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Antioxidant systems of the avian embryo: tissue-specific accumulation and distribution of vitamin E in the turkey embryo during development

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Abstract 1. Tissue-specific accumulation of tocopherols and tocotrienols in turkey tissues during embryonic development and their susceptibility to lipid peroxidation were investigated.
2. Fertile turkey eggs were incubated using standard commercial conditions. Embryonic tissues were collected at 16, 22, 25 d of incubation and from day-old poults (referred to as day 29) and α-; β-+γ- and δ-tocopherols and respective tocotrienols were analysed by HPLC.
3. A turkey diet provided to the parent hens contained the complete range of tocopherols and tocotrienols. Between days 16 and 22 of embryo development, the α-tocopherol concentration in the liver remained constant and then increased significantly ($P<0{\cdot}01$) reaching a maximum just after hatching. Similar changes were observed for the other tocopherols and tocotrienols.
4. The accumulation of α-tocopherol in the yolk sac membrane (YSM) started after day 20 of development and at hatching the α-tocopherol concentration in the YSM was twice that of β-+γ-tocopherols and 15 times greater than that of α-tocotrienol.
5. In the kidney, heart, lung, muscle and adipose tissues a gradual increase in tocopherol and tocotrienol concentrations took place between days 20 and 25 of development with a sharp increase in particular of α-tocopherol between days 25 and 29. There was a discrimination between tocopherols and tocotrienols during their assimilation from the diet by the parent hen and during metabolism by the developing turkey embryo.
6. Tissue-specific features in the susceptibility to lipid peroxidation were found with the brain being the most susceptible to lipid peroxidation at day 25 and in day-old poults.

INTRODUCTION

The development of the avian embryo is associated with the accumulation of highly polyunsaturated lipids within the tissues (Noble and Speake, 1997) with the rate of oxidative metabolism increasing dramatically over the hatching period (Freeman and Vince, 1974). In such conditions, oxidative stress may be a problem during the last days of prenatal and 1st days of postnatal chick life. These necessitate the development of effective antioxidant capacities in the tissues to prevent lipid peroxidation.

The antioxidant system of the developing chicken embryo consists of the natural antioxidants vitamin E (Noble *et al.*, 1993; Gaal *et al.*, 1995; Surai *et al.*, 1996), carotenoids (Surai *et al.*, 1995, 1996), ascorbic acid (Wilson *et al.*, 1990; Surai *et al.*, 1996), glutathione and the antioxidant enzymes superoxide dismutase, glutathione peroxidase and catalase (Surai, 1999). The chicken embryo accumulates fat-soluble antioxidants in the liver and yolk sac membrane (YSM) during the last week of development (Gaal *et al.*, 1995; Surai *et al.*, 1996). Turkeys, ducks and geese also accumulate vitamin E and carotenoids in the liver and YSM (Surai *et al.*, 1998). The accumulation of natural antioxidants in the avian embryonic tissues before hatching may be considered as an adaptive mechanism to protect polyunsaturated fatty acids in membranes from lipid peroxidation during the oxidative stress of the hatching process (Surai, 1999).

When compared with other avian species, the turkey is characterised by possessing unique differences in vitamin E metabolism. For example, it has been shown that turkey eggs contain significantly lower quantities of vitamin E than those of the chicken even though their diets are very similar in vitamin E content (Surai *et al.*, 1998). As a result, tissues of d-old turkey poults, including the liver, contain much lower concentrations of α-tocopherol than those of d-old chickens (Soto-Salanova and Sell, 1996). The turkey excretes 2- to 7-fold more tocopheryl glucuronides (Sklan *et al.*, 1982), explaining, in part, the lower plasma and tissue concentrations of tocopherol observed in turkeys than in chickens.

During posthatch development the vitamin E content of the livers of chickens, turkey poults, ducklings and goslings have been shown to decrease

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dramatically (Surai and Ionov, 1994; Surai *et al.*, 1998; Soto-Salanova *et al.*, 1993; Soto-Salanova and Sell, 1995, 1996). To improve this position different approaches have been used with varying success. These have included extra dietary supplementation of vitamin E (Applegate and Sell, 1996), inclusion of bile salts in the diet (Soto-Salanova *et al.*, 1993), fat manipulation (Soto-Salanova and Sell, 1995) as well as vitamin E injection (Soto-Salanova and Sell, 1996). None of these approaches were able to completely prevent the decrease in the vitamin E concentration in the turkey poult liver during the first 2 to 3 weeks of postnatal development.

Surai *et al.* (1998) considered the metabolism of α-tocopherol in avian embryonic tissues. However, poultry diets contain other tocopherols and tocotrienols (Lynch, 1996) which possess antioxidant properties and may be assimilated and thus have an effect on the efficiency of the antioxidant system (Packer, 1995). The aim of the study was to investigate tissue-specific accumulation of tocopherols and tocotrienols in the turkey tissues during embryonic development and their susceptibility to lipid peroxidation.

MATERIALS AND METHODS

Eggs and embryos

Fertile BUT turkey eggs were incubated using standard commercial conditions (37·8° C and 60% relative humidity). Embryonic tissues were collected after 16, 22 and 25 d of incubation, and from d-old poults (referred to as day 29) and analysed immediately or stored at –20°C under nitrogen for not more than 2 weeks before the analysis of tocopherols and tocotrienols.

