Response of Whole Blood, Erythrocyte and Plasma Vitamin E Content to Dietary Vitamin E Intake in the Chick

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Hassan, S. and J. Hakkarainen: Response of whole blood, erythrocyte and plasma vitamin E content to dietary vitamin E intake in the chick. Acta vet. scand. 1990, 31, 399–408. – Whole blood, red blood cells (RBC), and plasma vitamin E (VE) levels in chicks fed dietary VE (dl-α-tocopheryl acetate, dl-αTa) supplementation in steps of 0.0, 5.0, 10.0, 15.0, 20.0 and 30.0 mg/Kg were determined to examine their usefulness as an index of VE status. The increase in VE level was significant and linear in whole blood (r = 0.90), RBC (r = 0.89) and plasma (r = 0.93) in response to dietary VE intake. There was a close correlation between VE in plasma *vs* whole blood (r = 0.90), plasma *vs* RBC (r = 0.91) and whole blood *vs* RBC (r = 0.95). The plasma VE content was 1.2–1.8 times greater than that of whole blood, and 6.6–12.5 times greater than that of RBC. The plasma total lipids content was not affected by the dietary VE intake, whereas the level of VE in the plasma total lipids was significantly increased with increasing supplementation. Alpha tocopherol was the major isomer (ca 92 %) of VE in whole blood, RBC and plasma at hatching. The small proportions of β-tocopherol (ca 2 %), γ-tocopherol (ca 5 %) and α-tocotrienol (ca 1 %) observed at 1 day of age had decreased or totally disappeared by 7 days of age after feeding the VE-free basal diet. The data showed that in the chick, the whole blood and RBC levels of VE were as sensitive and reliable indexes of dietary VE status as was that of the plasma.

α-tocopherol; β-tocopherol; γ-tocopherol; dl-α-tocopheryl acetate; plasma total lipids.

Introduction

Vitamin E (VE) and selenium are well-recognized as being important components of the cell membrane defense system against peroxidative damage (*Hoekstra* 1975). *Noguchi et al.* (1973) reported that the plasma membrane in chicks was the site of the biological action of VE, where it neutralizes free radicals, preventing the development of exudative diathesis. Red blood cell (RBC) membranes deficient in VE are susceptible to hemolysis, and maintenance of the integrity of the RBC membrane partially depends on an adequate VE status (*Rose & Gyorgy* 1950).

Vitamin E is widely distributed in human and animal tissues, with appreciable amounts stored in the liver (*Gallo-Torres* 1980). The assessment of VE status in different species may be determined by the direct measurement of the VE content in a specific tissue, which is then assumed to reflect the whole body VE status. Early studies with mammals used the plasma and liver VE contents as an index. However, the validity of using the plasma VE content was

questioned by *Horwitt et al.* (1972), who
found that it increased in response to an in-
crease in the level of the plasma lipids.
These workers concluded that VE/total li-
pids ratio in plasma would be the best index
of the vitamin status. Recently, *Vatassary et
al.* (1983) suggested that the level of VE in
the platelet was a reliable index of VE status
in human. In rats, the use of RBC as indica-
tor of the VE status was supported by *Mino
et al.* (1985). However, *Lehmann* (1984)
found that VE level in the platelets, but not
in the RBC, was the best index of VE status
of these animals.

With respect to avians, primarily the chick,
the plasma and liver VE levels have been
used as indicators of VE biopotency and sta-
tus (*Pudelkiewicz et al.* 1960, *Scott & Desai*
1964, *Hakkarainen et al.* 1984). A linear
increase in plasma and liver VE levels in
chicks in response to dietary supplementa-
tion was observed by *Hakkarainen et al.*
(1984). However, studies on the comparative
responses of whole blood, RBC and plasma
to dietary VE intake in the chick are lacking.
Apparently, less attention has been focused
on the whole blood and RBC content of VE
in various species due to technical difficul-
ties with the assay. However, recent progress
in VE analyses employing high performance
liquid chromatography (HPLC) have facili-
tated the accurate determination of VE con-
tent in RBC.

