Physiological effects of vitamin e and selenium

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Abstract

This study was planned to investigate the physiological properties of vitamin E and selenium on the human body. Vitamins are substances necessary for the body to maintain its vitality and most of them cannot be synthesised in the body, so they must be supplied with nutrients. Vitamin E and selenium together play an important role in protecting cells from oxidative damage, maintaining tissue health and supporting general health. Ensuring adequate intake of both nutrients is important for optimal health. Vitamin E, a powerful antioxidant, has been reported to protect cell membranes from oxidative damage, support the immune system and reduce inflammation. Selenium is an important trace element that enters the structure of antioxidants such as glutathione peroxidase and plays a vital role in enzymatic processes. However, Selenium is also important in the regulation of thyroid function. Selenium deficiency leads to cardiomyopathy types such as Keshan disease, and the immune system is weakened in deficiency. The synergistic effect of antioxidants such as vitamin E and selenium may reduce the risk of chronic diseases such as cancer, heart disease and neurodegenerative diseases by reducing oxidative damage at the cellular level. In this study, the sources, daily requirements and potential health effects of deficiencies of these antioxidants were discussed.

Keywords: *Vitamin E, Selenium, Physiological effect*

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E vitamini ve selenyumun fizyolojik etkileri

Özet

Bu çalışma E vitamini ve selenyumun insan vücudu üzerindeki fizyolojik özelliklerini incelemek amacıyla planlanmıştır. Vitaminler vücudun canlılığını sürdürmesi için gerekli olan maddelerdir ve çoğu vücutta sentezlenemez, bu nedenle besinlerle sağlanması gerekir. E vitamini ve selenyum birlikte hücrelerin oksidatif hasardan korunmasında, doku sağlığının korunmasında ve genel sağlığın desteklenmesinde önemli rol oynar. Her iki besinin de yeterli miktarda alınmasını sağlamak optimal sağlık için önemlidir.

Güçlü bir antioksidan olan E vitamininin hücre zarlarını oksidatif hasardan koruduğu, bağışıklık sistemini desteklediği ve inflamasyonu azalttığı belirtilmiştir. Selenyum glutatyon peroksidaz gibi antioksidanların yapısına giren ve enzimatik süreçlerde hayati rol oynayan önemli bir iz elementtir. Bununla birlikte, Selenyum tiroid fonksiyonlarının düzenlenmesinde de önemlidir. Selenyum eksikliği, Keshan hastalığı gibi kardiyomiyopati türlerine yol açmakla birlikte eksikliğinde bağışıklık sistemi zayıflar. E vitamini ve selenyum gibi antioksidanların sinerjik etkisi hücresel düzeyde oksidatif hasarı azaltarak kanser, kalp hastalıkları ve nörodejeneratif hastalıklar gibi kronik hastalıkların riskini azaltabilir. Bu çalışmada bu antioksidanların kaynakları, günlük gereksinimleri ve eksikliklerinin potansiyel sağlık üzerine etkileri tartışılmıştır.

Anahtar Kelimeler: *E Vitamini, Selenyum, Fizyolojik etki*

Introduction

Vitamins are substances that are necessary for the body to maintain vitality and most cannot be synthesized in the body, therefore need to be supplied with nutrients. Among them, vitamin E is an essential vitamin that is soluble in oils. In the body, fat is stored in organs such as muscles, liver, heart, testicles and uterus (Pereıra et al., 1999; Rizvi et al., 2014).

Vitamin E is in the structure of tocoferol and naturally contains various tokoferols such as alpha, beta, gamma, delta. Tocopherols are derivatives of chrome (2-methyl 6 chrome). Chromium is made up of a benzen with a piran circuit. α-tocoferol shows the most genis natural distribution and the greatest biological activity. The highest antioxidant activity of tocoferol

is α-tokoferol. Aromatic cells with the phenolic hydroxyl group found in their composition make up the chemically active part of the vitamin, and its antioxidant properties are derived from this group. α-tocoferol is found in different concentrations in tissues. The highest concentrations of vitamin E are found in membrane-rich cell fractions such as mitochondria and microsomes (Hall and Broughler, 1986; Buston and Ingold, 1986; Combs et al., 1975).

