

# Effects of Alpha-Tocopherol on Biochemical Parameters in Adult Mice

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## ABSTRACT

**Background:** Numerous reviews report the beneficial effects of alpha tocopherol in preventive supplementation and also as an adjuvant in the treatment of some pathologies (cardiovascular, cancers). In this work, we analyzed the effects of vitamin E at high doses on some biochemical parameters.

**Methods:** Thirty-two adult male and female mice (CD1 albino mice) were randomly selected for a 4-week experiment. The mice were supplemented with alpha tocopherol at doses of 150, 400 and 750mg/day. With a high dietary intake of vitamin E

**Results:** According to our analyses, we can note Excess weight predominated in groups 4 to 7.

All the blood lipid parameters showed an abnormal concentration, as of the 400 mg dose of  $\alpha$ -T-acetate.

Hyperglycemia and hyperlipidemia were observed. These variations were more pronounced for total cholesterol and triglycerides than for HDL and LDL fractions.

**Conclusion:** The study showed significant effects of high-dose  $\alpha$ -T supplementation on biochemical parameters, mainly hyperglycemia and dyslipidemia.

**Key words:** Vitamin E, Alpha-tocopherol, blood biochemical parameters

## INTRODUCTION

Vitamin E is an essential micronutrient for humans and animals. It is a fat-soluble molecule grouped into two major families (tocopherols and tocotrienols) with four iso forms each from the alpha, beta, gamma, and delta families [1].

Human diets including vegetable oils and cereals are generally fortified with  $\alpha$ -tocopherol to meet basic nutritional requirements [1,2]. Although there are other circumstances that call for supplementation.

Vitamin E plays a vital role in physiological processes, such as fertility, oxidative homeostasis, signal transduction

and gene regulation, and in diseases, such as obesity and cancers [3, 4].

$\alpha$ -Tocopherol is best known for its antioxidant property which gives it the potential to reduce oxidative damage in cancer cells to DNA, genome instability, lipids and proteins caused by chemotherapy and radiotherapy. It increases apoptosis, inhibits cell proliferation and decreases the activities of reactive oxygen species (ROS) or nitrogen species (RNS) in the body.

And, it is now established a relationship between oxidative stress and the onset of type 2 diabetes (T2D) which makes vitamin E ( $\alpha$ -tocopherol) an ally in

the fight against hyperglycaemia and thus insulin resistance in obesity [5].

This is why it is often used in preventive supplementation and also as an adjuvant in the treatment of some pathologies (cardiovascular, cancers); [6]. However, at high doses, vitamin E can be responsible for deleterious effects, which can promote the appearance or even recurrence of certain cancers [7].

Hence the interest of this work which aims to analyse in mice the effects of vitamin E on some biochemical parameters.

## MATERIAL AND METHODS

The experiment was carried out at the Institut National de Recherche en Sciences de la Santé du Congo (IRSSA). The protocol was approved by the ethics commission of Médical Congo, under the study number 048/MRSIT/IRSSA/CERSSA.

### Materials

The materials used for the experiment on mice were

### Mouse subjects

A total of 32 male and female CD1 albino mice were selected, all 9 weeks old. They were randomly divided into seven groups (n = 4 in each group). They were obtained from the IRSSA where they lived in good conditions.

### Drug

We used synthetic vitamin E: alpha tocopherol acetate ( $\alpha$ -T-acetate), viscous liquid capsules, 500 mg, Laboratoire Pharma GDD, France.

Dietary intake rich in vitamin E

The diet (Table 1) was based on patties (about 35g per day) consisting mainly of a mixture of leafy vegetables, oil and eggs. All these foods were purchased in the public markets of Brazzaville.

### Methods

#### Alpha tocopherol supplementation

The  $\alpha$ -T-acetate was administered in each group at different doses. It was dissolved in mineral water (450 mg/ml) and administered by gavage, 4 days a week for 3

months. The dose of  $\alpha$ -T-acetate was increased according to the group. A maximum dose of 750 mg was administered in group 6. After 3 months, we evaluated the changes in blood and body tissues (kidney and liver).