The present study was undertaken in order
to compare the validity of using whole
blood, RBC and plasma VE concentrations
in the chick as an index of dietary VE in-
take. The chicks were fed graded levels of
VE as dl-α-tocopheryl acetate (dl-αTa),
while the dietary fat was kept constant.
HPLC was used for the VE assay, and a
method for the analyses of whole blood and
erythrocyte VE in the chick is described.

Material and methods

Commercial White Leghorn male chicks
were used for the present study. At hatching,
6 chicks were immediately selected at ran-
dom. Whole blood samples were obtained
by cardiac puncture and they were then sa-
crificed. For 1 week after hatching, 19
groups of 10 chicks each were fed a VE-free
basal diet containing all other nutrients re-
quired by the chick. This diet consisted of
casein, 9.5 %; gelatin, 11.5 %; skim-milk
powder, 16.5 %; cellulose, 3.0 %; glucose,
44.94 %; distilled fatty acids obtained from
soybean oil, 3.0 %; amino acid mixture,
0.96 %; mineral mixture, 5.6 % and vitamin
mixture, 5.0 %. For descriptions of the ami-
no acid, mineral and vitamin mixtures, see
Hassan et al. (1987). Adequate amounts of
selenium (as sodium selenite) and ethoxy-
quin were included in the basal diet to pro-
tect the chicks against VE and/or Se defici-
ency. At 7 days of age, whole blood was
again collected from 6 chicks in 1 group,
and the remainder of the chicks in this
group were destroyed. The VE depletion
period was followed by a 6 week repletion
period, during which the basal diet was
supplemented with 0.0, 5.0, 10.0, 15.0, 20.0
or 30.0 mg VE as dl-αTa/kg (Vitamin E dry
powder, 50 %, Rovimix E-50 SE, F. Hoff-
mann-La Roche).

Each dietary treatment and water were pro-
vided to 3 groups of chicks ad lib. The
chicks were housed in electrically heated
wire batteries, and were wing banded. The
relative humidity was ca 65 % and artificial
light was provided 24 h/day. At the end of
the study, 6 chicks per treatment (2 per
group) were selected at random for whole
blood sampling.

Heparinized tubes were used to obtain
whole blood by cardiac puncture from
chicks killed at 1, 7 and 49 days of age. Each

sample was divided into 2 portions. One portion was immediately stored at −70°C, and the other was centrifuged at 2500 × g for 10 min to obtain plasma. The packed RBC were washed 3 times with buffer sulphate (pH 7.4). After the last washing, care was taken to obtain exactly the same volume of the whole blood noted prior to storage.

The determination of VE in whole blood and RBC was carried out using a modification of the method reported by *Stump et al.* (1984). Two ml ice cold methanol containing 1 % pyrogallol was added slowly to 1 ml RBC suspension or whole blood while carefully mixing (Vortex blender). Tocopherol was then extracted with a solution of 4 ml hexane containing 0.1 % butylated hydroxytoluene (BHT). Three ml of the hexane phase was evaporated under nitrogen gas flow at 30–35°C. The residue was then dissolved in 150 µl of heptane, and 20 µl were used for the manual loop injection. For plasma VE analysis, protein precipitation and extraction were done as reported by *Hakkarainen et al.* (1984). Mean recoveries of 8 analysed samples of whole blood or RBC and 4 of plasma, were 98, 97 and 97 %, respectively, while the corresponding values for the coefficient of variation (CV) were 2.8, 1,6 and 1.1 %, respectively. The solution was injected into a Merck Hibar 125-4 (5 µm) silica column held thermostatically at 37°C. The mobile phase contained 6 % ether in heptane. The flow rate during elution was 1.3 ml/min, and the retention time for α-tocopherol (α-T), α-tocotrienol (α-T3), β-tocopherol (β-T) and γ-tocopherol (γ-T) were 5.3, 7.1, 8.6 and 10.2 min, respectively. The Perkin-Elmer HPLC system consisted of a model PE Series 10 pump module, fitted with a 20 µl sample loop. Plasma total lipids content was analysed according to *Epstein et al.* (1972).

