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Changes in broiler chick tissue concentrations of lipid-soluble antioxidants immediately post-hatch

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ABSTRACT

The antioxidant protection of the chicken (*Gallus gallus*) embryo during incubation and early postnatal development plays an important role in chick viability. To assess the antioxidant capacity of the newly hatched chick, we determined the concentrations of vitamin A, vitamin E, carotenoids and coenzyme Q_{10} in the major tissues of chicks which had been held in an incubator for up to 36 h post-hatch. Concentrations of total carotenoids and free retinol and retinol esters in the tissues did not differ significantly over the 36 h period post-hatch ($p>0.05$). In contrast concentrations of vitamin E (α-tocopherol, γ-tocopherol, and α-tocotrienol and γ-tocotrienol) in various tissues (liver, heart, brain and leg muscle) decreased significantly in chicks that had been held in the incubator for 36 h when compared to younger chicks that were held for up to 18 h. Comparatively high concentrations of coenzyme Q_{10} were detected in the yolk sac membrane, liver and heart, the concentrations being dependent on age of chicks, the highest value being recorded 18 h post-hatch. In most of the tissues studied, coenzyme Q_{10} concentrations decreased substantially between 18 and 36 h post-hatch. This study demonstrated that there are tissue-specific changes in the concentrations of the major antioxidants (vitamin E and coenzyme Q_{10}) during the 36 h post-hatch.

1. Introduction

Traditional breeding programmes were designed to produce a broiler with the potential to maximise growth, yield and feed efficiency. These traits can however compromise health status since there is a trade-off between growth- and health-related traits (Siegel et al., 2001). Chick viability is an important factor in determining profitability and, from fertilisation to placement at the broiler farm, factors such as egg quality, egg storage conditions, incubation conditions and post-hatch environment will all affect chick quality (Decuypere et al., 2001).

The 21 day incubation period and the 10 day post-hatch period of the chick composes about 50% of a 2 kg broiler's lifespan in current intensive production system. Therefore, anything that hinders or promotes growth and development during this neonatal period will have a marked effect on overall performance and health of poultry (Ferket, 2006). So the success of embryonic development depends on the interactions between egg composition and the conditions of egg incubation. During chick embryo development there is an antioxidant/prooxidant balance in the tissues which supports normal embryonic development and post-hatch chick viability. It has been suggested that an accumulation of the natural antioxidants like vitamins A, E and carotenoids as well as an increase in GSH-Px activity in the embryonic liver may have an adaptive significance, evolving to protect unsaturated lipids against peroxidation during the stress imposed by hatching (Surai, 2002). It seems likely, that in postnatal development there is a different strategy in relation to antioxidant defence compared to the embryonic chicken (Surai, 2000). For example, during mammalian prenatal development the antioxidant system is considered to be immature (Allen and Venkatraj, 1992; Fantel, 1996) with maturation occurring in the postnatal period. Probably the same is true in avian embryo development i.e. an embryo relies on natural antioxidants accumulated in the egg yolk to protect tissues during hatching against lipid peroxidation.

Antioxidant protection at hatching time is considered to be an important determinant of chick viability during first post-hatch days (Surai, 1999a, 2000). Depending on the so-called spread of hatch (i.e. the time from the first to last chick to hatch) it would not be uncommon for a significant percentage of any hatch to spend up to 36 h in the hatcher having emerged from the egg. Given the relatively high temperature and humidity in the hatcher, it is easy to make the argument that the chick may be under chronic oxidative stress during this holding time. However antioxidant defences during this important time have not been studied yet.

The objective of our work, therefore, was to investigate the content of commonly recognised antioxidants (vitamin A, E, total carotenoids

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and coenzyme Q_{10}) in the tissues of chicks held in the incubator for times ranging from 0 to 36 h.

2. Material and methods

2.1. Chicks (*Gallus gallus*)

In the incubator 100 Hubbard Flex eggs (10 eggs on each of 10 trays) were marked and incubated according to standard hatchery practices (37.5 °C). The humidity of incubator was about 58–60% during 1–18 days. After day 18, the humid was raised to 65%. From each tray 1 chick was taken for laboratory analyses at 0, 18 and 36 h after hatching (in total 30 chicks).

