Selenium and Vitamin E Supplementation on Blood Antioxidants in Moderately Exercised Horse

Elías Velázquez Cantón and Aurora H Ramírez Pérez

Facultad de Medicina Veterinaria y Zootecnia. UNAM. Ciudad de México, Mexico

Submission: September 01, 2018; Published: September 11, 2018

*Corresponding author: Aurora H Ramírez Pérez, Animal Nutrition and Biochemistry Department. Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México. UNAM-CU, 04510, Mexico, Email: rperez@unam.mx

Abstract

The objective of this article is to discuss the effect of selenium and vitamin E supplementation on the activity of erythrocyte superoxide dismutase and zinc (Zn) and copper (Cu) blood concentrations in moderately exercised horses. Exercise changes a horse’s physiology and metabolism, depending on its intensity and duration, and the animal’s training. The reactive oxygen species produced during physical activity could provoke oxidative damages to biomolecules (lipids, proteins, nucleic acids). The first enzymatic antioxidant of red-blood cells is cytosolic superoxide dismutase (SOD) which dismutes superoxide anion (O\textsuperscript{2-}) to hydrogen peroxide. SOD contains zinc (Zn) and copper (Cu). Selenium (Se) and vitamin E hinder oxidative harm, but more research is needed to understand their antioxidant effect during exercise. We concluded that Se and vitamin E are powerful antioxidants, which did not affect erythrocyte SOD activity, and Zn and Cu blood concentrations; however, they were strongly modified by physical activity, results that would imply that moderate exercise also causes oxidative stress.

Keywords: Horses; Selenium; DL-alphatocopherol; Exercise; Superoxide dismutase; Zinc; Copper; Ascorbic acid; α-tocopherol; β-carotene; Hydrogen peroxide; Erythrocyte; Osteogenesis; Pigmentation; Oxygen

Introduction

Exercise changes a horse’s physiology and metabolism, depending on its intensity and duration, and the animal’s capabilities and training, but changes due to moderate exercise have scarcely been studied in horses. Depending on the intensity, duration, and type of exercise, the reactive oxygen species produced could provoke oxidative stress [1]. Cytosolic superoxide dismutase (SOD) is the first enzymatic antioxidant of red-blood cells; it dismutes superoxide anion (O\textsuperscript{2-}) to hydrogen peroxide and it contains zinc (Zn) and copper (Cu). Selenium (Se) and vitamin E (E) hinder oxidative harm, but more research is needed to understand their antioxidant effect during exercise. The aim of this article is to briefly discuss the effect of selenium and vitamin E supplementation (synthetic vitamin E form) on the activity of erythrocyte superoxide dismutase and zinc (Zn) and copper (Cu) blood concentrations in moderately exercised horses.

Erythrocyte Superoxide Dismutase Activity

SOD (EC 1.15.1.1) is necessary to maintain life in aerobic conditions [2]; two types of SOD have been identified in mammals: 1) Manganese-SOD (MnSOD) located mainly into the mitochondrial matrix; 2) CuZnSOD located mainly in the cytosol [3]. Each monomer of cytoplasmic SOD contains one atom of each Cu and Zn [4]. SOD dismutes the superoxide anion to hydrogen peroxide and molecular oxygen [5]. In the absence of SOD, the superoxide anion can generate hydroxyl radicals, and trigger lipid peroxidation. Both, the diet and other environmental conditions influence the activity of CuZnSOD and MnSOD, enzymes that increase their activity under conditions that favor the production of superoxide radicals. It has been consigned, that Se and vitamin E supplementation affect physical performance [6]. However, physical activity increases the activity of the SOD [6,7]; this fact implies an increase in the production of superoxide anion. In jumping competitions performed in tropical conditions, where horses were not supplemented with antioxidants, it was observed that the activity of erythrocyte SOD increased 1.59 times 24 h after the event; this increase was explained in terms of the oxidants generated [8]. On the other hand, in elite cyclists it was observed that after two months of supplementation with vitamins E (400mg / day) and C (500mg / day), serum SOD activity decreased (from 14.60 to 13.24 U / mg protein) [9]. In another study in equines [10], the erythrocyte activity of SOD was not affected by physical exercise, when Se and vitamin E were applied. De Moffarts et al. [1] reported that in horses supplemented with Se, the use of the glutathione peroxidase system (main antioxidant enzyme of the erythrocyte) seems to be privileged with respect to SOD; in addition, erythrocyte has a...
superoxide anion pumping system that contributes to decrease anion concentrations [11].