Analytical procedures

The determination of tocopherols and tocotrienols in the food and tissues were performed by the method of McMurray *et al.* (1980) with slight modifications. In brief, the samples were saponified with ethanolic potassium hydroxide in the presence of pyrogallol in an atmosphere of nitrogen for 30 min at 70°C; after cooling and diluting with distilled water, tocopherols and tocotrienols were extracted from the mixture with hexane. The extract was dried under nitrogen, redissolved in methanol and injected on to a Spherisorb type S3ODS2 3 μm C18 reverse phase high performance liquid chromatography (HPLC) column, 15 cm×4·6 mm (phase separation, Clwyd, UK). Chromatography was performed using a mobile phase of methanol/water (97:3, v/v) at a flow rate of 1·1 ml/min. Fluorescence detection utilised excitation and emission wavelengths of 295 and 330 nm, respectively. Calibration was performed using standard solutions of α, γ, and δ-tocopherols (Fluka, Gillingham, Dorset, UK) and α-, β-, γ- and δ-tocotrienols (Merck, Germany) in methanol. Because the HPLC system was not able to separate the β- and γ-forms of tocopherols and tocotrienols they were eluted as a single peak. Lipid peroxidation was estimated by spectrophotometric determination of thiobarbituric acid reactive substances (TBARS) as previously described (Surai *et al.*, 1996).

Table 1. *Tocopherols and tocotrienols in turkey diet and egg yolk (μg/g)*

Vitamin E forms	Diet	Egg yolk
α-tocopherol (α-tph)	23·62±1·2	11·4±1·3
β-+γ-tocopherols (β-+γ-tph)	10·35±0·6	4·91±0·26
δ-tocopherol (δ-tph)	1·71±0·1	0·33±0·02
α-tocotrienol (α-ttn)	4·09±0·2	1·15±0·11
β-+γ-tocotrienols (β-+γ-ttn)	17·09±1·1	2·21±0·13

Values are means±SEM of measurements from 5 samples

Statistical analysis

All results are the means±standard error of the mean (SEM) of measurements on 5 samples. Statistical analysis was performed by *t* test and by analysis of variance using the ANOVA procedure.

RESULTS

The tocopherol and tocotrienol compositions of the turkey diet and egg yolk are presented in Table 1. The turkey diet contained the complete range of tocopherols and tocotrienols. The quantity of α-tocopherol was twice that of β-+γ-tocopherols as a result of dietary vitamin E supplementation. The δ-tocopherol content of the diet was 13 times lower than that of α-tocopherol. The diet also contained tocotrienols. For example, the amount of β-+γ-tocotrienols was similar to that of α-tocopherol, the α-tocotrienol concentration in the diet was 7 times lower than that of α-tocopherol. Data presented in Table 1 indicate that the accumulation of α- and β-+γ-tocopherols in the egg yolk was proportional to their content in the diet. On the other hand, tocotrienol transfer from the diet to the egg yolk was much less effective compared to tocopherols.

Table 2 shows the distribution of vitamin E in the embryonic liver. Between days 16 and 22 of embryo development, the α-tocopherol concentration in the liver remained constant and later it increased significantly ($P<0·01$), reaching a maximum value just after hatching. Similar changes were observed for the other tocopherols and tocotrienols. At hatching the α-tocopherol concentration in the liver was 3 times greater compared to β-+γ-tocopherols and 15 times greater than α-tocotrienol.

Vitamin E distribution in the YSM is shown in Table 3. The accumulation of α-tocopherol in the

Table 2. *Vitamin E distribution in the liver (μg/g)*

Days of development	α-tph	β-+γ-tph	δ-tph	α-ttn	β-+γ-ttn
16	13·42±0·62	1·23±0·11	nd	0·33±0·12	0·10±0·01
20	14·99±0·41	2·04±0·14	nd	0·64±0·15	0·11±0·02
22	16·07±1·02	2·98±0·24[a]	nd	0·83±0·06[b]	0·13±0·01
25	39·17±2·22[c]	10·1±0·11[c]	0·19±0·02	2·62±0·03[c]	0·47±0·05[c]
29	95·62±7·44[c]	31·45±2·88[c]	0·44±0·03	5·81±0·61[c]	0·69±0·04[c]

Values are means±SEM of measurements from the liver from 5 embryos or chicks Significance of difference from day 16: [a] $P<0·05$; [b] $P<0·01$; [c] $P<0·001$. nd- not detected

Table 3. *Vitamin E distribution in the yolk sac membrane (μg/g)*

Days of development	α-tph	β-+γ-tph	δ-tph	α-ttn	β-+γ-ttn
16	22·66±7·44	6·10±0·55	0·12±0·04	1·33±0·14	0·14±0·05
20	23·09±1·77	7·49±0·81	0·16±0·01	2·08±0·26	0·29±0·04
22	35·29±3·66[a]	9·20±0·88[a]	0·19±0·03	2·27±0·33	0·50±0·04[b]
25	63·56±5·96[c]	20·56±1·77[c]	0·23±0·02	2·97±0·33[a]	1·59±0·12[c]
29	100·31±8·76[c]	49·47±4·11[c]	0·48±0·04[b]	6·68±0·55[c]	3·50±0·29[c]

Values are means±SEM of measurements from the liver from 5 embryos or chicks Significance of difference from day 16: [a] $P<0·05$; [b] $P<0·01$; [c] $P<0·001$.

YSM started after day 20 of development whereas the increase in tocotrienol concentrations started between days 16 and 20 of development. As in the liver, at hatching the concentration of α-tocopherol in the YSM was twice that of β-+γ-tocopherols and 15 times greater than α-tocotrienol.

In the blood plasma (Figure 1) the α-tocopherol and β-+γ-tocopherol concentrations increased gradually between days 16 and 22 with a rapid increase taking place between days 25 and 29. The tocotrienol content in the embryo blood increased gradually between days 16 and 29 with the most pronounced increase between days 22 and 25.