All chicks were dissected and tissue mass were assessed (n = 10). After that, tissues (liver, residual yolk (YSR), yolk sac membrane (YSM), lung, brain, heart, leg and breast muscle) from 5 chicks per group were taken for biochemical analysis (n = 5). Carotenoids, vitamins A, E and coenzyme Q_{10} were assessed in tissues.

The study was approved by the SAC Animal Ethics Committee.

2.2. Analytical work

Vitamins in tissues were determined by high-performance liquid chromatography (HPLC) as previously described (Surai et al., 2001). An aliquot of tissue was vortexed in 0.7 mL 5% NaCl, then 1 mL ethanol was added and homogenized (1 min), during homogenization 2 mL hexane was added. Samples were then centrifuged and the hexane phase, containing the carotenoids, was collected. Extraction with hexane was repeated and the combined hexane phase was dried under N_2 gas, and then re-dissolved in dichloromethane/methanol (1:1 v/v). Carotenoids, vitamin A (retinol and retinol esters), E and coenzyme Q_{10} were quantified by HPLC.

Vitamin E (α-, γ-, δ- tocopherols and α-, γ-tocotrienols) were determined as previously described (Surai and Speake, 1998a, b) using an HPLC system (Shimadzu Liquid Chromatography, LC-10AD, Japan Spectroscopic Co Ltd. with JASCO Intelligent Spectrofluorometer 821-FP) fitted with a Spherisorb, type S30DS2, 3 μ C_{18} reverse phase HPLC column, 15 cm × 4.6 mm (Phase Separations Limited, UK). Chromatography was performed using a mobile phase of methanol/water (97:3, v/v) at a flow rate of 1.05 mL/min. Fluorescence detection of tocopherols and tocotrienols involved excitation and emission wavelengths of 295 and 330 nm respectively. Standard solutions of tocopherols and tocotrienols in methanol were used for instrument calibration and tocol was used as an internal standard.

Coenzyme Q_{10} was analysed in the same extract by injecting 50 μL into the same HPLC system, but using a Vidac 201TP54 column (5 μm, 25 cm × 4.6 mm) and mobile phase ethanol/methanol/2-propanol (70:15:15 by volume) and flow rate 1.5 mL/min with a diode array detection at 275 nm (Mattila and Kumpulainen, 2001). Coenzyme Q_{10} (Sigma) standard was used for calibration.

Total carotenoids were analysed by injecting 10 μL into the same HPLC system, using Waters NH2 column (5 μm, 25 cm × 4.6 mm) and mobile phase methanol/water (97:3 by volume) and with a flow rate of 1.5 mL/min with a diode array detection at 444 nm (Surai et al., 1998a, b).

Retinol and retinol esters were determined by injecting 10 μL into the HPLC system, but using Waters supherisorb ODS 2, 5 μ C_{18} reverse phase HPLC column, 25 cm × 4.6 mm (Phase Separations Limited, UK) with a diode array detection at 325 nm (Karadas et al., 2005).

2.3. Statistical analysis

Statistical analysis was carried out by the General Linear Models (GLM) procedure of SAS (statistical Software for Windows 6.12, 1996; SAS Inc., Cary, NC, USA). Significant differences between treatment means were examined by Duncan's multiple range tests. Significance was assigned as a level of $p < 0.05$ unless otherwise stated.

3. Results

The body mass (36.21 ± 0.94; 36.85 ± 2.52; 35.32 ± 0.73 g) and liver mass (0.92 ± 0.04; 1.02 ± 0.05; 1.00 ± 0.05 g) of chicks (0, 18 and 36 h respectively) were not affected by the age of birds (different holding times in the incubator). However, the masses of the yolk sac membrane (YSM) (0.33 ± 0.05^a; 0.18 ± 0.02^b; 0.14 ± 0.012^b g) and residual yolk (YSR) (4.07 ± 0.30^a; 3.76 ± 0.20^b; 2.85 ± 0.16^b g) were reduced significantly ($p < 0.05$) for chicks held in the incubator for 18 h or more.

