menadione, 4 mg; thiamine, 3 mg; riboflavin, 9 mg; niacin, 70 mg; calcium D-pantothenate, 18 mg; pyridoxine, 5 mg; cyanocobalamin, 0.02 mg; folic acid, 2 mg; D-biotin 0.1 mg; ronozyme P 750 FYT.
[2] Provides per kg of diet: manganese, 100 mg; iron, 60 mg; zinc, 60 mg; copper, 5 mg; cobalt, 0.2 mg; iodine, 1 mg; selenium, 0.15 mg.
[3] Provides per kg of diet: 66 mg/kg salinomycin sodium (Bio-Cox). Not supplemented to grower diet for the last five days of the experiment.

peroxide, which is under the catalytic influence of peroxidase, oxidizes 3.5-Dichloro-2-hydroxybenzenesulfonic acid and 4-aminophenazone to form a red-violet quinoneimine compound. Lipid peroxidation was measured using the TBA method (Satoh, 1978; Yagi, 1984). This method evaluates the oxidative stress assayed for malondialdehyde (MDA), the last product of lipid breakdown caused by oxidative stress.

Data was subjected to one-way ANOVA using the General Linear Model Procedure of SAS (1985). Means were compared using the Duncan test ($\alpha = 0.05$).

Results and Discussion

The effects of oxidised oil and vitamin E supplementation on broiler performance are shown in Table 2. Oxidised dietary oil and vitamin E supplementation did not affect body weight gain and feed intake during the four to six week experimental periods. These results agree with other studies which reported that body weight and feed intake were not adversely affected by oxidised oil in rats (Zdunczyk *et al*, 2000.; Eder *et al.*, 2002; Keller *et al.*, 2004) and in miniature pigs (Eder, 1999b). There were no differences in FCR of the treatment groups, which is in accordance with the results reported for broilers by Lin *et al.* (1989) and Engberg *et al.* (1996).

The findings showed that body weight and FCR were not adversely affected by oxidised oil and implied that oxidised oil did not alter palatability, odour or digestibility of the diet. In fact, it is reported that neither protein nor fat digestibility was affected by mildly oxidised oil (Zdunczyk *et al.*, 2000). In contrast, there are some studies that report depressed growth and impaired health in birds fed oxidised oil (Lin *et al.*, 1989; Jacobsen *et al.*, 1993; Sheehy *et al.*, 1993; Engberg *et al.*, 1996; Jankowski *et al*., 2000; Zdunczyk *et al.*, 2002; Eder *et al.*, 2003). In these studies, the detrimental effects of oxidised oil may be due to the administration of oils with high peroxide values or higher secondary lipid peroxidation products which have toxic effects. In the present study the peroxide value of the grower diet was 6.0 meq/kg and was lower than in most other studies. Interestingly, an increased cell proliferation in the gut and liver of birds fed oxidised oil occurred, suggesting that it may be related to a reduced FCR because of an increase in the maintenance requirement of these tissues (Fei, 1995; Dibner *et al.*, 1996). The supplementation of oxidised oil, with or without vitamin E, to grower diets did not affect the levels of glucose and total cholesterol in plasma. However, the triglyceride level in plasma was strongly influenced by the oxidised oil treatment. The birds fed oxidised oil had a significantly lower triglyceride level in their plasma than the control group. The depression in the triglyceride level caused by oxidised oil was less pronounced in the group supplemented with vitamin E (Table 3). The effects of oxidised oil on cholesterol and triglyceride concentrations are largely speculative. While some researchers obtained no significant effects in rats (Eder, 1999a) or birds (Juskiewicz, 2000), others reported that oxidised dietary oil reduces cholesterol and triglyceride concentrations (Eder & Kirchgessner, 1999; Eder, 1999b; Eder *et al.*, 2003). However, Narasimhamurthy & Raina (1999) observed higher cholesterol and lower triglyceride concentrations in the plasma of rats fed heated/fried oils compared to the fresh group. The reduction in cholesterol concentrations has been explained by elevated faecal excretion of cholesterol, impaired liver cholesterol uptake (Hocgraf *et al.*, 2000) and increased plasma thyroxin levels (Eder, 1999a). However, the oxidised oil-induced decline in triglyceride