In the kidney (Figure 2), heart (Figure 3), lung (Figure 4), muscle (Figure 5) and adipose tissues (Figure 6), gradual increases in the tocopherol and tocotrienol concentrations took place between days

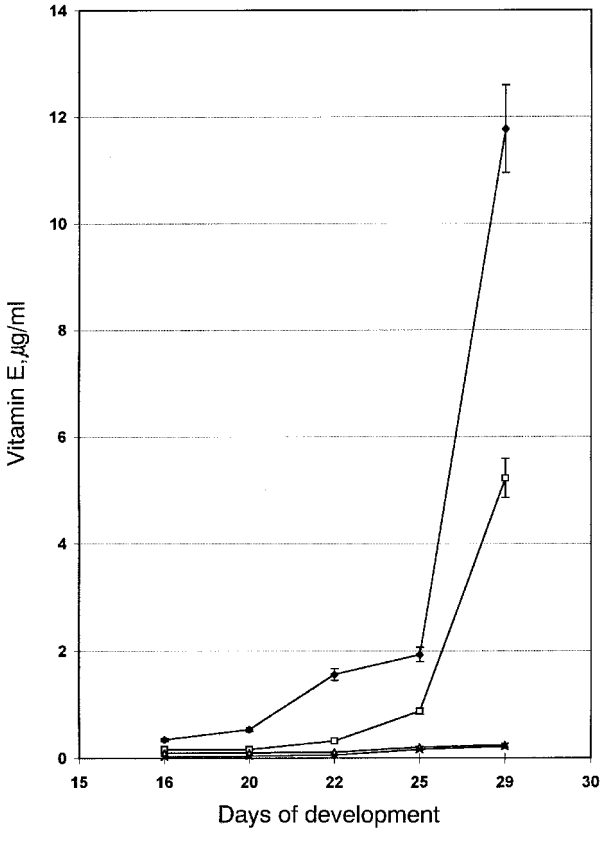

Figure 1. *Vitamin E in blood plasma: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

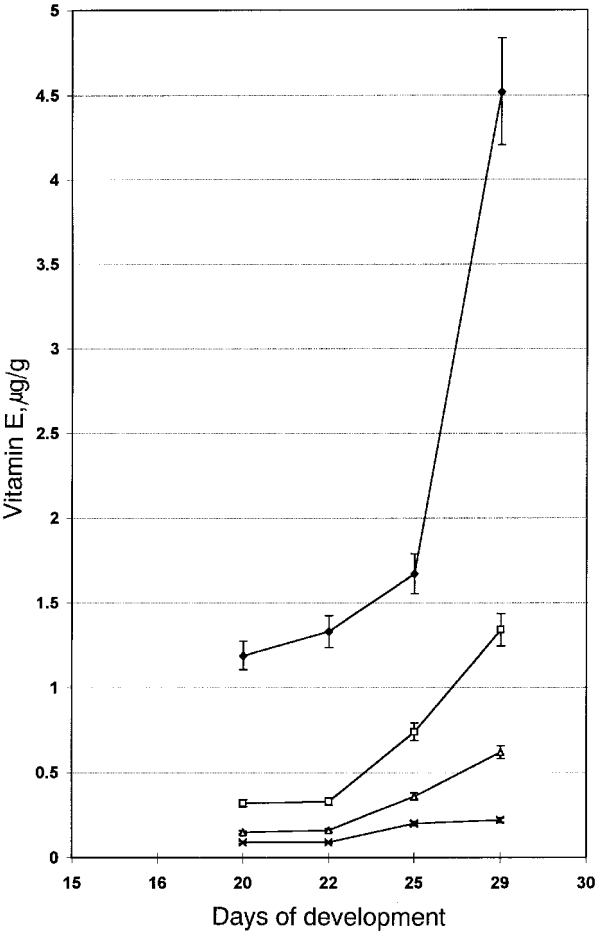

Figure 2. *Vitamin E in the kidney: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

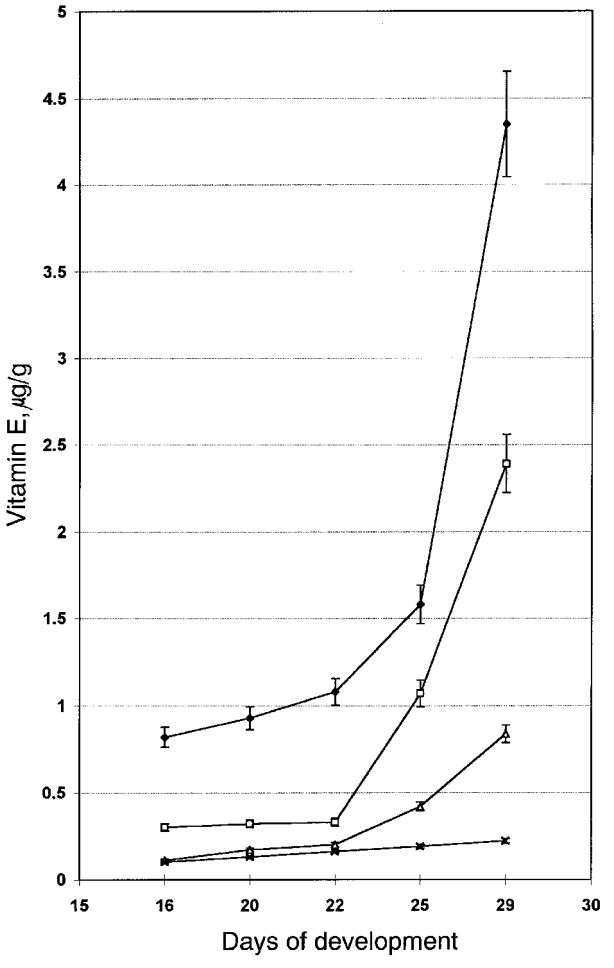

Figure 3. *Vitamin E in the heart: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