Absorption, transport, storage and disposal

Vitamins are absorbed from the intestines. Bile salts with oils and other fat-soluble vitamins (A,D,K) facilitate the absorption of vitamin E (Kremidjıan-Schumacher and Statzky,1987; Pereira et al., 1999).

Absorption mainly occurs in the proximal axis of the duodenum and jejunum. What is necessary for a good absorption is sufficient production of bile and, accordingly, the formation of specimens. Vitamin E absorption is negatively affected by the increase in long-chain unsaturated fatty acids (Combs et al., 1975). Pankreas enzymes (lipase) play an important role in the hydrolysis of vitamin E esters. The main product of hydrolysis of oils are free tocoferol (Ohtsuka et al., 1998).

E-vitamin or free alcohol, whether taken in the form of an ester, is absorbed as a free alcohol and from here it passes into the intestinal lymph vessels. They are absorbed by passive diffusion without any carrier protein. It is first taken from the liver, along with chilomicron residues, and participates in the blood flow through the lymph depending on lymphatic-derived lipoproteins (LDL, VLDL and Triglyceride). Tocoferols entering the general circulation in free form bind to plasma proteins (β-lipoproteins and globulines) to the liver, then transfer to extrahepatic tissues (Fenech and Ellul-Micallef, 1998; Spallhalz, 1990).

Tokoferol is transported through the blood to all the tissues of the body, where they concentrate in cell membrane-containing structures such as mitochondria, microsomes, nucleus and plasma membranes (Pehrson and Johnson, 1985).

Vitamin E is mostly stored in organs such as the liver, glands, hypofysis, lymph, testicles, pancreas, lungs, kidneys, muscle tissue, thyroid glands etc. (Oldfiel, 1987; Halliwell, 1991).

It is that the highest amount of vitamin E is excreted through the bile (65-80% in humans). It is also that vitamin E is excreted through the feces through the bile in the liver after it is converted to α-tokoferylquinone, as well as into the urine by turning into α-tocofenonic acid (Yu, 1994; Aruamo et al., 1991).

Physiological functions of vitamin E

One of the most important known properties of vitamin E is that it prevents the autooxidation of unsaturated fatty acids because it is an antioxidant. Vitamin E forms the first line of defense that protects the polyunsaturated fatty acids found in cell membrane phospholipids from the effects of free radicals. Unsaturated fatty acids react quickly with oxygen because they have double bonds, reducing the reactivity of peroxide radicals by saturating peroxides and hydroperoxides that disrupt the structure and metabolism of mitochondrial, microsomal and intrasellular membranes. The peripheral effect is prevented (Kremıdjıan-Schumacher and Statzky, 1987; Putnam and Comben, 1987).

Vitamin E is known as a chain-breaking antioxidant. Because their functions are to break down lipid peroxide radicals (LOO) and thus terminate lipide peroxidation chain reactions (Carter et al., 2005 ; Lee et al.,1998).

LOO. + α-tocoferol- OH→LOOH + α - tocoferole-O.

After all, the resulting tocoferoxyl radical is relatively stable and is not reactive enough to initiate lipid peroxidation on its own. This product of oxidation is discharged through the gallbladder by being conjugated with glucocoric acid. The antioxidant effect of tokoferol is effective in high oxygen concentrations. The highest oxygen acid tends to concentrate in lipid structures exposed to basin, such as erythrocytes and respiratory membranes .

All of the tocoferol in the erythrocytes is localized in the membrane. This localization plays an important role in the preservation of the hemolysis of cells, increasing the hemolyse of erythrocytes in the case of vitamin E deficiency. In vitro and in vivo, vitamin E supplementation reduces the hemolysis of erythrocytes (Lessard et al., 1991; Halliwell, 1991; Pennings et al., 1999; Rajashree and Puvanakrishran, 1998).

It has been argued that tocoferols play a role in the metabolism of acrylic acid leukotriens, prostoglandins and prosthocyclines, as well as in the

synthesis of DNA, and that xanthine oxitase and creatinine kinase enzymes are increased in animals with insufficient vitamin E levels (Pennings et al., 1999).