**Zinc and Copper**

Zinc is a structural part of different enzymes, including mono-oxides; it participates in osteogenesis and reproductive function, prevents chronic diseases as arteriosclerosis, diabetes and neurodegenerative disorders. It acts as an antioxidant and anti-inflammatory [12]; in addition, it reduces DNA damage [13]. Its deficiency causes stomatitis, parakeratosis, rough hair, joint stiffness, and reproductive disorders. In addition, an increase in lipoperoxidation markers is observed [14]. Zn performs its antioxidant function through proteins, such as metallothioneins; besides being a structural part of the SOD, in relation to copper [14]. Zn facilitates the neutralization of free radicals [15]. The dietary recommendations for the horse are 50 mg / kg DM [16]. Zn toxicity is rare, since it has a wide margin of safety in the horse [17]. Copper helps iron to be incorporated into hemoglobin; participates in osteogenesis, in pigmentation of the skin and iris, is an enzymatic cofactor (cytochrome oxidase C, tyrosinase, monoamine oxidase, ceruloplasmin, and galactosidase). Cu takes part of non-enzymatic proteins (erythrocuprein, hepaocuprein, brain cuprein, milk protein), besides it participates in gene expression.

The deficiency of Cu decreases the effectiveness of the immune system, causes anemia, fractures, achromotrichia, ataxia, spastic paralysis, severe incoordination, brain damage, alteration in the function of the respiratory chain. In humans, Cu has occasionally been associated with cardio-metabolic alterations, such as insulin resistance and diabetes, associated with obesity. Its main role as an antioxidant is its participation in the structure of SOD [18]. In the horse, dietary recommendations for Cu are 10 mg / kg DM [16]; with significant tolerance to dietary copper, until 250mg / kg of food [17]. The effect of physical activity on blood concentrations of Zn and Cu is controversial. Some authors do not report it [1] when antioxidants are supplemented (ascorbic acid, 11.5, a-tocopherol, 7.0, f-carotene, 0.5, Cu, 0.187, Zn, 0.769, g / day; 7.0 mg / day) in the diet. Other authors mention that blood Zn and Cu are not affected by race, gender, age or discipline [19]. In Anatolian horses an increase of Cu and Zn has been reported in animals that were not supplemented with Se and vitamin E [20]. We supplemented horses with both Se and vitamin E to studied erythrocyte SOD activity and Cu and Zn blood concentrations; our results just showed an effect of moderate exercise on the studied variables.

**Conclusion**

Selenium and vitamin E supplementation effect on erythrocyte SOD activity or Zn and Cu blood concentrations remains controversial. However, those variables are affected by moderate physical activity, results that could imply that moderate exercise also causes oxidative stress; in this case Se and vitamin E supplementation could benefit moderately exercised horses.