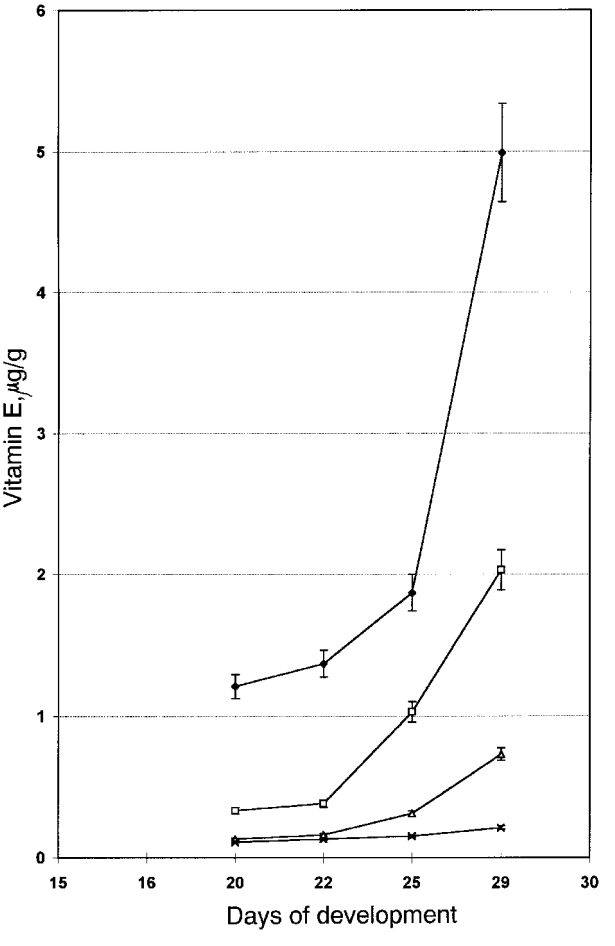

Figure 4. *Vitamin E in the lung: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

20 and 25 of the development with a sharp increase of α-tocopherol, in particular, between days 25 and 29.

The brain differed from the other tissues studied (Figure 7) with relatively low tocopherol concentrations throughout the period of development, an absence of β-+γ-tocotrienols and a very low concentration of α-tocotrienol. In all the tissues studied in the d-old turkey poults, the concentrations of α-tocotrienol were several times greater than those of the β-+γ-tocotrienols. In the blood plasma the concentrations were almost equal.

Tissue-specific features in the susceptibility to lipid peroxidation in the 25-d-old turkey embryo are shown in Figures 8 and 9. As can be seen from these data, most of the tissues had similar susceptibilities to spontaneous lipid peroxidation but TBARS accumulation in the brain was some 8 to 9 times greater than in other tissues. Adipose tissue was also characterised by an increased susceptibility to spontaneous lipid peroxidation. The inclusion of Fe^{2+} in the incubation medium significantly increased TBARS production in all the tissues studied. With respect to TBARS accumulation, as a result of Fe^{2+}-stimulated lipid peroxidation, the tissues can be placed in the following descending order: brain=heart=kidney>muscle>adipose>lung> YSM=liver.

In the d-old poult, the susceptibility of most tissues to lipid peroxidation decreased (Figures 8 and 9) compared with that seen in tissues from 25-d-old embryos. Nevertheless, in the brain of the d-old poults TBARS accumulation, as a result of spontaneous lipid peroxidation, was similar to that in the embryo at day 25 of development. The susceptibility of the tissues to the Fe^{2+}-stimulated lipid peroxidation decreased as well. Again, in the brain, TBARS accumulation as a result of Fe^{2+}-stimulated lipid peroxidation, was highest and did not change compared to that of the embryo at day 25 of development.

DISCUSSION

Tocopherol and tocotrienol accumulation in the embryonic tissues

The diet contained a range of tocopherols and tocotrienols, the concentration of β-+γ-tocotrienols being similar to that of α-tocopherol. The quantity of tocotrienols in the diet depends on the extent of grain processing before its inclusion into the diet because tocotrienols are predominantly located in

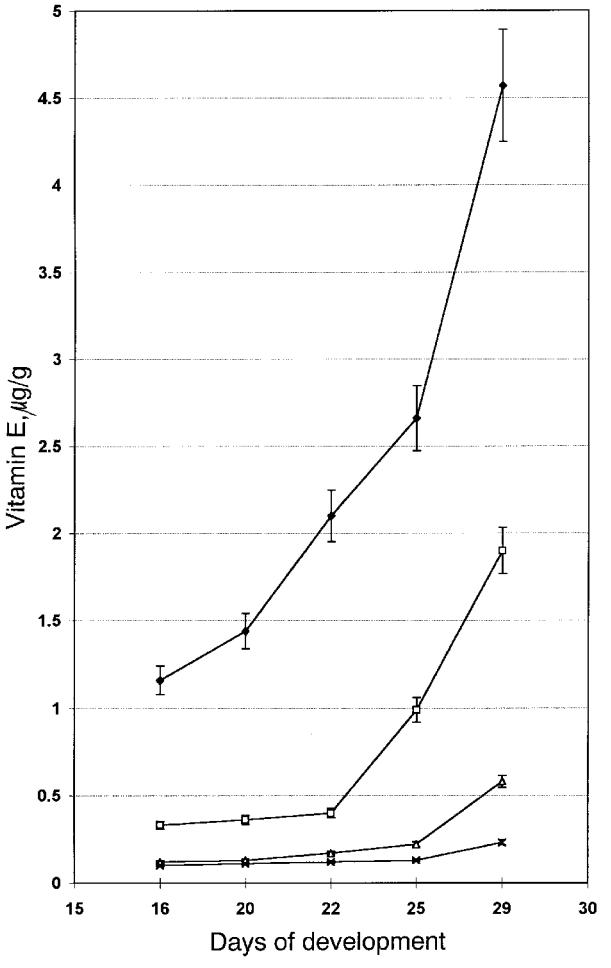

Figure 5. *Vitamin E in the muscle: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