The concentrations (μg/g fresh tissue) of the free retinol and retinol esters (R-linoleate, R-oleate, R-palmitate and R-stearate) in liver and yolk sac membrane are shown in Table 1. The only significant changes in the concentrations of these compounds however were the retinol concentrations, which decreased after 18 h, and the total vitamin A concentration in the YSM which peaked after 18 h and decreased again by 36 h post-hatch.

The total carotenoids concentrations (μg/g) are presented in Table 2. Generally there were no significant changes in the concentration of carotenoids in the various tissues, the only changes occurring in the brain and heart where there was a tendency for the concentrations to decrease by 36 h post-hatch.

The vitamin E and coenzyme Q_{10} concentrations are shown in Table 3. As would be predicted the concentrations of vitamin E were greatest in the liver and the YSM with values in other samples being <10% of those recorded in the liver and the YSM. In general, in the liver alpha-tocopherol represents about 85.7% of total vitamin E, while gamma-tocopherol, alpha- and gamma-tocotrienol represent 11.4, 2.4 and 0.5 respectively. Vitamin E concentration in the liver is about 3-fold higher than coenzyme Q_{10} concentration in this tissue. In the YSM this difference is only 2-fold, while in other tissues (lung, heart, brain, breast and leg muscles) coenzyme Q_{10} concentrations are several fold higher than that of alpha-tocopherol.

The concentration of coenzyme Q_{10} did vary among tissues, or for liver and yolk sac membrane for the different holding periods ($p < 0.05$). The highest concentration was detected in YSM while the lowest was detected in the YSR. Coenzyme Q_{10} concentration even if it is minor in most tissues, decreased significantly and reached significant level in liver, brain, heart, lung and YSM ($p < 0.05$) at 36 h.

4. Discussion

The antioxidant system of the chicken embryo and newly hatched chick has been studied extensively (Surai, 2002). It was shown that it includes fat-soluble antioxidants (vitamin E and carotenoids) originating from the maternal diet (Surai et al., 1996), as well as water-soluble antioxidants (ascorbic acid, glutathione, and uric acid) and antioxidant enzymes (superoxide dismutase, glutathione peroxidase and catalase; Surai, 1999a) which are synthesised during embryonic development (Surai et al., 1998a; Surai, 1999a).

We would argue that because hatching places a stress upon the emerging chick that natural antioxidant concentrations (vitamin E and carotenoids) have evolved to reach the maximum to protect the unsaturated lipid in the tissues and so limit lipid peroxidation (Surai et al., 1998a; Surai, 1999a). Furthermore, any delay in accessing food (Noy et al., 2001; Bigot et al., 2003) and/or water intake after hatching as well as hatchery treatments such as vaccination, sexing and transport to the farm can result in additional stress (Geyra et al., 2001). Tocopherol and tocotrienol concentrations in various chicken tissues reported in this study were comparable with those published earlier (Surai and Speake, 1998a; Surai et al., 1999a). Previously a dramatic decrease in vitamin E concentrations in the chicken liver for the first 10 days of post-hatch development has been shown for

Table 1
Concentration of retinol and retinol esters (μg/g) in liver and yolk sac membrane.