Although the effect of vitamin E on sterility is not exactly known, it has only been observed in experimental animals. The storage of vitamin E in the liver, especially in the pituitary gland, the adrenal glands, the gonates and the uterus, is considered important for the physiological functions of these organs in reproduction (Ansay, 1983).

It has been that a-tokoferol can play an important role in the structure of membrane phospholipids due to its relationship with the formation of structural components of biological membranes (Dunn, 2007; Packer, 1991).

Other functions of vitamin E include; vitamin E plays a role as an inhibitor of thrombocyte aggregation. Thus, it inhibits the peroxidation of acrylic acid, which is necessary for the synthesis of the prostoglandins necessary in thrombocyte aggregation. Thrombocyte aggregation is inhibited by prostoglandin E (Packer, 1991).

It has also been that vitamin E also plays a role in high-energy phosphate compounds such as creatine phosphat and adenysine fosphate, in the synthesis of ascorbic acid, in ubiquinone syntheses, in sulfuramino acid metabolism, and in vitamin B12 metabolism (Dunn, 2007).

Deficiency of vitamins

The disorders associated with vitamin E deficiency in animal species and humans are very different. Vitamin E deficiency in laboratory animals causes disorders in reproductive systems. Male mice with vitamin E deficiency showed testicular atrophy, testicular degeneration and infertility, fetal resorption (Julıan, 2005; Dhur et al, 1990; Dunn, 2007; Ohtsuka et al, 1998). In particular, liver necrosis, muscular dystrophy, tubular degeneration in the kidneys and vascular degenerations in the embryo are also formed (Ohtsuka et al., 1998; Pennings et al., 1999; Rajashree and Puvanakrishran, 1998).

Experimentally found vitamin E deficiency in rats in the fragilities of their lysosomes, hydrolytic enzymes released caused a general decomposition of nucleic acid, protein, carbohydrates, mucopolysaccharide and other cell

components, resulting in a typical muscle dystrophy (Hidiroglu et al., 1990; Packer, 1990).

It has been that ruminants who received insufficient vitamin E gave birth to cyclically weak puppies and that these puppies were prone to nutritional muscular dystrophy [NMD], therefore weakness and abnormal posture were especially in young animals (Ansoy,1983; Placer et al, 1966), shortness of breath due to heart and diaphragm, deterioration of heart functions, decrease in the hormones of the pituitary gland, reduction of the body's resistance along with decreased levels of gamma globülin (Ansoy, 1983).

Humans and animals havely tolerated high doses of vitamin E well. Vitamin E has been found to be less toxic compared to vitamins A and D (Dunn,2007; Ohtsuka et al, 1998). However, it cannot be said that there are no side effects at all in high doses. In studies with high doses of vitamin E in rats, chickens and humans, when 1000-2000 IU/kg of tocoferol was added to their diets, hematocrit values and reduction in cellular respiratory activity, prolongation of prothrombin in time differed in the blood table. There is a noticeable tendency to reticulosis (Vatassery et al., 1988).

It has been stated that taking high doses of vitamin E for long periods of time causes arthritis in blood pressure, nausea, allergies and disorders in iron metabolism (Combs et al., 1975).

Selenium (Se)

Selenium (Se) is a trace element necessary for the normal life of humans and animals. It was first discovered in 1918 by Berzelius, a chemist from Isvicre (Pehrson and Johnsson, 1985). Selenium has long been considered a toxic substance.

Selenium sources

The concentration of selenium in nutrients and feed is directly proportional to the Se concentration in the soil. The main source of the land is the land. If the amount of Se in the soil is sufficient, then in animals and humans fed with plants and feed grown in this soil, while in areas with insufficient level of Se, the signs of Se deficiency have been detected. Se is found in plants in the form of selenomethionine, se-methyl-selenomethione, selenosystine and selenosysteine (Bieri, 1959; Packer and Landvik, 1990; Gerloff, 1992).

An antagonistic effect has been detected between minerals such as zinc, sulfate and vanadium in the soil. Even if the amount of sulfate in the soil in excessive amounts is sufficient, it causes its insufficiency, preventing plants from binding Se (Hall and Braughlar, 1986; Lunec and Blaker, 1990).