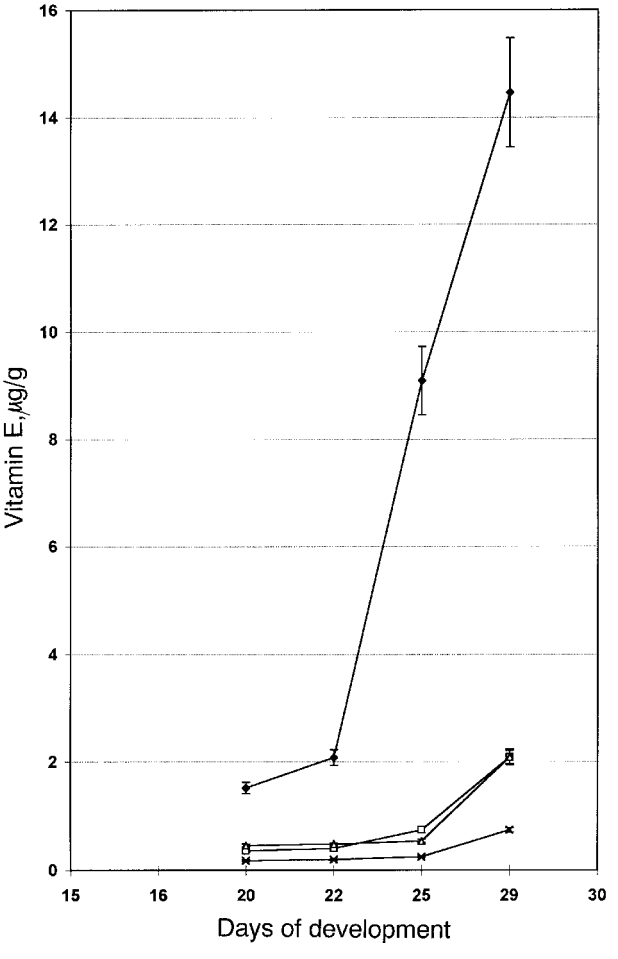

Figure 6. *Vitamin E in the adipose tissue: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

the endosperm, whereas tocopherols are concentrated in the germ (Peterson and Wood, 1997).

When compared with the other avian species, the turkey accumulates relatively small amounts of tocopherols in its tissues (Soto-Salanova *et al.*, 1993; Soto-Salanova and Sell, 1995, 1996) and egg yolk (Surai *et al.*, 1998). During early postnatal life the concentration of tocopherols in the turkey liver have been observed to decrease sharply (Soto-Salanova and Sell, 1995; Surai *et al.*, 1998). Marusich *et al.* (1975) suggested that the low hepatic vitamin E concentrations in turkey poults was the result of inefficient intestinal absorption of vitamin E; this could be a result of low pancreatic lipase activity (Sell *et al.*, 1991) as well as greater (compared to chicken) production and excretion of tocopheryl glucuronides (Sklan *et al.*, 1982).

The tocopherol accumulation in the embryonic liver started after day 22 of embryo development, that is simultaneously with lipid absorption from the residual yolk (Ding *et al.*, 1995). In the YSM this process started earlier, meaning that, as in the chicken (Surai *et al.*, 1996) the YSM absorbs tocopherols from the residual yolk and then transfers them to the embryonic liver. Similarly, in the other tissues α-tocopherol accumulation takes place during the last week of embryonic development. The vitamin E content of the embryonic brain was much lower than that in the other tissues studied, a finding consistent with previous observations with chickens (Surai *et al.*, 1996). In general, the hepatic vitamin E concentration in d-old turkeys was 1·5 times lower when compared with previous observations (Surai *et al.*, 1998), but greater than values reported by Soto-Salanova *et al.* (1993) and Soto-Salanova and Sell (1995), probably reflecting differences in the amounts of this vitamin in the diet and egg yolk.

The tocotrienol concentrations in egg yolk and turkey embryonic tissues are reported in the present work for the first time. The greatest tocotrienol concentrations were found in the liver, YSM and adipose tissue. The importance of this finding lies in the antioxidant properties of the tocotrienols (Packer, 1995). In spite of the low concentration of tocotrienols in embryonic tissues, they can play a role in the antioxidant system of the embryo. In phosphatidylcholine liposomes, α-tocotrienol exhibited significantly greater peroxyl radical potency than α-tocopherol (Suzuki *et al.*, 1993) and there is a growing body of evidence that a combination of tocopherols and tocotrienols has a higher protective effect than tocopherols alone (Serbinova *et al.*, 1992). The greater antioxidant potency of