**References**

1. De Moffarts B, Kirschvink N, Art T, Pincemail J, Lekeux P (2005) Effect of oral antioxidant supplementation on blood antioxidant status in trained thoroughbred horses. Vet J 169: 65-74.
2. Michels C, Raes M, Toussaint O, Remacle J (1994) Importance of Se-glutathioneperoxidase, catalese, and Cu/Zn-SOD forcellular survival againstoxidative stress. Free RadicalBioMed 17(3): 235-248.
3. Zidenberg-Cherr S, Keen CL, Lönnerdal B, Hufnay LS (1983) Superoxidedismutaseactivity and lipidperoxidation in threet developmental correlations affected by manganesedeficiency. J Nut 113(12): 2498-2504.
4. Fridovich I (1976) Superoxidedismutases: studies of structure and mechanism. In Iron and CopperProteins. Springer, Boston, MA. pp. 530-539.
5. Juarez JC, Manuia M, Burnett ME, Betancourt O, Boivin B, et al. (2008) Superoxide dismutase 1 (SOD1) issestentialfor H2O2-mediated oxidation and inactivation of phosphates in growth factor signaling. Proceedings of the NationalAcademy of Sciences, 105(20): 7147-7152.
6. Lamprecht ED, Williams CA (2012) Biomarkers of antioxidantstatus, inflammation, and cartilage metabolism are affected by acute intense exercise but not superoxidedismutase supplementation in horses. Oxid. Med. Cell. Longev 2012: 920932.
7. Akil M, Gurbuz U, Bicer M, Sivrikaya A, Mogulkoc R, et al. (2011) Effect of seleniumsupplementation on lipidperoxidation, antioxidant enzymes, and lactatelevels in rats immediately afteracuteswimming exercise. Biol Trace Elem Res 142(3): 651-659.
8. Soares JCM, Zanella R, Bondan C, Alves LP, de Lima MR, et al. (2011) Biochemical and antioxidantchanges in plasma, serum, and erythrocytes of horsesbefore and after a jumping competition. J Equine Vet Sci 31(7): 357-360.
9. Gupta C, Gupta PH, Singh B (2009) Effect of Vitamin Supplementation on Exercise Induced Oxidative Stress in Trained Elite Indian Cyclists. AJBIO 1: 166-70.
10. Ono K, Inui K, Hasegawa T, Matsuki N, Watanabe H et al. (1990) The changes of antioxidative enzymeactivities in equine erythrocytes following exercise. JVS 52(4): 759-765.
11. Haftos JE, Whillier S, Kuchel PW (2010) Glutathione synthesis and turnover in the human erythrocytealignment of a modelbased on detailed enzymekinetics with experimental data. JBC 285(31): 23557-23567.
12. Prasad AS (2014) Zinc: antioxidant and anti-inflammatory agent: role of zinc in degenerative disorders of aging. J Trace Elem Med Biol 28(4): 364-371.
13. Sharif R, Thomas P, Zalewski P, Fenech M (2015) Zinc supplementation influences genomics, stability, biomarkers, antioxidant activity, and zinc transporter genes in an elderlyAustralian population with low zinc status. Mol NutrFood Res 59(6): 1200-1212.
14. Bashandy SAEM, Omara EAA, Elbad H, Amin MM, Soliman MS (2016) Role of zinc as antioxidant and anti-inflammatorycyto relieve cadmiumoxidative stress induced testicular damage in rats. Asian Pac J Trop Biomed 6(12): 1056-1064.
15. Cruz KJC, da Oliveira ARS, do Nascimento MarreiroD (2015) Antioxidant role of zinc in diabetes mellitus. WJD 6(2): 333-337.
16. MartinRosset W (2012) Nutrition et Alimentation des Chevaux. Editions Quae.
17. NRC (2007) Nutrient requirements of horses, (6thedn). Nat Acad Press. Washington, D.C, USA.
18. Kamiya T, Takeuchi K, Fukumoto S, Hara H, Adachi T (2018) Copper-chaperone antioxidant-1, Atox-1, is involved in the induction of SOD3 in THP-1 cells. BioMetals 31(1): 61-68.

19. Kirschvink N, De Moffarts B, Farnir F, Pincemail J, Lekeux P (2006) Investigation of blood oxidant/anti-oxidant markers in healthy competition horses of different breeds. Equine Vet J 38: 239-244.

20. Yur F, Dede S, Deger Y, Kilicalp D (2008) Effects of vitamin E and selenium on serum trace and major elements in horses. Biol Trace Elem Res 125(3): 223-228.

This work is licensed under Creative Commons Attribution 4.0 License
DOI: 10.19080/JDVS.2018.07.555720