Absorption, transport and storage

In the small intestine, it is absorbed from the regions between from the areas between duodenum-ilium and ilium-sekum. It also absorbs abomasum and rumene in ruminants. Inorganic form of Na-Selenite in monogastric It is better absorbed than ruminants (Smutna and Synes, 1979) .

Emylene Se is not fully identified in the body, but is transferred by binding to albumin in rats and LDL in humans (Jensen et al.,1988; Ullrey, 1992). The highest concentration of serum (0.2-0.8 μg/g) is found in the kidneys and liver. Skeletal muscles contain 50% of Se in the body (0.2 μg/g) (Burton and Ingold, 1986).

It is excreted with urine in single stomach animals, with feces in ruminants. The rate of excretion in the body; the pathway varies according to the amount of nutrients and the type of animal (Freeman and Cropo, 1982). When taken in toxic doses, excessive amounts are excreted through the respiratory tract. When taken orally, it is excreted in faeces and when given by injection, it is excreted in urine (Karakılcık and Aksakal, 1993).

The physiological functions

The best known function of selenium is that it plays an important role in the conversion of peroxides and hydroperoxides, resulting from lipid peroxidation by the enzyme glutathione peroxydase (GSH-Px), into water and alcohols by catabolizing. The integrity of the cell membranes is preserved. Therefore, it plays an active role inining the physiological functions of cells. (Chang et al., 1994 ; Machlin, 1980; Pennings et al., 1999; Yu , 1994).

GSH-Px, a selenoprotein, is most active in the liver and erythrocytes, moderate in the heart, stomach, adrenal glands, lungs, kidneys and adipose tissues, and at least in the brain, eye lenses, skeletal tissue and testicles. It is also necessary for the production of pancreatic lipase and the normal morphology of the pancreas, which are responsible for the normal absorption of lipids and tocopherols from the gastrointestinal tract. It has

also been that Se is involved in the structure of enzymes such as GSH-px, 5'deidonase, and enzyms such as SGOT play an important role in synthesis and activity (Burton and Ingold,1986; Placer et al., 1966).

As it prevents the hemolysis of blood vessels and the oxidation of hemoglobin by free radicals, it also serves as a carrier in plasma proteins while participating in endocrine activity and has been to have a positive effect on reproductive functions (Valko et al.2007; Combs et al., 1975).

In experimental infections in animals with selenium deficiency, these animals have been to be resistant to infections, while those animals are even more resistant against infections if they are given Se (Calvert and Cornelius, 1990; Cheeseman and Stater, 1993). It is said that it is an effective rolintln in the prevention of clinical and subclinical mastitis, which can be caused by its stimulating effect on immune functions and antibody production (Packer and Landvik, 1990; Smutna and Synes 1979). In humans, it has been recorded that in physiological amounts that Se is anti-cancer effective, there has been a positive relationship between cancer and Se concentration (Aruoma et al., 1991).

The relationship between vitamin E and Selenium

GSH-Px is a selenoprotein containing 4 atoms of selenium in each molecule. It is that GSH-Px catalyzes the transformation of fatty acids peroxides into alcohols by protecting cell membranes and subcellular membrane from oxidative destruction (McIntosh et al, 1998 ; Pennings et al., 1999).

The tocoferol molecules bind to the doymamide fatty acid molecule and form weak chemical compounds with them during cellular respiration (Pennings et al., 1999; Packer, 1991). They do this in this way; because unsaturated fatty acids have double bonds, they quickly react with oxygen to form peroxides and hydroperoxides. Here, vitamin E saturates the hydrogen protons with peroxide and hydroperoxide, which stops the reaction by reducing the activity of peroxide radicals. The formed peroxides are partitioned via GSH-Px (Buston and Ingold, 1986; Halliwell, 1991).
If the tocoferols are not sufficient to bind to all of the saturated fatty acid molecules, or if the amount of GSH-Px is not enough to block all peroxides, then tissue coagulation occurs. The symptoms of vitamin E deficiency are similar. Vitamins and vitamin E can never replace each other (Vanmetre and Collan, 2001; McIntosh et al., 1998).

