

Vitamin E status of muscle as related to vitamin E supply and dietary fat in the Turkey.

Michèle Viau, Brigitte Métro, Claude Genot, Hervé Rémygnon*, Gilles Gandemer

INRA, LEIMA, Lipid-Flavour group, BP 71627, 44316 Nantes Cedex 3 - France

* INRA, SRA, 37380 Nouzilly - France

BACKGROUND

Turkey meat is very sensitive to lipid oxidation because of its high polyunsaturated fatty acid (PUFA) and low vitamin E contents (Wilson et al., 1976). These two parameters are, in part, under the control of dietary lipids. Thus, fatty acid composition of muscles are strongly related to that of dietary lipids (Sklan et al., 1983; Ajuyah et al., 1993). Similarly, vitamin E status of muscles can be improved by increasing vitamin E amount in the diet (Sheldon, 1984). However, unlike pigs or chickens, a supply of 200 ppm vitamin E in turkey diet is not enough to prevent lipid oxidation in muscles. This is the consequence of (i) the low intestinal absorption of vitamin E in turkeys and (ii) the high unsaturation of lipids needing a large amount of vitamin E to prevent their oxidation. Consequently, improving oxidative stability of turkey meat requires both a decrease in PUFA content and an increase in vitamin E content of muscles. The first step of a rationale approach of this challenge is to evaluate the ability of turkeys to store vitamin E in various tissues. This can be achieved by supplying large amount of vitamin E in the diet and measuring the changes in vitamin E content in several tissues during growth. The second step consists in manipulating PUFA content of muscles through dietary lipids using fat source with various PUFA contents.

Objective: The first objective of this study dealt with the age related changes in vitamin E content of several tissues in turkeys fed commercial diets containing low or very high vitamin E amounts. The second one was to determine the effect of several dietary fat on vitamin E status in muscle.

MATERIEL AND METHODS

Turkey of BUT (British United Turkey) strain were reared in controlled conditions.

Experiment 1: At birth, animals were divided into four groups according to vitamin E content in the diet (30 - 400 ppm) and sex (males or females). Turkeys were fed *ad libitum* with commercial diets containing 7-8 % fat (21-53 % of 18:2n-6 and 2-7% of 18:3n-3). Six males were slaughtered at 4, 7, 11 and 16 weeks of age and six females at 4, 7 and 11 weeks. Animals were bled and samples were collected from breast skin, liver, *Pectoralis major* and *Sartorius* muscles.

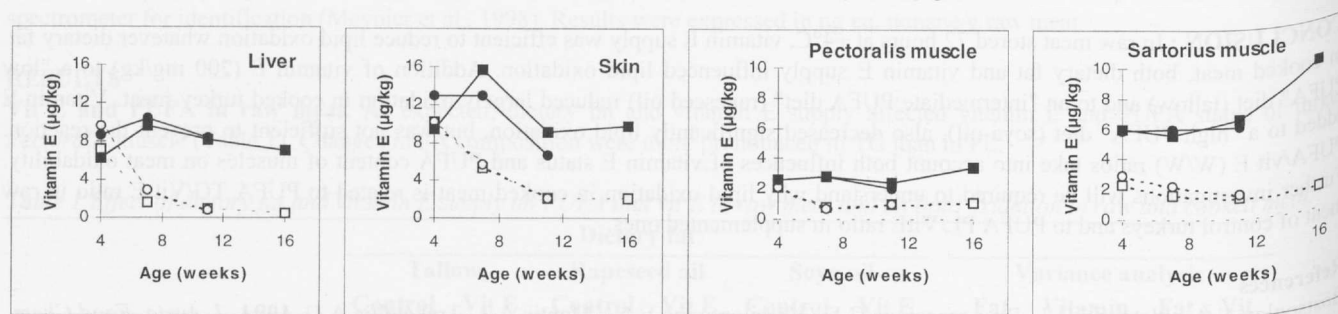
Experiment 2: At birth, male turkeys were divided into 6 batches of 6 animals according to the source of fat added to the diet (6% tallow, rapeseed oil or soya oil) and to the vitamin E level of the diet (400, 200 or 30 mg α tocopherol acetate/kg). Animals were slaughtered at 16 weeks of age and samples of *Pectoralis major* and *Sartorius* were collected.