Table 2 The effects of moderately-oxidised oil and vitamin E supplementation to grower diet on performance

Treatment groups	Body weight gain* (g) 4 to 6 weeks	Feed intake* (g) 4 to 6 weeks	FCR* (g/g) 4 to 6 weeks
Control	1517 ± 23.9	3240 ± 27.5	2.21 ± 0.06
Oxidised oil	1520 ± 30.6	3274 ± 22.2	2.17 ± 0.04
Oxidised oil + vitamin E	1516 ± 27.1	3327 ± 79.2	2.25 ± 0.05
Source of variation	Probabilities (*P* value)		
Group	0.995	0.494	0.570

* LS Mean ± SEM; FCR - feed conversion ratio (g feed/g gain).

concentrations might be due to a reduced *de novo* synthesis of fatty acids (Eder & Kirchgessner, 1998; 1999; Eder, 1999b). Fei (1995) suggested that birds fed oxidised oil had a higher glucose uptake capacity primarily due to an increase in the energy-dependent uptake. However, in the present study the plasma glucose level of the oxidised oil group did not differ from that of the fresh oil group.

There was a large individual variation in response to the oxidised oil treatment, ranging from 0.08 µM to 1.17 µM, but no statistically significant differences were obtained among treatment groups for MDA concentrations (Table 3). Nevertheless, the birds fed the oxidised oil diet showed a tendency towards a higher MDA concentration in erythrocytes compared to the control group, suggesting an increase in lipid peroxidation. In other words, the vitamin E level of the basal diet might be insufficient to ensure optimum protection against the damaging effect of oxidised oil. These results seem to agree with many previous reports in which higher TBARS levels were obtained in birds fed oxidised oil (Sheehy *et al.*, 1993; 1994; Engberg *et al*., 1996). Similar findings have also been reported in rats (Eder & Kirchgessner, 1999; Juskiewicz *et al.*, 2000; Eder *et al.*, 2003; Tabatabaei, 2008). However, Eder (1999a) suggested that the susceptibility of plasma lipids to lipid peroxidation was not significantly affected by feeding oxidised oil to miniature pigs.

Vitamin E, a major chain-breaking antioxidant of membranes, can scavenge the hydroxyl, alkoxyl, peroxyl and superoxide anion radicals and increase membrane stability (Surai, 2003). Dietary vitamin E supplementation has been shown to protect cells from oxidative stress mediated by oxidised oil and to improve the stability of meat (Lin *et al.*, 1989; Wang *et al*., 1997; Asghar *et al.*, 1999). Supporting these results was the negative correlation between the dietary vitamin E and hepatic TBARS levels obtained by Mercier *et al.* (1998) and Guo *et al.* (2001).

In this study, although the effect of vitamin E supplementation was not statistically significant, the lower MDA levels of the vitamin E supplemented group (0.49 ± 0.09 µM) compared with the oxidised oil group (0.60 ± 0.05 µM) might be a sign of the beneficial effect of vitamin E in alleviating oxidative damage induced by the oxidised oil. In agreement with our findings, Eder & Kirchgessner (1998) reported that with a low vitamin E supply, the susceptibility of low-density lipoprotein (LDL) to lipid peroxidation was slightly increased by feeding oxidised oil. In contrast, with a high vitamin E supply, there was no adverse effect by using oxidised dietary oil on the susceptibility of LDL to lipid peroxidation. Higher levels of vitamin E may be sufficient to scavenge free radicals or increase fatty acid saturation and decrease instauration, thus making lipids more resistant to lipid peroxidation (Barja *et al.*, 1996; Iqbal *et al.*, 2002). The activity of GSH-Px was significantly different, with the oxidised oil group having the highest value, the vitamin E supplemented group the lowest value and fresh oil group having an intermediate GSH-Px activity (Table 3).