Selenium affects vitamin E needs in at least three ways. First, it enables the absorption of vitamin E, a vitamin that dissolves in fat by ensuring that its fat loss occurs normally by preserving the integrity of the pancreas. Secondly, the unknown It helps to prevent the retention of Evitamini in blood plasma. Third, it reduces the use of vitamin E by entering the structure of the GSH-Px enzyme by converting free radicals into water (McGrath et al.,1997; Packer and Landvik, 1990).

Conclusion

Vitamins are substances that are necessary for the body to maintain vitality and most cannot be synthesized in the body, therefore need to be supplied with nutrients. Together, vitamin E and selenium play crucial roles in protecting cells from oxidative damage, maintaining tissue health, and supporting overall well-being. Ensuring an adequate intake of both nutrients is important for optimal health.

References

Ansay, M. (1983). Les glucocorticoides: pharmacologie et principes d'utilisation en clinique. Pt. 2. Synthese. *Annales de Medecine Veterinaire* (Vol. 127). 249-264(4)

Aruoma, O. I., Kaur, H., & Halliwell, B. (1991). Oxygen free radicals and human diseases. *Journal of the Royal Society of Health*, 111(5), 172–177. https://doi.org/10.1177/146642409111100506

Bıerı J. G. (1959). An effect of selenium and cystine on lipide peroxidation in tissues deficient in vitamin E. *Nature*, 184(Suppl 15), 1148–1149. https://doi.org/10.1038/1841148a0

Burton, G. W., & Ingold, K. U. (1986). Vitamin E: application of the principles of physical organic chemistry to the exploration of its structure and function. *Accounts of Chemical Research*, 19(7), 194-201.

Calvert, C. A., & Cornelius, L. M. (1990). The pharmacodynamic differences among glucocorticoid preparations. *Veterinary Medicine*, 85 (8), 860-865.

Carter, J. N., Gill, D. R., Krehbiel, C. R., Confer, A. W., Smith, R. A., Lalman, D. L., ... & McDowell, L. R. (2005). Vitamin E supplementation of newly arrived feedlot calves. *Journal of Animal Science*, 83(8), 1924-1932.

Chang, W. P., Hom, J. S., Dietert, R. R., Combs, G. F., Jr, & Marsh, J. A. (1994). Effect of dietary vitamin E and selenium deficiency on chicken splenocyte proliferation and cell surface marker expression. *Immunopharmacology and Immunotoxicology*, 16(2), 203–223. https://doi.org/10.3109/08923979409007091

Cheeseman, K. H., & Slater, T. F. (1993). An introduction to free radical biochemistry. *British Medical Bulletin*, 49(3), 481–493. https://doi.org/10.1093/oxfordjournals.bmb.a072625

Combs, G. F., Jr, Noguchi, T., & Scott, M. L. (1975). Mechanisms of action of selenium and vitamin E in protection of biological membranes. *Federation Proceedings*, 34(11), 2090–2095.

Dunn, A., Galan, P., & Hercberg, S. (1990). Relationship between selenium, immunity and resistance against infection. Comparative biochemistry and physiology. *Comparative Pharmacology and Toxicology*, 96(2), 271–280. https://doi.org/10.1016/0742-8413(90)90007-v

Fenech, A. G., & Ellul-Micallef, R. (1998). Selenium, glutathione peroxidase and superoxide dismutase in maltese asthmatic patients: effect of glucocorticoid administration. *Pulmonary Pharmacology & Therapeutics,* 11(4), 301–308. https://doi.org/10.1006/pupt.1998.0122

Freeman, B. A., & Crapo, J. D. (1982). Biology of disease: free radicals and tissue injury. Laboratory investigation; *Journal of Technical Methods and Pathology*, 47(5), 412-426.