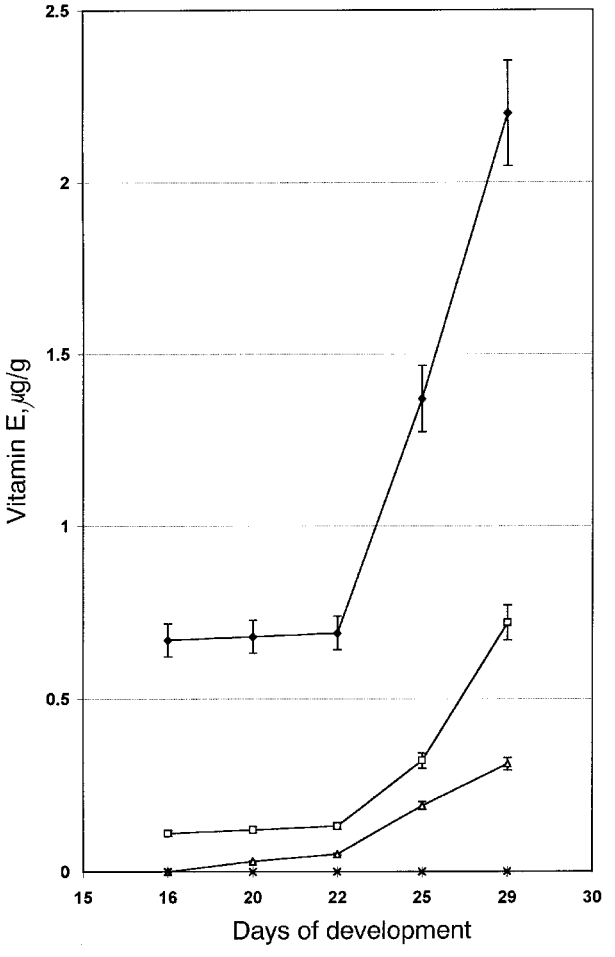

Figure 7. *Vitamin E in the brain: (◇)-α-tocopherol, (□)-β-+γ-tocopherols, (□)-α-tocotrienol, (□)-β-+γ-tocotrienols. Mean values±SEM for 5 samples*

α-tocotrienol compared to α-tocopherol in membranes is a consequence of the combined effects of 3 properties: higher recycling efficiency from chromanoxyl radicals; more uniform distribution in membrane bilayer and stronger disordering of membrane lipids, making interaction of chromanols with lipid radicals more efficient (Serbinova *et al.*, 1991).

The confirmation of the possible protective effect of tocotrienols in the liver and YSM of d-old poults comes from the comparison between the concentrations of tocotrienols in these tissues and the α-tocopherol content of the peripheral tissues. In poults the α-tocotrienol concentrations in liver and YSM were similar to those of α-tocopherol in most of the tissues studied and more than twice the brain α-tocopherol concentration. Previous observations indicate that a low vitamin E content in the embryonic chicken brain is able to protect this tissue against lipid peroxidation in physiological conditions (Surai *et al.*, 1996) as a result of its efficient recycling. In this respect preferential accumulation of α-tocotrienol in the tissues may be of importance.

Tocopherol and tocotrienol discrimination

The results indicate that the transfer of α- and β-+γ-tocopherols from the diet to the egg yolk is proportional to their concentrations. In contrast the assimilation of δ-tocopherol and tocotrienol from the diet was much lower compared to that of α-tocopherol. At the same time the efficiency of the transfer of α-tocotrienol was twice that for other tocotrienols, in agreement with Ikeda *et al.* (1996)

