ANTIOXIDANT ENZYMES STATUS AND OXIDATIVE STRESS MARKERS IN DRAFT HORSES WITH ACUTE AND CHRONIC LOWER AIRWAY DISEASES

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ABSTRACT

The aim of the present study was to evaluate the oxidative stress level and antioxidant enzymes status associated with lower respiratory tract disorders in draft horses. For this purpose, venous blood samples were collected from (90) draft horses exhibiting signs of lower respiratory tract disorders and other (20) clinically healthy horses were considered as a control group. Plasma reduced glutathione (GSH) concentration and antioxidant enzymes activity; including glutathione reductase (GR), glutathione-S-transferase (GST), superoxide dismutase (SOD) and catalase (CAT) as well as plasma vitamin C and total antioxidant capacity (TAC) levels were assayed. Serum uric acid and malondialdehyde (MDA) levels and low density lipoprotein (LDL) concentrations were also measured. Biochemically, GR and SOD activities, uric acid, MDA, LDL were significantly increased ($p<0.05$) in diseased horses compared with control group. Meanwhile, there was a significant decrease ($p < 0.05$) of GST and CAT activities and vitamin C level in diseased horses in comparison with control group. The present investigation indicates that oxidative stress with alteration of antioxidant enzymes activities are feature of respiratory diseases in draft horses.

Key Words: *Respiratory diseases; Antioxidant enzymes; Free Radicals; Oxidative stress markers; Draft horses.*

INTRODUCTION

Respiratory diseases continue to be a major problem for horses worldwide (**Bailey et al., 1999**). In recent years; it has become increasingly apparent that inflammatory airway diseases (IAD) is a common disorder which can affect horses of any age and are more common in young race horses, despite marked differences in management and climate; their prevalence and incidence were around 12% and 10% monthly, respectively (**Christley et al., 2001 and Wood et al. 2005**).

Reactive oxygen species (ROS) have been shown directly to damage pulmonary tissue resulted in direct destruction of pulmonary epithelium and supportive structures (**Reddy et al., 1992**), degrade alveolar surfactant with subsequent collapse of the alveoli and signifi-

cant increase in pulmonary vascular resistance **(Mills and Higgins, 1997)**. Also, they contribute to cell death and lysis of sensitive cells of the lung tissue causing microvascular and alveolar disruptions with subsequent inflammation of the lung tissue **(Haddad, 2002)**. so, it would be erroneous to suggest that ROS are the primary mediators of the pulmonary inflammatory response.

ROS are not always the primary or initiating cause of the disease process but probably contribute to and certainly exacerbate it **(Doelman and Bast 1990)**. ROS have been associated with exercise-induced pulmonary hemorrhage **(Derksen, 1997)** and have been implicated in the pathogenesis of recurrent airway obstruction, IAD (**Rahman and MacNee, 2000 and Deaton et al., 2004) and** chronic airway diseases (**Kirschvink et al., 2002a)** in horses causing disturbance in oxidant-antioxidant equilibrium.

As with the chemical antioxidants, cells are protected against oxidative stress by an interacting network of antioxidant enzymes which are manufactured in the body providing an important defense against free radicals (**Davies, 1995 and Helmut, 1997**). Catalase (CAT), superoxide dismutase (SOD) and the glutathione antioxidant system reduced glutathione (GSH), glutathione peroxidase (GPx), glutathione reductase (GR), glutathione-S-transferase (GST) are the most important antioxidant enzymes **(Schriner et al., 2005)**.

Glutathione status appeared to be a sensitive and discriminating marker for the degree of airway inflammatory diseases and their synthesis appeared to be regulated through many inflammatory mediators **(Rahman and MacNee, 2000 and Kirschvink et al. 2002b)**. GR is an important cellular antioxidant enzyme requiring NADPH for the reduction of glutathione disulfide to GSH **(Meister, 1988)**. Studies measuring GR in asthma are more scant but, **Gumral et al. (2009)** found a 5.7% higher erythrocyte GR activity in asthma compared to healthy controls and associated with current symptoms of bronchial hyperreactivity. GST has oxidant-scavenging and anti-inflammatory properties protecting the cells against numerous oxidant generating compounds, including pollutants, lipid hydroperoxides and other end products of oxidative metabolism but, its relative importance in lung diseases is still unknown **(Ognjanovi_ et al. 1995 and Tripathi et al., 2010)**.