Gerloff, B. J. (1992). Effect of selenium supplementation on dairy cattle. *Journal of Animal Science*, 70(12), 3934–3940. https://doi.org/10.2527/1992.70123934x

Hall, E. D., & Braughler, J. M. (1986). Role of lipid peroxidation in post-traumatic spinal cord degeneration: a review. *Central Nervous System Trauma*, 3(4), 281-294.https://doi.org/10.1089/cns.1986.3.281

Halliwell, B. (1991). Reactive oxygen species in living systems: source, biochemistry, and role in human disease. *The American Journal of Medicine*, 91(3), S14-S22.https://doi.org/10.1016/0002-9343[91]90279-7

Hidiroglou, M., Batra, T. R., & Zhao, X. (1997). Bioavailability of vitamin E compounds and the effect of supplementation on release of superoxide

and hydrogen peroxide by bovine neutrophils. *Journal of Dairy Science*, 80(1), 187–193. https://doi.org/10.3168/jds.S0022-0302(97)75926-5

Jensen, M., Hakkarainen, J., Lindholm, A., & Jönsson, L. (1988). Vitamin E requirement of growing swine. *Journal of Animal Science*, 66(12), 3101–3111. https://doi.org/10.2527/jas1988.66123101x

Julian R. J. (2005). Production and growth related disorders and other metabolic diseases of poultry--a review. *Veterinary Journal*, 169(3), 350–369. https://doi.org/10.1016/j.tvjl.2004.04.015

Karakılcık A. Z. and Aksakal, M. (1993). Some Metabolic Functions and Metabolism of Selenium and its relations with Vitamin E,4(2) 283-291 https://doi.org/10.58600/eurjther-199342-1111-x

Kiremidjian-Schumacher, L., & Stotzky, G. (1987). Selenium and immune responses. *Environmental research*, 42(2), 277–303. https://doi.org/10.1016/s0013-9351(87)80194-9

Lee, J. W., Iwatsuru, M., & Nishigori, H. (1998). Alteration of Activities of Hepatic Antioxidant Defence Enzymes in Developing Chick Embryos After Glucocorticoid Administration-A Factor to Produce Some Adverse Effects*? Journal of Pharmacy and Pharmacology*, 50(6), 655-660.https://doi.org/10.1111/j.2042-7158.1998.tb06901.x

Lessard, M., Yang, W. C., Elliott, G. S., Rebar, A. H., Van Vleet, J. F., Deslauriers, N., Brisson, G. J., & Schultz, R. D. (1991). Cellular immune responses in pigs fed a vitamin E- and selenium-deficient diet. *Journal of Animal Science*, 69(4), 1575–1582. https://doi.org/10.2527/1991.6941575x

Lunec, J., & Blake, D. (1990). Oxygen free radicals: their relevance to disease processes. Cohen RD, Lewis B, Albert KGMM. The Metabolic and Moleculer Basis of Acquired Disease. Balliere Tindall, London, 189-212.

Machlin, L. J. (Ed.). (1980). Vitamin E. A comprehensive treatise (pp. x+-660pp).

McGrath, L. T., Treacy, R., McClean, E., & Brown, J. H. (1997). Oxidative stress in cyclosporin and azathioprine treated renal transplant patients. *Clinica Chimica Acta; lnternational Journa of Clinical Chemistry*, 264(1), 1–12. https://doi.org/10.1016/s0009-8981(97)00088-0

McIntosh, L. J., Hong, K. E., & Sapolsky, R. M. (1998). Glucocorticoids may alter antioxidant enzyme capacity in the brain: baseline studies. *Brain Research*, 791(1-2), 209–214. https://doi.org/10.1016/s0006-8993(98)00115-2

Ohtsuka, A., Kojima, H., Ohtani, T., & Hayashi, K. (1998). Vitamin E reduces glucocorticoid-induced oxidative stress in rat skeletal muscle. *Journal of Nutritional Science and Vitaminology*, 44(6), 779–786. https://doi.org/10.3177/jnsv.44.779

Ohtsuka, A., Ohtani, T., Horiguchi, H., Kojima, H., & Hayashi, K. (1998). Vitamin E reduces glucocorticoid-induced growth inhibition and lipid peroxidation in rats. *Journal of Nutritional Science and Vitaminology*, 44(2), 237–247. https://doi.org/10.3177/jnsv.44.237

Oldfield, J. E. (1987). The two faces of selenium. *The Journal of Nutrition*, 117(12), 2002-2008. https://doi.org/10.1093/jn/117.12.2002