Vitamin E content was determined according to the method of Buttriss and Diplock (1984). The results were expressed in μg of vitamin E/g tissue. Total lipids were extracted as described by Foch et al. (1957) and fractionated into neutral and polar lipids according to Juaneda and Rocquelin (1981). Fatty acid composition was determined as previously described (Alasnier and Gandemer, 1998).

RESULTS

Experiment 1 : Changes in vitamin E contents of muscles, liver and skin during turkey growth.

Figure 1 : Changes in vitamin E content during turkey growth



(○--- Control Female, ●--- Vitamin E Female, □--- Control male ■--- Vitamin E male)

The vitamin E content of liver (8-11 $\mu\text{g/g}$) skin (7-15 $\mu\text{g/g}$), *Pectoralis* (2-3 $\mu\text{g/g}$) and *Sartorius* (6-10 $\mu\text{g/g}$) was higher in supplemented animals than in controls (0.5-3 $\mu\text{g/g}$, 2-6 $\mu\text{g/g}$, 1-1.5 $\mu\text{g/g}$ and 1.5 $\mu\text{g/g}$ respectively) excepted in 4 week-old turkeys (Figure 1). In tissues of 4 week-old turkeys, vitamin E content was similar in supplemented turkeys and in controls. This is explained by the unexpected high amount of vitamin E in the diet of controls during the 4 first weeks of the experiment (97 ppm instead of 30 ppm). From 4 weeks to 11 weeks for females and to 16 weeks for males, turkeys fed 400 ppm vitamin E diet maintained the level of vitamin E in their tissues while vitamin E content decreased in tissues of controls. At 11 weeks for females and 16 weeks for males, vitamin E content of liver, *Sartorius*, skin and *Pectoralis major* was higher in supplemented turkeys than in controls (x 11, 5, 4 and 2.5 respectively). The vitamin E contents of muscles were similar in both sexes but they were higher in *Sartorius* than in *Pectoralis*. These data are in good agreement with those previously published in turkeys (Sheldon, 1984). The difference between the two muscles is mainly related to their lipid content as suggested by the vitamin E/lipid ratio, which is similar in both muscles (2-2.5). Despite the high level of vitamin E supply (400 ppm) and the length of the supplementation (11 weeks for females and 16 weeks for males), vitamin E content of turkey muscles was lower than that observed in chicken supplemented with only 200 ppm vitamin E (6 instead of 20 $\mu\text{g/g}$ in *Sartorius* and 3 instead of 17 $\mu\text{g/g}$ in *Pectoralis*) (Morrissey et al., 1997). This result confirms the poor ability of turkeys to store dietary vitamin E in their muscles (Sheldon, 1984).



Experiment 2 : Vitamin E status of muscle is modulated by dietary fat (Table 1)

Table 1
Lipid and Vitamin E contents, and PUFA proportions in triglycerides and phospholipids of Pectoralis and Sartorius according to dietary fat and vitamin E supply.