Uric acid is one of the non-enzymatic endogenous antioxidants which scavenge hydroxyl radicals **(Chew, 1994)** and found to be increased with enhanced ROS generation in cases of oxidative stress **(De Moffarts et al. 2006)**. Vitamin C is considered as a highly effective antioxidant acting to lessen oxidative stress **(Higdon, 2006)** and found to be decreased in horses suffered recurrent airway obstruction (RAO) either in the presence or absence of airway inflammation as a result of oxidative stress **(Hargreaves et al. 2002 and Deaton et al. 2004)**.

Oxidative stress markers or "accumulators" are the end products of oxidative reactions which are generated during the procedure from the decomposition of lipid hydroperoxides, such as malondialdehyde (MDA) which is usually accumulates under stress conditions to a measurable level above the

unstressed condition **(Lykkesfeldt and Svendsen, 2007)**. The objective of the present study was to assess and evaluate the changes in the antioxidant enzymes and the associated oxidative stress in acute and chronic lower airway diseases in draft horses.

MATERIALS AND METHODS

1. Animals :

A total of 90 draft horses of both sexes aging between 1-4 years exhibiting the signs of lower airway diseases have been used in this study. In addition, 20 clinically healthy ones at the same age were randomly selected and served as a control group. The present study was carried out in the period between April, 2009 and October 2011 at Dakahlia Governorate. Data concerned with case history, clinical findings, and medical record for each horse were also recorded.

2. Clinical examination :

Detailed clinical examinations of the horses were carried out, and the clinical findings were recorded **(Kelly, 1984)**. Diseased horses were categorized into "group 1" representing horses with acute IAD and "group 2" representing horses with chronic IAD based on the criteria: pattern of fever, nasal discharges, cough, signs of dyspnea, tracheal and lung auscultative sounds and signs of chest pain.

3. Sampling :

Three venous blood samples (ten ml for each) were collected via jugular vein puncture from each horse; the first blood sample was collected into clean tube containing 5mg sodium ethylene diamine tetra acetic acid (EDTA) as anticoagulant was used for evaluation of total and differential leukocytic counts. The second blood sample was collected into heparinized syringe and centrifuged at 3000 rpm for 10 minutes to collect blood plasma; meanwhile, the third blood sample was collected without anti-coagulant and centrifuged at 3000 rpm for 10 minutes to collect blood serum. Both serum and plasma samples were kept frozen at -20 C for further biochemical analysis.

4. Hematological examinations :

Total and differential leukocytic counts were counted in all horses under investigation **(Kelly, 1984)**.

5. Biochemical Analysis :

The plasma GSH concentration, the activity of plasma GR, GST, SOD and CAT and plasma levels of TAC and vitamin C as well as serum uric acid, MDA and LDL levels were spectrophotometerically estimated following standard methods using commercial test kits supplied by Biodiagnostics, Cairo, Egypt.

6. Statistical analysis :

Data were statistically analyzed using statistical software program (Graphpad Prism for Windows Version 5.0, GraphPad Software, Inc., San diego, CA, USA). D'Agostino and Pearson omnibus normality test was used to assess normality. Data were normally distributed; therefore, Kruskal-Walis with post hoc Bonferroni multiple comparison tests were used to assess statistical differences between groups. For all statistical examinations, results were considered significant at $p< 0.05$.

RESULTS AND DISCUSSION

Results of the clinical as well as clinicopathological variables of examined horses

were summarized in tables 1, 2, 3 and 4. Based on history and clinical examination, acute (29/90) and chronic (61/90) lower respiratory tract diseases were recorded and diagnosed. Clinically, there was a significant increase of rectal temperature and respiratory rates ($p < 0.05$) in horses with acute lower respiratory tract diseases compared with chronic cases. Nasal discharges varied greatly in the diseased cases depending on the severity of airway inflammation where it was mucopurulent and absent in some cases (23/90). All animals exhibited cough with varying degrees. Tracheal rales were evident in most cases of acute and chronic lower respiratory tract diseases. Lung sounds varied according to the severity and site of airway inflammation where there were normal vesicular sound, exaggerated vesicular sound, crackles, wheezes, and mixed from more than one abnormal lung sounds (Table 1).