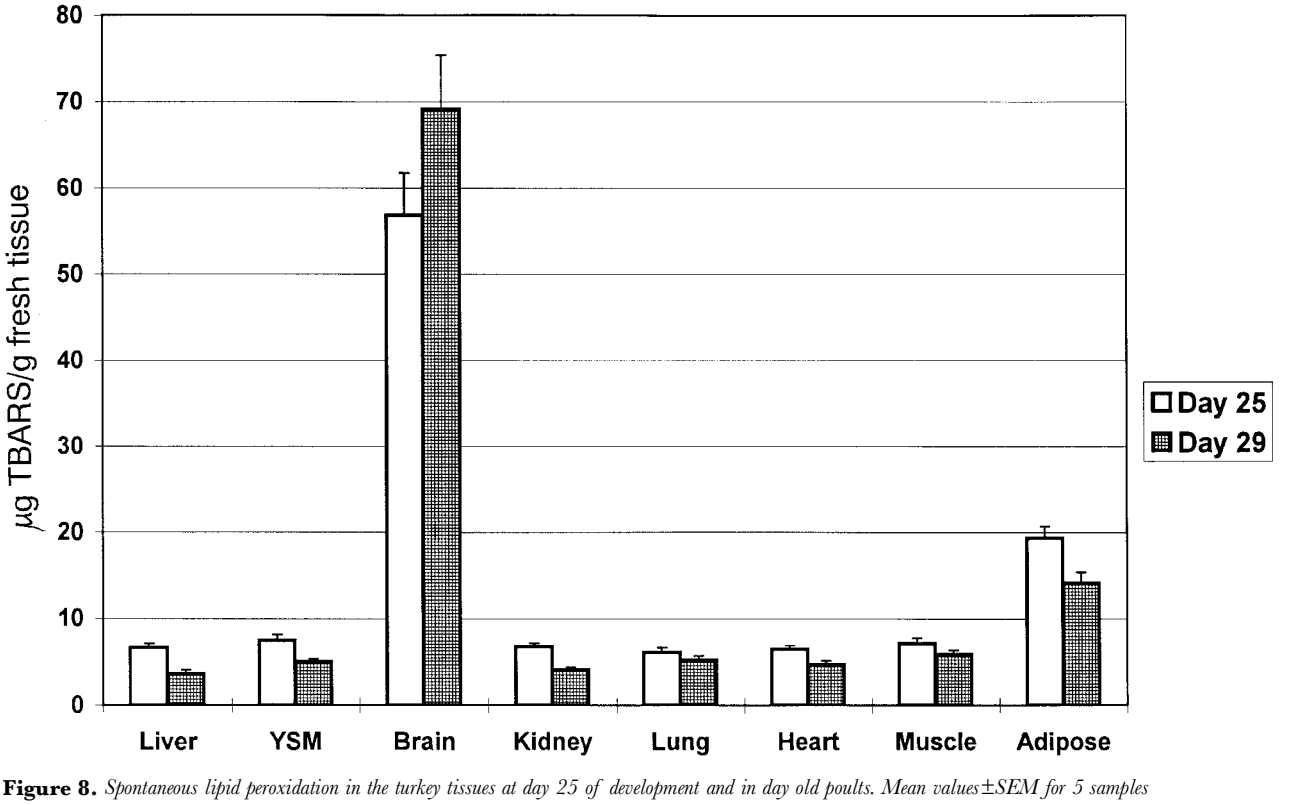

Figure 8. *Spontaneous lipid peroxidation in the turkey tissues at day 25 of development and in day old poults. Mean values±SEM for 5 samples*

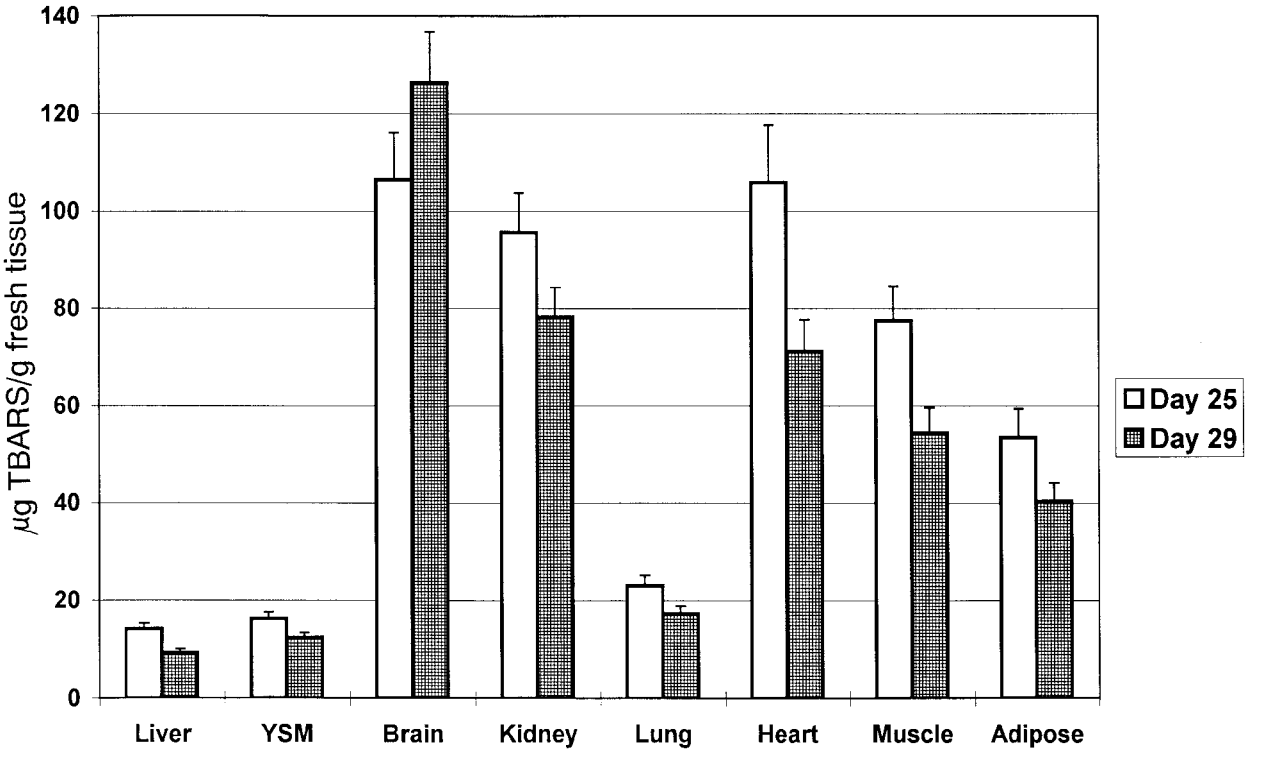

Figure 9. *Fe-stimulated lipid peroxidation in the turkey tissues at day 25 of development and in day old poults. Mean values±SEM for 5 samples*

who showed a preferential absorption of α-tocotrienol compared to γ- and δ-tocotrienols.

A 2nd aspect of discrimination between different forms of vitamin E is their transfer and distribution from the yolk to the developing tissues. The concentration of β-+γ-tocotrienols in the egg yolk was almost twice that of α-tocotrienol, but in the liver of a d-old turkey poult the concentration of α-tocotrienol was 9 times greater than β-+γ-tocotrienols. Similarly, in the egg yolk the concentrations of α-tocotrienol was 3·5 times greater than that of δ-tocopherol, but in the liver of the d-old poults this difference was much higher (13·2 fold).

It is interesting to underline that in the YSM the discrimination between α- and other tocotrienols was less obvious, probably reflecting the function of the organ in absorption, transport and distribution of nutrients. In all other organs the α-tocotrienol contents were several times greater than the other tocotrienols. Possibly the liver preferentially releases α-tocotrienol into the blood stream incorporated into very low density lipoproteins (VLDL); as a result the other tissues preferentially obtain this form of tocotrienol. On the other hand, the discrimination between α- and β-+γ-tocopherols in the turkey embryonic tissues was not very pronounced, although there was some in favour of α-tocopherol in most tissues in the early stages of the embryonic development, similar to that found in adult mammals as a result of the presence of tocopherol-binding protein (TBP) in the liver (Traber and Sies, 1996).