Holding time (h)	Retinol	R-linoleate	R-oleate	R-palmitate	R-stearate	Total vitamin A
Liver						
0	$0.66 \pm 0.058^{\mathbf{b}}$	0.66 ± 0.088^{a}	2.39 ± 0.18^{a}	7.21 ± 0.45^{a}	2.39 ± 0.26^{a}	13.32 ± 0.69^{a}
18	$2.90 \pm 0.76^{\mathbf{a}}$	0.76 ± 0.15^{a}	1.78 ± 0.50^{a}	5.18 ± 1.55^{a}	1.60 ± 0.41^{a}	12.21 ± 2.08^{a}
36	$2.03 \pm 0.35^{\mathbf{a}}$	0.77 ± 0.071^{a}	1.88 ± 0.19^{a}	5.67 ± 0.57^{a}	2.23 ± 0.39^{a}	12.59 ± 1.52^{a}
Holding time (h)	Retinol	R-linoleate	R-oleate	R-palmitate	R-stearate	Total vitamin A
Yolk sac membrane						
0	1.66 ± 0.24^{a}	2.82 ± 1.22^{a}	4.38 ± 0.22^{a}	11.97 ± 0.78^{a}	4.27 ± 0.19^{a}	25.15 ± 1.63^{b}
18	1.40 ± 0.15^{a}	2.75 ± 0.60^{a}	5.37 ± 0.49^{a}	16.70 ± 0.80^{a}	6.57 ± 0.74^{a}	32.72 ± 1.27^{a}
36	1.43 ± 0.18^{a}	0.81 ± 0.60^{a}	6.77 ± 1.79^{a}	13.73 ± 2.80^{a}	3.93 ± 1.56^{a}	26.67 ± 3.38^{ba}

Values are means ± S.E.M. ($n = 5$). Means of each factor in columns are not sharing a common superscript differ significantly ($P < 0.05$).

Table 2
Total carotenoids concentration (μg/g) in the day-old chick tissues held at different period in the incubator.

Holding time (h)	Liver	YSM	YSR	Heart	Lung	Brain	Breast muscle	Leg muscle
0	16.76 ± 4.36^{a}	22.34 ± 3.10^{a}	3.37 ± 0.28^{a}	1.69 ± 0.32^{a}	4.57 ± 1.31^{a}	1.64 ± 0.31^{ba}	1.63 ± 0.42^{a}	1.47 ± 0.30^{a}
18	15.50 ± 2.40^{a}	22.68 ± 2.18^{a}	5.96 ± 1.12^{a}	2.59 ± 0.42^{a}	7.66 ± 1.99^{a}	2.38 ± 0.28^{a}	2.16 ± 0.41^{a}	2.51 ± 0.44^{a}
36	16.54 ± 1.04^{a}	$27.9342.19^{a}$	4.99 ± 1.68^{a}	1.20 ± 0.32^{a}	5.88 ± 0.44^{a}	0.24 ± 0.04^{b}	2.40 ± 0.03^{a}	1.56 ± 0.16^{a}

Values are means ± S.E.M. ($n = 5$). Means of each factor in columns are not sharing a common superscript differ significantly ($P < 0.05$).

chickens, turkey, duck and goose (Surai et al., 1998b). An increase in dietary vitamin E supplementation slowed down this process but did not change the trend (Surai et al., 1999a; Surai, 1999b; for review see Surai, 2002).

Total carotenoids concentrations in various chicken tissues reported in this study were comparable with those published earlier (Surai and Speake, 1998b; Surai et al., 1999b). Retinol and retinol esters in YSM and liver were comparable to our previous results (Karadas et al., 2005).

Coenzyme Q is the only fat-soluble antioxidant synthesised in body, originating from endogenous synthesis and from food. There are two major forms of coenzyme Q in the human tissues namely coenzyme Q_{10} comprising 95–97% in heart, kidney, liver and spleen and 92% in brain and 87% in lung (Dallner and Sindelar, 2000), the remainder, coenzyme Q_9. However specific roles for coenzyme Q_{10} as an antioxidant component of the chicken antioxidant system have not been characterised.

This is the first study to determine coenzyme Q_{10} in a range of chicken tissues. The coenzyme Q_{10} concentration in residual yolk was 1.45–2.10 μg/g, with this data being consistent with the results of Weber et al. (1997); Overvad et al. (1999) and Mattila and Kumpulainen (2001), leg tissues (mean 21.55 μg/g), breast muscle (mean 11.70 μg/g) and muscle tissues (mean 16.62 μg/g) are consistent with the results of Overvad et al. (1999).

Table 3
The concentration of vitamin E and coenzyme Q_{10} (μg/g) in the tissues of chicks incubated in the incubator.