Hematologically, there was a significant increase ($p < 0.05$) in total leukocytic counts, band cell and neutrophils percent and a significant decrease ($p < 0.05$) of lymphocyte percent in all diseased cases. Meanwhile, monocyte and eosinophil percent showed a significant increase ($p < 0.05$) in chronic lower respiratory tract diseases only (Table 2).

Regarding antioxidant enzymes and oxidative stress markers; tables 3, 4 showed that there was a significant increase ($p < 0.05$) of GR and SOD activities, uric acid, MDA and LDL in diseased horses compared with healthy ones. Meanwhile, there was a significant decrease ($p < 0.05$) in GST and CAT activities and vitamin C level in diseased horses compared with healthy ones. On the other hand, GSH and TAC showed non-significant changes in both acute and chronic lower respiratory tract diseases.

Horses under investigation were proven to be affected with acute lower and chronic lower respiratory tract diseases. The high incidence rate appeared to be of chronic lower respiratory tract diseases which could be attributed to miss-diagnosis, shortage in treatment protocol, neglected acute cases and sub-clinical form of IAD. These findings were in agreement with those reported by **Hodgson and Hodgson (2002); Radostits et al. (2007) and Smith (2009)**.

The increased rectal temperature could be attributed to the increased cytokines which had not only a pyrogenic effect, but also mediate the acute phase response describing the reaction of the animal to pathogen invasion, tissue injury, immunological reactions and inflammatory processes in agreement with those reported by **Radostits et al. (2007)**. Fever, airway inflammation, airway stenosis and the amount of the formed exudate inside the respiratory passage have been found to cause an increase of both respiratory and heart rate **(Hodgson and Hodgson, 2002; Couëtil, 2007; Radostits et al., 2007 and Smith, 2009)**.

Nasal discharge varied greatly in the affected cases depending on the severity of airway inflammation where it was mucopurulent or absent. It was recorded that most cases suffered mucopurulent nasal discharge indicating more severe form of inflammation. These findings were in agreement with those reported by **Couëtil (2007) and Radostits et al.**

(2007). Moist cough was more prevalent than dry cough, suggesting long standing inflammation of the airway which could be attributed to the airway inflammation, airway stenosis and increased airway secretion as those previously reported by **Hodgson and Hodgson (2002)**. Tracheal rales appeared to be more evident in horses suffered acute and chronic lower respiratory tract diseases which could be attributed to tracheal inflammation and accumulation of exudate inside the tracheal lumen in agreement with those stated by **Radostits et al. (2007) and McGorum et al. (2007)**. Lung sounds varied according to the severity and site of airway inflammation where there were exaggerated vesicular sound resulted from pulmonary congestion in the early stages of lower air way inflammation; whereas, crackles sound could be due to increase in the bronchiolar exudation; however, wheezes might be audible due to presence of bronchiolitis, stenosis of respiratory tract by dried exudate especially in chronic form of pulmonary inflammation. These findings were in agreement with those reported by **Radostits et al. (2007); Couëtil (2007); McGorum et al. (2007) and Smith (2009)**.

The results of hematological examination reflected a state of respiratory tract infection as leukocytosis can be an appropriate physiological response to an infectious or inflammatory process as previously reported by **Radostits et al. (2007)**.

GR activity was increased during oxidative stress as GR restores GSH by reduction of glutathione disulfide through the use of NADH. Thus, the increase in GR activity suggests a response to oxidative stress as previously reported by **Frankiewicz-Jozko and Szarska (2000)**. Oxidative stress is supported by the simultaneous decrease in antioxidant enzymes, GST, which are in accordance with earlier reports previously reported by **Kumar and Naidu (2002) and Muñoz-Escassi et al. (2006)**. In contrast, **Somani (1996) and De Haan et al. (1998)** found higher GST activity in cases of oxidative stress associated with increased lipid peroxides.

The increased SOD activity could be attributed to the compensatory response against increased oxidative stress to counteract the effect of free radicals, and also despite an adequate antioxidant capacity (enzymatic and non-enzymatic); a marked increase in ROS production may overwhelm the antioxidant system with subsequent development of oxidative stress in agreement with those reported by **Frankiewicz-Jozko and Szarska (2000) and Deaton and Marlin (2003)**. Meanwhile, the decreased CAT activity could be attributed to the oxidative stress and the reduction of antioxidant enzymes. These findings were similar to those previously mentioned by **Deaton and Marlin (2003)** who decided that the worst case scenario of oxidative stress was both a reduction in antioxidant capacity combined with increases in ROS production that might happen in certain disease conditions such as human asthma or equine RAO which induce chronic reduction in antioxidant defenses and dramatically increased ROS production.