Statistical analyses of the data were performed by linear regression (*Finny* 1978) and analyses of variance (*Dunn & Clark* 1974) using the GLM procedure of the SAS computer programs (Statistical Analyses System 1985).

Results

The initial VEs level in the whole blood, RBC and plasma of chicks at hatching were 48.06 ± 7.12, 8.17 ± 1.13 and 62.48 ± 3.65 mg/ml, respectively. Feeding the VE-free basal diet without supplementation to chicks for the first week posthatching significantly ($p < 0.01$) reduced the 1 day of age VE levels to 9.15 ± 2.43, 0.17 ± 0.05 and 12.54 ± 1.75 mg/ml, respectively. The effect of dietary VE supplementation as dl-αTa for 6 weeks on VE levels in whole blood and blood components, and on plasma total lipids content and their VE concentrations is presented in Table 1. Observations at the end of the repletion period showed significantly ($p < 0.01$) lower levels of VE in whole blood, RBC and plasma of chicks without VE supplementation in comparison to those found at 7 days of age. Supplementation of 5.0 or more mg VE (dl-αTa)/Kg diet led to significant ($p < 0.05$) increases in whole blood, RBC and plasma levels of VE in comparison to those in the control chicks. With each increment in dietary VE supplementation, there was a further slight or significant ($p < 0.05$) increase in the VE levels of the whole blood and blood components. At each supplemental level, slightly or significantly ($p < 0.05$) higher concentrations of VE were found in the plasma in comparison to those in the whole blood, whereas the levels of VE in RBC were constantly lower ($p < 0.05$) than those of whole blood and plasma.

The VE content of the plasma total lipids decreased significantly ($p < 0.01$) from 18.10 ± 1.74 at hatching to 3.11 ± 0.25 mg/g

S. Hassan & J. Hakkarainen

Table 1. Effect of dietary vitamin E supplementation on the vitamin E level in whole blood, erythrocytes (RBC), plasma and plasma lipids, and on the plasma content of total lipids in the chick[1].

Diet	Dietary VE supplement mg/kg[2]	Vitamin E content				
		Whole blood mg/ml	RBC mg/ml	Plasma mg/ml	Plasma lipids	
					mg/g	g/l
1	0.00	0.48 ± 0.14[a,3,4]	0.07 ± 0.02[a]	0.78 ± 0.18[a]	0.16 ± 0.03[a]	4.36 ± 0.24[n.s.]
2	5.00	1.11 ± 0.09[b]	0.17 ± 0.04[b]	1.43 ± 0.11[b]	0.35 ± 0.02[b]	4.19 ± 0.27
3	10.00	1.48 ± 0.15[b]	0.39 ± 0.09[c]	2.61 ± 0.32[c]	0.69 ± 0.07[c]	4.15 ± 0.28
4	15.00	3.36 ± 0.54[c]	0.49 ± 0.06[d]	3.98 ± 0.37[d]	1.12 ± 0.08[d]	4.06 ± 0.38
5	20.00	3.96 ± 0.50[d]	0.67 ± 0.08[d]	4.81 ± 0.57[e]	1.26 ± 0.14[d]	4.09 ± 0.23
6	30.00	5.05 ± 0.53[e]	0.83 ± 0.14[f]	6.33 ± 0.88[f]	1.65 ± 0.23[e]	4.13 ± 0.31

[1] Prior to a repletion period of 6 weeks, the chicks were depleted of VE for 1 week. [2] Supplemented as dl-αTa. [3] Means± SD of 6 observations per treatment. [4] Means within a column with different lower case superscripts are significantly different (with at least $p < 0.05$). [n.s.] The effect of the treatments was not significant ($p > 0.05$).

lipids at 7 days of age in response to VE depletion. As shown in Table 1, there was no effect ($p > 0.05$) of dietary VE supplementation on the plasma total lipids content at 7 weeks of age. However, the VE level in the plasma total lipids increased numerically or significantly ($p < 0.05$) with each increment in VE supplementation.