Table 3 The effects of moderately-oxidised oil and vitamin E supplementation in the grower diet, on the plasma lipid and glucose levels, lipid peroxidation and antioxidant defence system

Treatment groups	Triglyceride* (mmol/L)	Cholesterol* (mmol/L)	Glucose* (mmol/L)	MDA* (µM)	GSH-Px* (U/L)	Uric acid* (µmol/L)
Control	0.65^{a} ± 0.10	2.60 ± 0.10	11.7 ± 0.20	0.39 ± 0.06	136^{ab} ± 23.4	335 ± 49.1
Oxidised oil	0.33^{b} ± 0.05	2.38 ± 0.23	11.6 ± 0.30	0.60 ± 0.05	173^{a} ± 31.0	394 ± 60.6
Oxidised oil + vitamin E	0.47^{ab} ± 0.09	2.53 ± 0.09	11.5 ± 0.33	0.49 ±0.09	89.2^{b} ± 9.23	380 ± 62.9
Source of variation	Probabilities (*P* value)					
Group	0.035	0.635	0.883	0.194	0.041	0.771

* LS - Mean ± SEM.
[a,b] LS - Means within columns with no common superscript differ significantly ($P < 0.05$).
MDA – malondialdehyde; GSH-Px – glutathione peroxidise.

A large variation in GSH-Px activity appears to have been caused by individual variation in response to the oxidised oil treatment. Shaaban *et al.* (2003) also obtained significant differences in GSH-Px activity in different genotypes of chicken. GSH-Px removes hydrogen peroxide and lipid hydroperoxides from cells

(Shaaban *et al*., 2003; Surai, 2003). Therefore, a higher GSH-Px activity in birds fed oxidised oil, can be explained by enzyme synthesis which may be an adaptive response to greater hydrogen peroxide production and to greater oxidative stress in the present study. Supporting this result, Davies (2000) and Surai (2003) reported that organs can adapt to oxidative stress by increasing their production of antioxidant and repair enzymes and restore the antioxidant/pro-oxidant balance. A similar response has been reported for rats, in which feeding oxidised oil resulted in increased antioxidant enzyme activities (Hayam *et al.*, 1995; Juskiewicz *et al.*, 2000). Furthermore, Iqbal *et al.* (2002) suggested that higher GSH-Px and SOD activities observed in the liver tissue of broilers with ascites, may be the response to a greater degree of oxidative stress. Supporting our findings, Maraschiello *et al.* (1999) observed that lipid and cholesterol oxidation were positively correlated with GSH-Px activity, suggesting that the GSH-Px activity can be used as an indicator of meat oxidative stability. However, some contradictory results were reported for antioxidant enzyme activity in relation to dietary oxidised oil. While Engberg *et al.* (1996) reported that the total GSH-Px activity in broilers was not influenced by oxidised oil, Keller *et al.* (2004) suggested that oxidised dietary oils reduced the antioxidant enzyme activities such as super oxide dismutase (SOD), catalase (CAT) and GSH-Px of erythrocytes in rats and guinea pigs.