The experiment was repeated twice under the same conditions to re-evaluate the results.

### Anthropometric parameters

In view of the medication, body mass was measured daily.

### Biochemical analysis

Blood samples were collected and centrifuged to obtain serum and plasma. The parameters immediately analysed on the same day were plasma creatinine, blood uric acid, cholesterol, triglycerides, high density lipoprotein (HDL), low density lipoprotein (LDL) and glucose.

All lipid parameters were measured by biochemical spectrophotometer (CyanSmart, ref cy009, version 20160902-(5.1), Belgium) in the biochemistry laboratory of the University Hospital of Brazzaville.

### Statistical analysis

The data were organised and analysed using the following software: Microsoft Excel version 2016 for the creation of the database, Epi-Info version 7.2. The ANOVA test allowed us to conclude that there was a significant difference between the batches when the p-value was less than or equal to 0.5. The data were expressed as mean  $\pm$  standard deviation. The F-test statistics and the degree of freedom (DDR) associated with each comparison were expressed.

## RESULTS

### Anthropometric data, blood lipid parameters

These are reported in (Figure 3). It can be noted that overweight is predominant in the subjects of groups 4 to 7.

### All blood lipid parameters (Table 2)

Our analyses showed an abnormal concentration of  $\alpha$ -T-acetate as early as 400 mg.

Hyperlipidaemia was observed much more pronounced for total cholesterol and triglycerides than for HDL and LDL fractions.

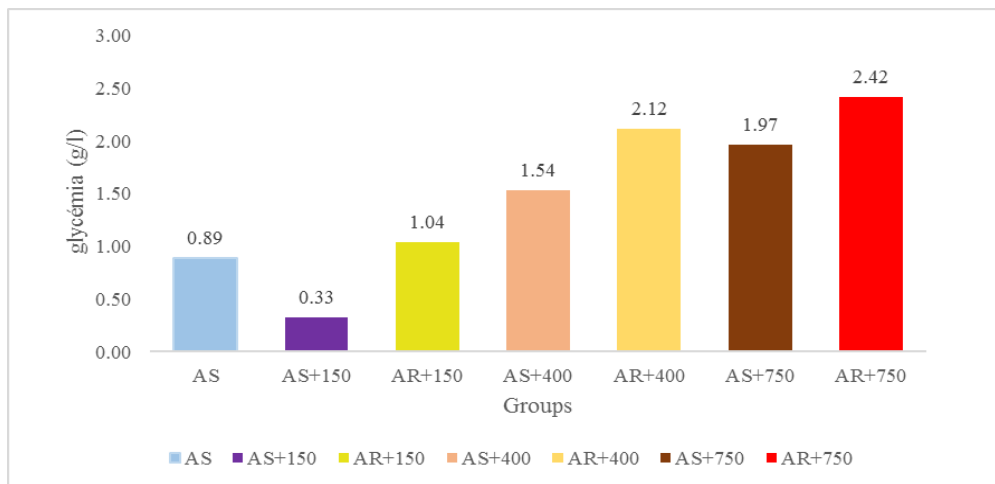


Figure 1: Variation in blood glucose levels of the different batches in the experiment

The results are expressed as mean average. The F-test statistics and the degree of freedom (DDR) associated with each comparison.

AS= control;

AS+150= batch consuming standard feed + vit E at a dose of 150mg;

AR +400 = batch consuming rich food + vit E at 400mg;

AS +400 = batch consuming standard food + vit E at 400mg;

AS + 750 = batch consuming standard feed + vit E at 750mg;

AR + 750 = batch consuming rich feed + vit E at 750mg.