Over the present VE supplemental range (5.0–30.0 mg/Kg) there was a linear increase in the VE content of the whole blood, RBC, plasma, and plasma lipids vs dietary VE intake (Table 2). In addition, a strong positive correlation between the VE levels in plasma vs whole blood, plasma vs RBC and whole blood vs RBC were observed (Table 2). Calculations of the ratios of VE levels in plasma vs whole blood and plasma vs RBC (unpresented data), showed clear trend for lower ratios with increasing supplementation.

The per cent distribution of the VE isomers in whole blood, RBC and plasma at 1 and 7 days of age is given in Table 3. At hatching, α-T comprised 92 % of the total VE content, γ-T was ca 5 % while β-T and α-T3 were observed in lesser amounts (ca 2.8 %). After

Table 2. Relationship between vitamin E levels in the whole blood, erythrocytes, plasma and plasma lipids vs dietary vitamin E supplement, and between vitamin E level in plasma vs whole blood, plasma vs erythrocytes and whole blood vs erythrocytes in chicks at 7 weeks of age.

Response variable (Y)	Exposure variable (X)	Linear regression equation	
Whole blood VE content	Dietary VE content	$Y = 0.39 + 0.16\,X, r = 0.90$	$p < 0.001$
Erythrocyte VE content	Dietary VE content	$Y = 0.08 + 0.03\,X, r = 0.89$	$p < 0.001$
Plasma VE content	Dietary VE content	$Y = 0.73 + 0.19\,X, r = 0.93$	$p < 0.001$
Plasma lipids VE content	Dietary VE content	$Y = 0.18 + 0.05\,X, r = 0.92$	$p < 0.001$
Plasma VE content	Whole blood VE content	$Y = 0.14 + 0.82\,X, r = 0.90$	$p < 0.001$
Plasma VE content	Erythrocyte VE content	$Y = 0.03 + 0.12\,X, r = 0.91$	$p < 0.001$
Whole blood VE content	Erythrocyte VE content	$Y = 0.03 + 0.15\,X, r = 0.95$	$p < 0.001$

Table 3. The relative per cent distribution of the tocopherol and tocotrienol isomers in the whole blood, erythrocyte (RBC) and plasma of chicks at hatching and at 7 days of age[1].

Tissue site	Age (days)	Relative percentage of isomer distribution (%)[2]			
		α-T	β-T	γ-T	α-T3
Whole blood	1	91.82 ± 0.76[3]	1.94 ± 0.26	5.11 ± 0.85	0.84 ± 0.13
RBC	1	92.45 ± 0.21	1.93 ± 0.14	4.96 ± 0.17	0.71 ± 0.10
Plasma	1	92.75 ± 0.24	1.87 ± 0.06	4.59 ± 0.21	0.79 ± 0.09
Whole blood	7	99.01 ± 0.30	–	0.99 ± 0.29	–
RBC	7	100.00 ± 0.00	–	–	–
Plasma	7	99.18 ± 0.57	–	0.82 ± 0.36	–

[1] Chicks were fed a VE-free basal diet for 7 days posthatching. [2] α-T: α-tocopherol, β-T: β-tocopherol, γ-T: γ-tocopherol, α-T3: α-tocotrienol. [3] Mean ± SD of 6 observations.

the depletion period, none of β-T and α-T3 isomers could be detected at 7 days of age, only trace amounts of γ-T were found in the plasma and whole blood, while the proportion of α-T had increased (≥ 99 %). By the end of this study, α-T was the sole isomer that could be detected in the various chick groups.

Discussion

The high VE content in the plasma and RBC of newly hatched chicks indicated that there had been an efficient carry over of VE from the mothers to their progeny. Immediate VE depletion led to a rapid decrease in the VE concentration by 7 days of age. The percentage of the VE levels observed at hatching remaining after the depletion period were in whole blood, 19 %; RBC, 2 %; plasma 20 % and plasma lipids, 17 %. Thus, the loss in the RBC content of VE was proportionally more than that in the plasma. *Mino et al.* (1981) reported a similar pattern of decrease in plasma and RBC levels of VE in VE depleted rats. The present rapid deprivation of the VE reserves agreed with previous studies and may explain the observed sus-

ceptibility of growing chicks to VE deficiency diseases at 2–3 weeks of age when fed a VE low diet after hatching (*Hakkarainen et al.* 1984, *Hassan et al.* 1985). Therefore, the present data showed that dietary VE supplementation was essential for chicks after hatching, regardless of their tissue reserves of VE at 1 day of age.