In the present study the broilers were reared at a high ambient temperature, and the heat stress caused increases in MDA levels and antioxidant enzyme activities (GSH-Px, SOD and CAT), leading to oxidative stress (Altan *et al.*, 2000; 2003). Eder *et al.* (2002) also indicated that feeding rats with oxidised fat reduced the activity of GSH-Px in either high or low selenium diets. In birds fed oxidised oil, supplementing the diet with 200 mg vitamin E/kg depressed the GSH-Px activity in erythrocytes (Table 3). It seems possible, therefore, that the higher level of vitamin E might control the level of oxygenated radical scavenging systems such as GSH-Px and depress antioxidant enzyme activities by homeostatic compensation. The decrease in the GSH-Px activity found in the present study, agrees with that of Maraschiello *et al.* (1999) who reported a lower oxidant enzyme activity in broilers supplemented with α- tocopherol. In some stress conditions, the first stage of antioxidant defence in cells (antioxidant enzymes and metal binding proteins) is not able to prevent free radical formation. In this case, the second stage of defence consists of chain-breaking antioxidants. Uric acid, an end-product of purine metabolism, is a chain-breaking antioxidant and scavenger of single oxygen and radicals (Surai, 2003). In the present study, feeding oxidised oil and vitamin E supplementation did not alter uric acid levels in treatment groups, suggesting more mildly oxidative stress (Table 3).

Table 4 The effects of moderately-oxidised oil and vitamin E supplementation in the grower diet, on meat colour and pH values of breast meats (LS Mean ± SEM)

Treatment groups	Colour parameters			pH
	L*	a*	b*	
Control	42.1 [b] ± 0.78	-1.07 ± 0.09	6.37 ± 0.34	5.83 ± 0.01
Oxidised oil	46.1 [a] ± 1.15	-0.80 ± 0.12	6.15 ± 0.30	5.84 ± 0.02
Oxidised oil + vitamin E	47.1 [a] ± 1.29	-1.16 ± 0.12	5.49 ± 0.32	5.82 ± 0.02
Source of variation	Probabilities (*P* value)			
Group	0.0275*	0.0644	0.1419	0.8808

[a, b] Means within columns with no common superscript differ significantly ($P < 0.05$).
L* - brightness, a* - redness; b* - yellowness.

There were no significant differences between the treatments in redness (a*), yellowness (b*) or pH, whereas the L* value was significantly lower in the control group compared to the oxidised oil and vitamin E supplemented groups (Table 4). As the b* value increases, poultry meat is characterized as yellow-brown whereas lower b* values cause poultry meat to go ivory-cream pink (Jensen *et al.*, 1997). Lower L* and slightly higher b* values in the control group indicated darker coloured breast meat compared to the vitamin

E supplemented group. Colour differences on CIE-Lab coordinates can be discerned by the most normal observers, and consumers can detect one CIE-Lab unit change in colour coordinates (Zhu & Brewer, 1999). Although there were no statistically significant differences in the b* values of groups, colour development in the vitamin E supplemented group could be detected by the naked eye as light coloured breast meat, compared to other groups. Likewise, dietary vitamin E increases the stability of membrane-bound lipids and delays oxidation of red oxymyoglobin to brown metmyoglobin, thus improving the colour stability of the meat. These beneficial effects of vitamin E have been reported by Faustman *et al.* (1998) and Morrisey *et al.* (1998). However, Coetzee & Hoffman (2001) were not able to obtain a beneficial effect of vitamin E on colour measurements in broiler meat. In turkey meat, Mercier *et al.* (1998) also reported that vitamin E supplementation delays lipid oxidation, but has no positive effect on colour stability.

Conclusion

We have hypothesized that detrimental effects of dietary oxidised oil are more pronounced in broilers reared at high ambient temperatures than at lower temperatures. Surprisingly, in spite of a high ambient temperature, feeding moderately oxidised oil did not cause drastic decreases in growth, feed consumption or feed efficiency of the broilers. Although the consumption of oxidised oil induces oxidative stress, there was only a slight increase in GSH-Px activity and MDA level. Supplementation of a diet with vitamin E (200 mg/kg) resulted in some alleviation of these adverse effects. Feeding oxidised oil did not cause a marked discolouration of breast meat, although vitamin E supplementation had a beneficial effect on colour measurements.

In conclusion, birds receiving adequate nutrients, particularly protein and vitamin E, were able to metabolise the undesirable components in oxidised oil and respond to mild oxidative stress by reregulating antioxidant defences and other protective mechanisms that exist in the cell for maintaining homeostasis.

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