Pectoralis	Dietary Fat								
	Rapeseed			Soya			Tallow		
	Control	200	400	Control	200	400	Control	200	400
Total lipids (g/100g)	← 1.6 →			← 1.2 →			← 1.3 →		
Triglycerides PUFA (%)	← 22.4-26.2 →			← 41.2-44.2 →			← 15.8-19.4 →		
PUFA n-6	← 4.4-5.1 →			← 4.0-4.6 →			← 1.0-1.5 →		
PUFA n-3	← 26.8-31.3 →			← 45.8-48.4 →			← 16.8-20.7 →		
Total PUFA	← 33.4-33.9 →			← 40.2-45.7 →			← 36.6-37.3 →		
Phospholipids PUFA (%)	← 5.8-7.9 →			← 4.7-6.1 →			← 3.3-5.0 →		
PUFA n-6	← 39.2-41.8 →			← 44.9-51.5 →			← 40.6-41.3 →		
PUFA n-3	1.2	3.4	5.4	0.8	2.9	6.4	0.7	4.0	8.5
Total PUFA	← 45.8-48.4 →			← 51.1-54.9 →			← 46.9-47.0 →		
Vitamin E (µg/g)	2.5	6.9	11.2	1.7	5.2	14.1	1.5	7.6	16.7
Sartorius									
Total lipids (g/100g)	← 2.7 →			← 3.8 →			← 2.4 →		
Triglycerides PUFA (%)	← 25.6-25.8 →			← 43.4-43.2 →			← 15.7-18.2 →		
PUFA n-6	← 5.3-5.4 →			← 4.8-4.9 →			← 1.1-1.4 →		
PUFA n-3	← 30.9-31.2 →			← 48.2-48.1 →			← 16.9-19.5 →		
Total PUFA	← 39.8-42.3 →			← 47.3-50.8 →			← 43.8-44.1 →		
Phospholipids PUFA (%)	← 6.0-6.1 →			← 3.8-4.1 →			← 2.9-3.1 →		
PUFA n-6	← 45.8-48.4 →			← 51.1-54.9 →			← 46.9-47.0 →		
PUFA n-3	2.5	6.9	11.2	1.7	5.2	14.1	1.5	7.6	16.7
Total PUFA	← 45.8-48.4 →			← 51.1-54.9 →			← 46.9-47.0 →		
Vitamin E (µg/g)	2.5	6.9	11.2	1.7	5.2	14.1	1.5	7.6	16.7

Vitamin E content of diet did not affect lipid content and fatty acid composition of triglycerides and phospholipids in both muscles. Muscles of turkeys fed tallow diet had a higher vitamin E content than those of turkeys fed soya or rapeseed oil diets. This is related to the low PUFA proportions of triglycerides and phospholipids in both muscles of tallow turkeys. These results strongly support that feeding a low PUFA diet improves vitamin E status of muscles. This could be explained by a lower turnover of vitamin E in the low PUFA content muscles because their low propensity to oxidation.

CONCLUSION

Vitamin E status of muscle from turkeys can be improved by vitamin E by both the supply of large amounts of vitamin E in the diet and the reduction of PUFA proportions in both triglycerides and phospholipids. In these conditions, oxidative stability of muscles during processing is largely improved as shown in the other poster present in the same session (Genot et al., 1998).

REFERENCES

Alasnier C., Gandemer G., 1998. *Meat Science*, **48**, 225-235.
 Ajuyah A.O., Hardin R.T., Sim J.S., 1993. *Canadian J. Animal Science*, **73**, 177-181.
 Buttriss J.L., Diplock A.T. 1984. *Methods in enzymology*, **105**, 131-138.
 Foch J., Lees M., Sloane Stanley G.H. 1957. *J. Biological Chemistry*, **226**, 497-509.
 Genot C, Meynier A., Viau M., Metro B., Gandemer G., 1998. *Proceeding of 44th International Congress of Meat Science and Technology*. In press.
 Juaneda P., Rocquelin G., 1985. *Lipids*, **20**, 40-41.
 Lin C.F., Gray J. I., Asghar A., Buckley D.J., Booren A.M., Flegal C.J. 1989. *J. Food Science*, **54**, 1457-1460.
 Marusich W.L., DeRitter E., Ogrinz F., Keating J., Mitrovic M., Bunnell R.H. 1975. *Poultry Science*, **54**, 831-844.
 Morrissey P.A., Brandon S., Buckley D.J., Sheehy P.J.A., Frigg M. 1997. *British Poultry Science*, **38**, 84-88.
 Sheldon B.W. 1984. *Poultry Science*, **63**, 673-681.
 Sklan D., Tenne Z., Budowski P. 1983. *Poultry Science*, **62**, 2017-2021.
 Wilson, F.B., Pearson, A.M. & Shortland, F.B. (1976). *J. Agricultural Food Chemistry*, **24**, 7-10.

Acknowledgement : This work was a part of the DIETOX program supported financially by the EC (AIR2-CT-94-1577).