The continuous increase in VE levels observed in the 2 blood components with each increment in supplemental VE suggested that the plasma and RBC were sensitive to changes in the VE intake, and that their storage capacity was not saturated by the present supplemental range. This agreed with previous observations in rats (*Lehmann* 1981) and chicks (*Hakkarainen et al.* 1984) fed similar nutritional VE levels. The present findings that the plasma VE level increased linearly when chicks were fed graded nutritional levels of dl-αTa confirmed the findings of *Pudelkiewicz et al.* (1960) and *Hakkarainen et al.* (1984) that the concentrations of VE in the plasma and liver showed a close relationship to the dietary natural and synthetic VE intake and its biopotency in the prevention of nutritional encephalomalacia in the chick.

The assessment of VE status in animals may be based on the measurement of VE in a certain tissue (Gallo-Torres 1980), the erythrocyte hemolyses test (Horwitt et al. 1972), or the observation of clinical signs and pathological lesions of VE-deficiency specific disease (Scott & Desai 1964, Hakkarainen et al. 1984). The present low concentrations of VE observed in the plasma and RBC of the unsupplemented chicks indicated that there had been an efficient mobilization of VE in both blood components during depletion. This is in accordance with observations in chicks fed encephalomalacia inducing diets (Hakkarainen et al. 1984) and rats maintained on a low VE and selenium basal diet (Hakkarainen et al. 1986).

In absolute values, the plasma showed the highest level of VE, irrespectively of the supplemental level. It was 1.2–1.8 times greater than that in the whole blood and 6.6–12.5 times higher than that in RBC. Equivalent VE contents in the plasma and RBC have been reported for rats (Poukka & Bieri 1970) and a higher level of VE in the RBC than in the plasma was observed for rabbits (Kaludin 1963). Bieri et al. (1977) and Mino et al. (1985) noted that the RBC concentration of VE decreased with an increase in the plasma lipids contents in rats and children, respectively. Apparently, species differences and differences in the VE supplemental ranges may account for the discrepancies in the results.

The presence of α-T, β-T, γ-T and α-T3 isomers in the plasma and RBC of newly hatched chicks reflected the isomer composition of the diet fed to their dams, which was obviously transferred to the chicks. The observed isomers were proportionally equally distributed between the plasma and RBC. In the human, Mino et al (1979) found only α-T in the RBC while both α-T and γ-T were observed in the plasma. Alpha toco-

pherol was shown in the present study to be the major isomer in both plasma and RBC, accounting for ca 93 % of the isomers at hatching and ≥ 99 % at 1 and 7 weeks of age. Similar observations were made in the plasma and liver of chicks even though the dietary content of the non-α-T isomers was ca 60 % of the total dietary VE (Hakkarainen et al. 1984). It has been reported that VE depletion causes a rapid and drastic loss of the various isomers in the tissue of chicks (Hakkarainen et al. 1984) and minks (Työpponen et al. 1984), which agreed with the partial or total disappearance of α-T, β-T, γ-T and α-T3 observed in the chicks at 7 days of age in the present study. In addition, the same authors suggested that increasing the dietary intake of dl-αTa will lead to an increase in the α-T concentration with a decline in the levels of non-α-T isomers in animal tissue, probably due to a dilution effect of the increased tissue VE concentration of α-T. This may partially explain the total disappearance of all isomers, with the exception of α-T in the RBC and the plasma, by the end of the present investigation. Peake et al. (1972) found that VE deprived rats lost tissue γ-T more rapidly than α-T. Thus, low concentrations of the non-α-T isomers in animal tissues may reflect a high rate of turnover of these isomers into α-T.