Tissues	Holding time (h)	α-tocopherols	γ-tocopherols	α-tocotrienols	γ-tocotrienols	Coenzyme Q_{10}
Liver	0	145.47 ± 9.75^{a}	19.37 ± 2.12^{a}	4.15 ± 0.14^{a}	0.85 ± 0.17^{a}	44.41 ± 5.11^{b}
	18	155.51 ± 10.77^{a}	23.24 ± 1.69^{a}	2.68 ± 0.60^{b}	0.63 ± 0.15^{a}	64.84 ± 2.73^{a}
	36	80.63 ± 5.63^{b}	8.20 ± 1.76^{b}	0.93 ± 0.16^{c}	0.05 ± 0.012^{b}	42.74 ± 4.22^{b}
Yolk sac membrane	0	127.94 ± 22.30^{a}	22.66 ± 2.5^{a}	3.70 ± 0.91^{a}	1.60 ± 0.24^{a}	67.45 ± 4.06^{a}
	18	110.67 ± 8.73^{a}	17.7 ± 0.80^{ab}	2.61 ± 0.67^{a}	0.79 ± 0.43^{ba}	92.15 ± 8.29^{a}
	36	90.26 ± 21.78^{a}	12.27 ± 3.22^{b}	1.97 ± 3.34^{a}	nd ^{b}	29.51 ± 3.32^{b}
Yolk sac residual	0	11.63 ± 1.40^{a}	2.28 ± 0.36^{a}	0.48 ± 0.07^{a}	0.34 ± 0.04^{a}	1.45 ± 0.18^{a}
	18	15.94 ± 3.74^{a}	4.26 ± 1.08^{a}	0.43 ± 0.11^{a}	0.03 ± 0.02^{b}	1.55 ± 0.39^{a}
	36	6.71 ± 2.11^{a}	3.32 ± 1.39^{a}	0.27 ± 0.08^{a}	nd ^{b}	2.10 ± 0.38^{a}
Lung	0	0.68 ± 0.13^{b}	0.90 ± 0.01^{b}	0.024 ± 0.01^{a}	1.60 ± 0.24^{a}	16.23 ± 5.04^{a}
	18	3.52 ± 0.58^{a}	1.9 ± 0.50^{a}	0.13 ± 0.07^{a}	0.79 ± 0.43^{ba}	9.23 ± 0.65^{a}
	36	1.52 ± 0.18^{b}	0.69 ± 0.18^{b}	0.06 ± 0.03^{a}	nd ^{b}	4.76 ± 1.01^{b}
Heart	0	4.52 ± 0.58^{a}	1.60 ± 0.19^{a}	0.32 ± 0.04^{a}	0.16 ± 0.07^{a}	39.14 ± 2.85^{a}
	18	4.16 ± 0.59^{a}	1.83 ± 0.16^{a}	0.16 ± 0.06^{ba}	0.12 ± 0.05^{a}	36.24 ± 4.86^{a}
	36	1.26 ± 0.22^{b}	0.41 ± 0.11^{b}	0.06 ± 0.04^{b}	nd ^{b}	8.52 ± 1.62^{b}
Brain	0	1.64 ± 0.31^{ab}	0.27 ± 0.09^{a}	0.46 ± 0.34^{a}	0.09 ± 0.05^{a}	17.13 ± 1.34^{a}
	18	2.51 ± 0.31^{a}	0.34 ± 0.08^{a}	0.49 ± 0.20^{a}	0.02 ± 0.00^{b}	12.95 ± 1.33^{a}
	36	1.22 ± 0.13^{b}	0.23 ± 0.02^{a}	0.10 ± 0.04^{a}	nd ^{b}	6.20 ± 1.86^{b}
Breast muscle	0	4.43 ± 0.17^{b}	0.86 ± 0.04^{a}	0.34 ± 0.06^{a}	0.10 ± 0.025^{a}	11.46 ± 2.28^{a}
	18	7.78 ± 1.13^{a}	1.45 ± 0.27^{a}	0.39 ± 0.13^{a}	0.13 ± 0.035^{a}	11.92 ± 1.26^{a}
	36	4.85 ± 0.35^{b}	1.13 ± 0.10^{a}	0.14 ± 0.03^{a}	nd ^{b}	11.73 ± 2.43^{a}
Leg muscle	0	4.02 ± 0.27^{a}	0.90 ± 0.09^{b}	0.24 ± 0.025^{b}	0.09 ± 0.008^{ba}	20.48 ± 1.13^{a}
	18	5.36 ± 0.83^{a}	1.46 ± 0.18^{a}	0.39 ± 0.22^{a}	0.17 ± 0.045^{a}	22.40 ± 1.65^{a}
	36	3.72 ± 0.34^{a}	0.99 ± 0.09^{b}	0.24 ± 0.050^{b}	nd ^{b}	21.76 ± 2.18^{a}