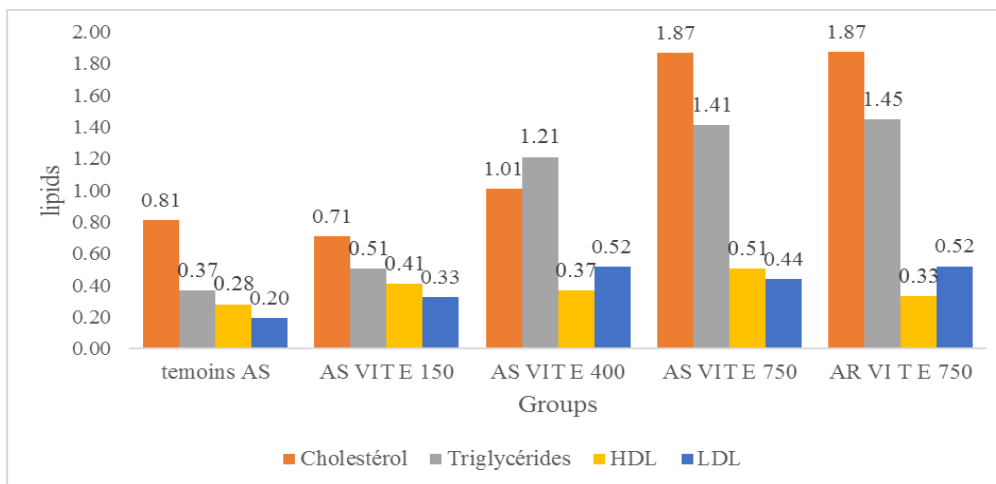


Figure 2: Variation in lipid parameters

Results are expressed as mean average. The F-test statistics and the degree of freedom (DOF) associated with each comparison.

AS= control;

AS+150= batch consuming standard feed + vit E at a dose of 150mg;

AR +400 = batch consuming rich food + vit E at 400mg;

AS +400 = batch consuming standard food + vit E at 400mg;

AS + 750 = batch consuming standard feed + vit E at 750mg;

AR + 750 = batch consuming rich feed + vit E at 750mg.

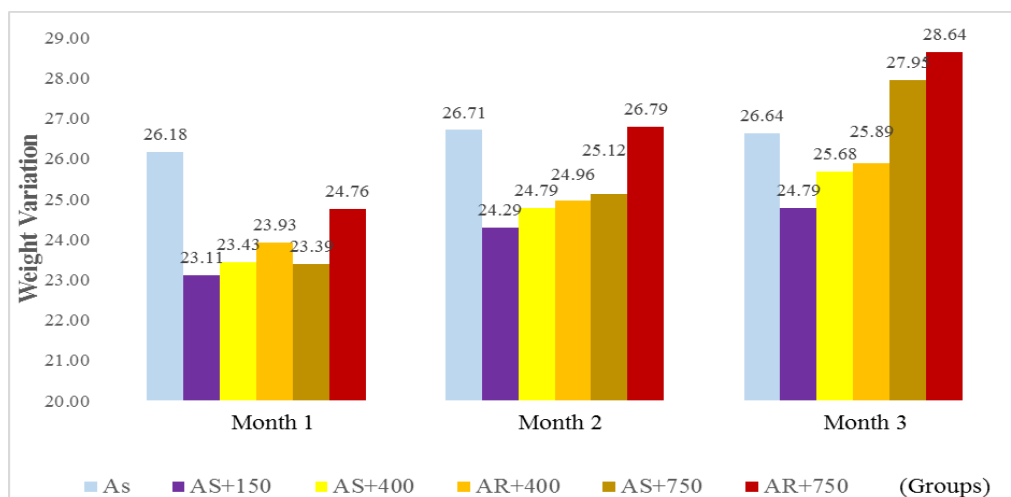


Figure 3: Weight variation

Results are expressed as mean average. The F-test statistics and the degree of freedom (DOF) associated with each comparison.

AS= control;

AS+150= batch consuming standard feed + vit E at a dose of 150mg;

AR +400 = batch consuming rich food + vit E at 400mg;

AS +400 = batch consuming standard food + vit E at 400mg;

AS + 750 = batch consuming standard feed + vit E at 750;

AR + 750 = batch consuming rich feed + vit E at 750mg.