The discrimination between different tocotrienols may be explained in the following 2 ways: firstly, the embryonic liver TBP which takes part in the preferential incorporation of α-tocopherol into the VLDL, has a different affinity to different tocotrienols. For example, it has recently been shown that the comparative affinity of α-tocotrienol to TBP is 12% that of α-tocopherol (Hosomi *et al.*, 1997); possibly other tocotrienols have even lower affinities to TBP. Secondly, because of differences in the structure of various tocotrienols their affinities for VLDL may differ and as a result they are incorporated in the lipoproteins with different efficiencies. For example, in hamsters tocopherols were found primarily in low density lipoprotein and high density lipoprotein in association with plasma surface components, whereas tocotrienols disappeared from plasma with chylomicron clearance (Hayes *et al.*, 1993) and the rate of this disappearance probably may distinguish between different tocotrienols.

Vitamin E accumulation and tissue-specific susceptibility to lipid peroxidation

The accumulation of vitamin E in the turkey embryonic tissues reached a maximum just after hatching, as in the chicken (Surai *et al.*, 1996). During the last days of development the susceptibility of tissues to lipid peroxidation decreased as well. It is necessary to emphasise that during the same period of development the unsaturation of lipids of embryonic tissues remained high with only a slight decrease in the arachidonic acid content (Ding and Lilburn, 1997). Thus, an accumulation of this most effective natural antioxidant simultaneously with other antioxidants such as carotenoids (Surai *et al.*, 1998) is considered a protective mechanism against lipid peroxidation during the stress conditions of

hatching (Surai, 1998). The brain is an exception to this rule. The low concentration of α-tocopherol in the brain may make young poults susceptible to environmental stress. It is especially important because during postnatal development of the turkey the brain is characterised in having low vitamin E concentrations and being relatively insensitive to dietary supplementation (Wen *et al.*, 1997). Similarly, a 4-fold increase in the amount of vitamin E in the yolk did not change the α-tocopherol concentration in the brain of hatched chicks (Cherian and Sim, 1997). This contrasts with the chicken where supplementation of the diet with α-tocopherol (250 mg/kg) caused a significant increase in vitamin E concentrations in the egg yolk and brain (Surai *et al.*, 1997) and resulted in a decreased susceptibility of the tissue to lipid peroxidation.

Vitamin E deficiency in the young chicken is associated with the development of encephalomalacia, a disease with oxidative deterioration to the cerebellum (Dror *et al.*, 1976; Furhmann and Sallmann, 1995). Jortner *et al.* (1985) also observed clinically confirmed encephalomalacia in turkey poults fed on a diet without vitamin E. There have also been reports of the appearance of cerebral encephalomalacia in commercial turkey poults, with no clear indications of the cause of the disease (Ficken *et al.*, 1993). Malabsorption of vitamin E, secondary to rotavirus infection was considered to be 1 of the causes (Frank and Bergeland, 1988).

Most of the tissues studied in embryos at day 25 showed similar susceptibility to spontaneous lipid peroxidation. The 2-fold increase in the TBARS accumulation in adipose tissue presumably reflected an increase in the lipid content of the incubation medium. The brain was characterised by having an exceptionally high susceptibility to lipid peroxidation, consistent with low vitamin E concentrations (Fig. 7) and high lipid unsaturation in this tissue (Applegate and Sell, 1996) similar to that found in chickens (Surai *et al.*, 1996). From all tissues studied, the liver and YSM were the most resistant to Fe^{2+}-stimulated lipid peroxidation, in agreement with the high vitamin E content found in these tissues.

Between day 25 of development and 1 day posthatch the susceptibility of the majority of the tissues to lipid peroxidation significantly decreased. Thus, the vitamin E accumulation, which takes place during this period in the tissues, serves as a protective mechanism against lipid peroxidation at hatching. Nevertheless, the tissues of d-old turkey poults showed comparatively high susceptibility to Fe^{2+}-stimulated lipid peroxidation with the liver being the most resistant to TBARS accumulation. In this respect, it is interesting to mention a high rate of peroxidation in livers of d-old poults observed by Soto-Salanova and Sell (1996).

During the same period of development a decrease in the susceptibility to lipid peroxidation was less pronounced for the lung and was not the case for the brain. In fact, there was a tendency for increasing brain susceptibility to peroxidation between days 25 and 29. Thus, high brain susceptibility to lipid peroxidation did not decrease as a result of an increase in vitamin E concentration (which still remained very low) during this period of the development.

The susceptibility to lipid peroxidation depends on the balance between antioxidants (vitamins A, E, C, carotenoids, glutathione, antioxidant enzymes) and pro-oxidants (Fe, Cu, polyunsaturated fatty acids etc.) in the tissue. It was suggested that during embryogenesis antioxidant compounds (vitamins E, C, glutathione and carotenoids) play the crucial role in antioxidant protection and in postnatal development antioxidant enzymes play the more important role (Surai, 1999). In this respect turkey tissues are characterised by having lower (compared to chickens) vitamin E concentrations, but containing less polyunsaturated fatty acids (Noble and Cocchi, 1990). As for other antioxidants/pro-oxidants there is a lack of information to allow a direct comparison to be made and species-specific differences in the tissue susceptibility to lipid peroxidation awaits further investigation.

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