Values are means ± S.E.M. ($n = 5$). Means of each factor in columns not sharing a common superscript differ significantly ($P < 0.05$). nd: not detected.

In general, dietary supplementation of coenzyme Q_{10} does not affect the endogenous synthesis of coenzyme Q_{10} in tissues. However, oxidative stress (physical exercise, thyroid hormone treatment, cold adaptation, vitamin A deficiency, etc.) is associated with increased coenzyme Q synthesis reflecting cellular adaptation (Ernest and Dallner, 1995). Therefore, coenzyme Q_{10} synthesis is considered to be an adaptive mechanism in response to stress conditions when other antioxidants are depleted. For example, in vitamin E and Se deficient rats the concentration of coenzyme Q_{10} was elevated and the coenzyme Q-dependent reductase system was activated (Navarro et al., 1998).

It is believed that exogenous coenzyme Q protects cells from oxidative stress by conversion into its reduced antioxidant form by cellular reductases. In particular cytosolic NADPH-coenzyme Q reductase is responsible for cellular coenzyme Q redox cycle as an endogenous antioxidant (Kishi et al., 1999). The plasma membrane oxidoreductase and DT-diaphorase are two such systems, likewise, they are overexposed under oxidative stress conditions (Genova et al., 2003). In addition, the selenoenzyme thioredoxin reductase is an important ubiquinone reductase and can explain how selenium and coenzyme Q, by a combined action, may protect the cell from oxidative damage (Xia et al., 2003). Since coenzyme Q is an essential part of oxidative phosphorylation complex in mitochondria the majority (molar amounts) of endogenous coenzyme Q is found in these organelles. However, exogenous coenzyme Q is usually found in the extra-mitochondrial fractions including lysosomes and Golgi vesicles (Dallner and Sindelar, 2000). From our data it is not clear if the increased coenzyme Q_{10} concentration in the liver at 18 h of chick holding is an adaptive reaction to the stress, but clearly combined with a high vitamin E concentration coenzyme Q_{10} potentially can take place in antioxidant defences in the chicken liver. On the other hand, decreased coenzyme Q_{10} concentration at 36 h of chick holding in conjunction with substantial (almost two-fold) decrease in vitamin E concentration in the liver could be a reflection of the redistribution of these antioxidants. Alternatively, excessive oxidative stress at this time could cause an imbalance between coenzyme Q_{10} synthesis, transport and usage.

The presence of high concentrations of coenzyme Q_{10} in all membranes provides a basis for antioxidant action either by direct reaction with radicals or by regeneration of alpha-tocopherol from its oxidised form. In fact, a protective effect of coenzyme Q_{10} against lipid peroxidation was shown in fatty acid emulsions, mitochondria, sub-mitochondrial particles and other model systems (Ernest and Dallner, 1995). Coenzyme Q_{10} protects not only membrane phospholipids from peroxidation but also mitochondrial DNA and membrane proteins from free-radical-induced oxidative damage (Pobezhimova and Voinikov, 2000). Taking into account the decrease of coenzyme Q_{10} concentrations in other tissues as well as similar changes in vitamin E compounds we are in favour of oxidative stress as a possible cause of these changes. However, there is a need for further research to identify possible molecular mechanisms and consequences of such changes.

In conclusion, this study demonstrated that there are tissue-specific changes in the concentrations of the major antioxidants (vitamin E and coenzyme Q_{10}) during the 36 h post-hatch. Indeed, there is a need for further research in this fascinating area.

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