Table 1: Vitamin E intake of rich foods by mice

Categories	Food	Quantities (g)
Oils	Unrefined palm oil	30
Vegetables	Spinach	50
	Squash	50
Cereals	Soya	450
	Wheat	450
Protein	Eggs	1(of 50)
Fruit	Avocado	50

Table 2: Biochemical parameters

Mice Groups	$\alpha$ -T (mg/day) and dietary Vit E intake	Glucose (g/l)	Total cholesterol (g/l)	Triglycerides (g/l)	HDL (g/l)	LDL (g/l)	Ureemia (g/l)	creatinine (mg/l)
	Littérature value	(0.6 -1.10 g/l)	(1,4 - 2,4 g/l)	(0,40- 1,65 g/l)	(0.31-0.5g/l) (g/l)	$\leq$ 1.25/ (g/l)	(25-65 g/l)	(6 - 14 mg/l)
Group 1 (Control group)	No $\alpha$ -T	0.96	0.70	0.30	0.22	0.20	21	4.5
	No rich dietary	0.90	0.91	0.41	0.31	0.19	22	5.2
	vit E intake	0.91	0.89	0.39	0.30	0.18	25	4.7
		0.80	0.75	0.37	0.29	0.21	22	4.2
	Median value	<b>0.89</b>	<b>0.81</b>	<b>0.37</b>	<b>0.28</b>	<b>0.19</b>	<b>22.5</b>	<b>4.65</b>
Group 2	$\alpha$ -T 150 mg,	0.31	0.52	0.41	0.33	0.25	25	5.2
	No rich dietary	0.22	0.82	0.62	0.50	0.33	29	4.9
	vit E intake	0.30	0.70	0.44	0.39	0.34	26	5.9
		0.48	0.80	0.55	0.41	0.38	24	3.7
	Mean value	<b>0.33</b>	<b>0.71</b>	<b>0.50</b>	<b>0.41</b>	<b>0.32</b>	<b>26</b>	<b>4.92</b>
Group 3	$\alpha$ -T 150 mg,	1.00	0.90	1.01	0.42	0.41	80	7.1
	and rich dietary	1.10	0.89	1.10	0.30	0.44	78	7.9
	vit E intake	1.08	0.98	1.20	0.40	0.49	62	6.9
		0.99	0.87	1.28	0.29	0.50	75	5.9
	Mean value	<b>1.04</b>	<b>0.90</b>	<b>1.15</b>	<b>0.35</b>	<b>0.46</b>	<b>73.75</b>	<b>6.95</b>
Group 4	$\alpha$ -T 400 mg	1.45	0.75	0.89	0.41	0.32	88	5.6
	No rich dietary	1.52	1.40	1.38	0.32	0.72	69	5.3
	vit E intake	1.49	1.36	1.29	0.21	0.30	73	6.3
		1.69	0.96	1.94	0.39	0.67	78	5.3
	Median value	<b>1.53</b>	<b>1.12</b>	<b>1.37</b>	<b>0.33</b>	<b>0.50</b>	<b>77</b>	<b>5.62</b>
Group 5	$\alpha$ -T 400 mg	2.50	0.85	1.25	0.28	0.62	121	8.6
	and rich dietary	1.92	1.00	1.21	0.30	0.58	89	7.9
	vit E intake	2.07	0.99	1.38	0.39	0.42	66	9.9
		1.99	1.20	1.00	0.49	0.45	62	8.7
	Median value	<b>2.12</b>	<b>1.01</b>	<b>1.21</b>	<b>0.36</b>	<b>0.51</b>	<b>84.5</b>	<b>8.8</b>
Group 6	$\alpha$ -T 750 mg	1.40	2.05	1.45	0.40	0.38	78	9.7
	No rich dietary	2.80	2.01	1.59	0.68	0.45	69	9.9
	vit E intake	2.00	2.41	1.62	0.49	0.52	72	9.0
		1.67	1.00	0.98	0.45	0.40	60	8.9
	Median value	<b>1.97</b>	<b>1.07</b>	<b>1.41</b>	<b>0.50</b>	<b>0.43</b>	<b>50.25</b>	<b>12.13</b>

Table no.2 continued...