Packer L. (1991). Protective role of vitamin E in biological systems. *The American Journal of Clinical Nutrition*, 53(4 Suppl), 1050S–1055S. https://doi.org/10.1093/ajcn/53.4.1050S

Packer, L., & Landvik, S. (1990). Vitamin E in biological systems. *Advances in Experimental Medicine and Biology*, 264, 93–103. https://doi.org/10.1007/978-1-4684-5730-8_13

Pehrson, B., & Johnsson, S. (1985). Selenium and glutathione peroxidase in blood and tissues and growth and feed efficiency in young bulls at different dietary selenium levels. *Zentralblatt fur Veterinarmedizin. Reihe A,* 32(7), 492–501. https://doi.org/10.1111/j.1439-0442.1985.tb01969.x

Pennings, H. J., Borm, P. J., Evelo, C. A., & Wouters, E. M. (1999). Changes in levels of catalase and glutathione in erythrocytes of patients with stable asthma, treated with beclomethasone dipropionate. *European Respiratory Journal*, 13(6), 1260-1266.

Pereira, B., Bechara, E. J. H., Mendonca, J. R., & Curi, R. (1999). Superoxide dismutase, catalase and glutathione peroxidase activities in the lymphoid organs and skeletal muscles of rats treated with dexamethasone. *Cell Biochemistry and Function: Cellular Biochemistry and Its Modulation by Active Agents or Disease*, 17(1), 15-19.

Placer, Z. A., Cushman, L. L., & Johnson, B. C. (1966). Estimation of product of lipid peroxidation (malonyl dialdehyde) in biochemical systems. *Analytical Biochemistry*, 16(2), 359–364. https://doi.org/10.1016/0003-2697(66)90167-9

Putnam, M. E., & Comben, N. (1987). Vitamin E. *The Veterinary Record*, 121(23), 541–545.

Rajashree, S., & Puvanakrishnan, R. (1998). Dexamethasone induced alterations in enzymatic and nonenzymatic antioxidant status in heart and kidney of rats. *Molecular and Cellular Biochemistr*y, 181, 77-85.

Rizvi, S., Raza, S. T., Ahmed, F., Ahmad, A., Abbas, S., & Mahdi, F. (2014). The role of vitamin e in human health and some diseases. *Sultan Qaboos University Medical Journal*, 14(2), e157–e165.

Smutna, M., & Synek, O. (1979). Lipid peroxidation in semen of the boar. *Acta Veterinaria Brno*, 48(1-4), 35-43.

Spallholz J. E. (1990). Selenium and glutathione peroxidase: essential nutrient and antioxidant component of the immune system. *Advances in Experimental Medicine and Biology*, 262, 145–158. https://doi.org/10.1007/978-1-4613-0553-8_12

Ullrey D. E. (1992). Basis for regulation of selenium supplements in animal diets. *Journal of Animal Science*, 70(12), 3922–3927. https://doi.org/10.2527/1992.70123922x.

Valko, M., Leibfritz, D., Moncol, J., Cronin, M. T., Mazur, M., & Telser, J. (2007). Free radicals and antioxidants in normal physiological functions and human disease. *The International Journal of Biochemistry & Cell Biology*, 39(1), 44–84. https://doi.org/10.1016/j.biocel.2006.07.001

Van Metre, D. C., & Callan, R. J. (2001). Selenium and vitamin E. *The Veterinary Clinics of North America. Food Animal Practice*, 17(2), 373–viii. https://doi.org/10.1016/s0749-0720(15)30034-7

Vatassery, G. T., Brin, M. F., Fahn, S., Kayden, H. J., & Traber, M. G. (1988). Effect of high doses of dietary vitamin E on the concentrations of vitamin E in several brain regions, plasma, liver, and adipose tissue of rats. *Journal of Neurochemistry*, 51(2), 621–623. https://doi.org/10.1111/j.1471-4159.1988.tb01083.x

Yu B. P. (1994). Cellular defenses against damage from reactive oxygen species. *Physiological Reviews*, 74(1), 139–162. https://doi.org/10.1152/physrev.1994.74.1.139