The present investigation demonstrated that there was a close relationship between the VE in plasma vs whole blood, plasma vs RBC, and whole blood vs RBC. Similar findings were reported in rats (Ishibashi et al. 1977), while Cunningham & Soares (1981) reported that the plasma and erythrocyte ghost VE content in chicks were not well correlated. On the other hand, Lehmann (1981) suggested that RBC and plasma VE are not directly related, since the RBC content of VE is independent of the plasma total lipids which affect the plasma

VE levels. The fact that the plasma VE level may be predicted from the RBC VE concentration and vice versa, as shown here, strengthens the validity of using the 2 blood compartments as indicators of this vitamin in the chick.

The level of the plasma total lipids in the present experimental chicks was not affected by the dietary intake of VE. Available reports regarding the effect of VE supplementation on the level of the plasma total lipids are contradictory. Increased (*Lehmann* 1981), lowered (*Yang & Dessi* 1977) and no effect of VE (*Aflin-Slater* 1960) supplementation on the amount of rat plasma total lipids has been observed. A standard amount of dietary fat was provided in the present treatments, and similar levels of plasma total lipids were observed in the various chick groups. Therefore, it was not possible to determine or to speculate whether the RBC and/or the plasma VE content was affected by the plasma lipids level in the chick. It has also been observed that obese rats have a plasma total lipids and α-T concentrations which are 3 times those of normal rats with the same intake of α-T (*Bieri & Evarts* 1975). The linear increase of the VE level in the plasma lipids in response to dietary dl-αTa supplementation, as observed in this study, indicated that the plasma lipids content of VE may be used as an index of VE status when chicks are kept at a constant level of dietary fat intake.

It was shown in the present study that VE levels in whole blood, RBC and plasma were similarly sensitive to dietary dl-αTa supplementation in the range of 5.0–30.0 mg/kg. A close correlation between VE concentration in plasma vs whole blood, plasma vs RBC and whole blood vs RBC was demonstrated. Plasma lipids concentration was not affected by dietary VE intake. Alpha tocopherol was the dominant isomer in both the plasma and

the RBC. It was concluded that VE levels in plasma, whole blood and RBC were similarly valid as indicators of the VE status in the chick.

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Sammanfattning

Inverkan av dietärt E-vitamin på E-vitamininnehållet i helblod, erythrocyter och blodplasma hos kycklingar.

E-vitaminnivåerna i helblod, röda blodkroppar och plasma hos kycklingar supplementerade stegvist med 0.0, 5.0, 10.0, 20.0 och 30.0 mg dl-α-tokoferylacetat/kg foder bestämdes i syfte att studera dessa parametrars användbarhet som kriterier för E-vitaminstatus. Ökningen av E-vitaminhalten var signifikant och lineär i helblod (r = 0.90), röda blodkroppar (r = 0.89) och plasma (r = 0.93) i förhållande till E-vitaminintaget. Det förelåg ett nära samband mellan E-vitaminhalten i plasma och helblod (r = 0.90), plasma och röda blodkroppar (r = 0.91) samt helblod och röda blodkroppar (r = 0.95). Plasma E-vitaminhalten var 1.2–1.8 gånger och 6.6–12.5 gånger större än i helblod resp. i röda blodkroppar. Halten av totalfett i plasma påverkades inte av dietärt intag av E-vitamin, medan E-vitaminhalten i plasmafettet ökade signifikant med stigande E-vitaminsupplementering. Alfa-tokoferol var den dominerande isomeren (ca 92 %) i helblod, röda blodkroppar och plasma hos kycklingar vid kläckning. Små mängder av isomererna β-tokoferol (ca 2 %), γ-tokoferol (ca 5 %) och α-tokotrienol (ca 1 %) observerade vid 1 dags ålder hade minskat eller helt försvunnit vid 7 dagars ålder efter utfodring med E-vitaminfri basdiet. Resultaten visade, att hos kycklingar var E-vitaminhalterna i helblod och i röda blodkroppar lika känsliga och pålitliga som kriterier för E-vitaminstatus som E-vitaminhalten i plasma.