Group 7	$\alpha$ -T 750 mg and rich dietary vit E intake	2.70	0.92	1.30	0.30	0.39	135	7.8
		2.12	2.49	1.43	0.42	0.58	79	9.2
		2.19	1.90	1.40	0.38	0.50	72	7.6
		2.68	2.18	1.66	0.22	0.61	70	6.9
	Median value	<b>2.42</b>	<b>1.87</b>	<b>1.45</b>	<b>0.33</b>	<b>0.52</b>	<b>89</b>	<b>7.9</b>

## DISCUSSION

### Anthropometric data

The excess weight of the mice in the different groups (groups) indicates marked obesity.

We observed in figure 3 that the evolution of the body weight varied in a similar way for all the working groups during the first and the second week of treatment. Then, it begins to vary differently compared to the control batches: lot1 and especially lot2 (Figure 3). During the experiment, the mice which took vitamin E grew little compared to the control mice, especially those which ate rich food exclusively without vitamin treatment, until reaching a plateau and increasing again when the treatment was stopped on the 21st day, which corresponds to a month and a half (there was still the consumption of high fat food but without vitamin E treatment).

Analysis of our results shows that vitamin E has an effect on excess body weight in mice. This increase in the body weight of mice could be explained by the antioxidant role that vitamin E. Indeed, Marlene et al in their work had already pointed out that vitamin E thanks to its antioxidant power, regulates body weight and prevents effectively obesity linked to oxidative stress [4]. This fat regulatory role of vitamin E depends not only on its antioxidant property but also on its ability to regulate certain signaling pathways [6]. Vitamin E was able to regulate body weight by inducing the expression of adiponectin in adipose tissue of an animal model via a  $\gamma$  receptor (PPAR $\gamma$ ) dependent mechanism activated by peroxisome proliferators [4,6]. Adiponectin is important in lipid metabolism because it promotes adipocyte differentiation, insulin sensitivity, and lipid accumulation [7]. Finally, recently Min Zhang et al demonstrated the regulatory role

of vitamin E on abdominal fat deposition in an animal model [8].

### Plasma biochemical parameters

Statistical analysis gives the mean values of each biochemical parameter in the different groups (2,3,4,5,6,7) and some differ widely from the control group.

Overall, the values of the control group and group 3 (diet rich in vitamin E and intake of 150 mg  $\alpha$ -T-acetate) are normal and do not show much difference. Thus, the adequate dose without adverse effects might be 150 mg of  $\alpha$ -T-acetate. It is interesting to note that group 2 without a diet rich in vitamin E had low concentrations for all parameters. This suggests that the intake of a small amount of vitamin E improves blood parameters, especially blood sugar and cholesterol. In other words, vitamin E consumed in low doses could have beneficial effects on diabetes. These observations are already reported in numerous studies [6,9].

Admittedly, the absence of a blood test for alpha tocopherol could be a limitation in this study. This is because serum vitamin E and glycated hemoglobin levels may contribute to varying the effects of dosages. The latter would therefore also require knowledge of the different blood levels of alpha tocopherol and also the degree of glycosylation of hemoglobin in laboratory mice. Our results from groups 4 to 6 show a deleterious dose of alpha tocopherol acetate from 400 mg per day regardless of the diet. Our results also made it possible to observe a biochemical disorder, especially in the mice of groups 5 and 7 (with a diet rich in vitamin E). This, leads us to believe that a diet rich in vitamin E would potentiate the effects of the supplement of  $\alpha$ -T-acetate. We can note that the balanced diet (from plant foods) provided enough vitamin E.

Finally, our results (figures 1 and 2) show that vitamin E had lipid-lowering and hypoglycemic effects at reasonable doses of 150 mg/day. Our analyzes are not isolated, Marlene et al. showed in their study that vitamin E could reduce lipid levels in mice [4].

Vitamin E given in high dose, caused considerable damage in mice including the onset of diabetes was observed. It has been reported that from doses of 400IU/day, vitamin E can become pro-oxidant and therefore harmful and cause the appearance of certain pathologies such as renal failure and type 2 diabetes linked to oxidative stress [10]. Our results can be superimposed with those of Qureshi et al, but according to them, it is rather the tocotrienol and non-tocopherol forms often used in supplementation which would be the most hypolipidemic [11]. In addition, Chin et al 2016 in their work, had even shown that the presence of alpha-tocopherol ( $\alpha$ -T-acetate) could attenuate the hypocholesterolemic effect of tocotrienol. He pointed out that the structure of tocotrienol with its three double bonds is similar to that of farnesyl, a compound which precedes the formation of squalene in the synthesis of cholesterol, and promotes the formation of farnesol from farnesyl, thus reducing the formation of squalene [12]. Furthermore, Zaiden et al. had also shown that gamma- and delta-tocotrienol can down-regulate the expression of genes involved in lipid homeostasis, such as the DGAT2 gene. APOB100, SREBP1/2 and 3-hydroxy-3-methylglutaryl-CoA reductase (HMGR) [13] This is sufficient proof that supplementation with the  $\alpha$ -T-acetate form alone would be less effective and even harmful, especially at high doses, as shown by our results (figure 1).

### **We review to clarify the mechanisms responsible for diabetes.**

Alpha-tocopherol is found in cell membranes, in the membranes of intracellular organelles and in the matrix of extracellular fibers [14]. The kidneys and

liver are one of the sites where vitamin E is primarily stored and these systems play an important role in glucose homeostasis [15].

Normally, vitamin E interacts with vitamin C to protect cell membranes, proteins, lipids (low density cholesterol lipoproteins) and DNA from oxidative stress [15]. It inhibits lipid peroxidation in cell membranes, but in high doses and when not accompanied by vitamin C supplementation, it has unwanted effects and acts as pro-oxidant [6]. Its association with vitamin C is of great physiopathological importance. Because the inhibition of lipid peroxidation by  $\alpha$ -tocopherol occurs by its conversion into an oxidized radical -tocopherol, which in turn is regenerated into  $\alpha$ -tocopherol by reduction by redox-active reagents, in particular vitamin C [15].

Hyperglycemia is also associated with oxidative stress, leaving increased production of reactive oxygen species (ROS). These activate the transcription factor RSTFKB which upregulates certain genes with multiple network connections (such as cytokines, adhesion molecules or endothelium 1), resulting in increased lipid peroxidation, protein damage and DNA [16]. According to the review, there is a link between the increase in adipocytes and the level of serum parameters leading to diabetes [4]. In humans but also in mice, it appears that factors derived from adipocytes are altered in obese subjects resulting in  $\beta$  cell dysfunction and insulin resistance [17].

### **Limitations of the study**

Our study, although preliminary, had certain limitations:

The absence of a blood test for alpha tocopherol before and after the experiment in order to relate the blood levels to the appearance of disturbances during and after the experiment.

It was also necessary to measure other blood parameters in relation to certain pathologies revealed such as glycated hemoglobin.

## CONCLUSION

The study showed significant effects of high dose  $\alpha$ -T supplementation on biochemical parameters, mainly hyperglycemia, dyslipidemia. At present, supplementation with alpha tocopherol alone would be detrimental without other antioxidants. It would also be important to check the dose and the form to be prescribed. It would therefore be preferable to consume a diet rich in vitamin E which would include all forms and would make the micronutrient available. As this study is preliminary, a human cohort is needed to assess the effects of other forms of vitamin E.

## Contributions from the Authors

Franck Arnaud Moukobolo Kinsangou and Henriette Poaty: conception, design of the experiments and writing of the article. Dimitry Moudiongui: analysis, interpretation of data and critical review of the article; Landry Martial Miguel: critical review of the article; Etienne Moukoundjimobe: critical review of the article; Jean Félix Peko: critical review of the article; Ange Antoine Abena: final approval of the version to be published. All authors have read and approved the final version of the manuscript.

## Conduct of Experiments, Data Analysis:

Franck Arnaud Moukobolo Kinsangou

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## Strong Points

1. Animal testing of alpha-tocopherol (vitamin E) with biochemical status.
2. Experience indicates that high dose vitamin E supplementation has harmful effects on the body.

**Conflict of Interest:** We do not declare any conflict of interest related to this article.

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**Ethical Approval:** Approved

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