Vitamin C level was found to be decreased as a result of oxidation of ascorbic acid during oxidative stress as activated neutrophils influenced the ascorbic acid pool by inducing oxi-

dation of it via the production of ROS during inflammation. Moreover, macrophages influenced the plasma ascorbic acid level as they activate and increase the accumulation of it, so there was a positive correlation between number of macrophages and ascorbic acid level. During inflammation; macrophages number was decreased accordingly, resulted in decreased ascorbic acid level. Consequently, the decrease in vitamin C level could be attributed to the increased utilization of ascorbic acid or accumulation by neutrophils and macrophages protecting themselves from its own respiratory burst. These findings were in agreement with those reported by **Hargreaves et al. (2002) and Deaton et al. (2004).** Meanwhile, uric acid is increased with enhanced ROS generation (oxidative stress) and also, could be attributed to the impaired lung function and increased hypoxic states occurred in respiratory tract diseases which resulted in an increased reliance on anaerobic glycolysis with subsequent increase lactate level which had been shown to result in ATP loss and uric acid accumulation. These findings were in agreement with those reported by **De Moffarts et al. (2006)**.

MDA level was increased due to the oxidative damage resulting from the free radicals produced. These findings were in harmony with those reported by **White et al. (2001) and Williams et al. (2005)**. Also, LDL level was increased due to the fact that LDL oxidation might decrease the catabolism of LDL cholesterol, thus causing increase in LDL cholesterol levels. These findings were similar to those reported by **Erciyas et al. (2004)**.

This study revealed synchronous changes in both the activity of antioxidant enzymes and oxidative tress markers, suggesting the roles of their changes in draft horses with lower airway diseases. Further investigations need to be done on the clinical efficacy of administration of antioxidants in horses with acute and chronic lower airway diseases.

Table 1. Clinical Findings in Horses with Acute and Chronic Inflammatory Airway Diseases.

Groups	Temperature T°C	R.R. Cycle/Min.	H.R. Beat/Min.	Nasal discharge	Cough	Tracheal sound	Lung sound
Control (*n* = 20)	37.5 ± 0.5	12 ± 2	35 ± 5	-	-	Normal CH sound	Normal vesicular sound
Acute Lower (*n* = 29)	39.82 ± 1.10*	27 ± 7*	41 ± 4	Mucopurulent (21/29) Absent (8/29)	Dry (5/29) Moist (24/29)	Tracheal rales (29/29)	Crackles (19/29) Wheezes (2/29) Exaggerated vesicular sound (4/29) Mixed (4/29)
Chronic Lower (*n* = 61)	38.15 ± 0.71	22 ± 6	42 ± 7	Mucopurulent (46/61) Absent (15/61)	Dry (36/61) Moist (25/61)	Tracheal rales (50/61) Normal (11/61)	Wheezes (22/61) Crackles (20/61) Exaggerated vesicular sound (3/61) Mixed (16/61)

*, **: Variables with different superscript in the same column are significantly different at P < 0.05.
R.R. = Respiratory rate; H.R. = Heart rate.

Table 2. Total and Differential Leukocytic Counts (mean values ± SD) in Clinically Healthy Horses and in those with Acute and Chronic Inflammatory Airway Diseases.

Groups	T.L.C x 10^3	Band cell %	Neutrophil %	Lymphocyte %	Monocyte %	Eosinophil %	Basophil %
Control (*n*=20)	9.1 ± 2.9	1.2 ± 0.9	40 ± 11.6	44 ± 13.5	4.4 ± 2.8	4.4 ± 2.8	1.5 ± 1.2
Acute Lower (*n* = 29)	15.4 ± 2.2*	11.3 ± 4.4*	54.3 ± 11.9*	22.1 ± 12.8*	4.63 ± 2.9	6.8 ± 3.7	1.0 ± 1.0
Chronic Lower (*n* = 61)	14.9 ± 2.2*	10.4 ± 3.8*	48.2 ± 12.0	21.2 ± 7.6*	5.0 ± 3.5*	14.0 ± 4.3*	1.2 ± 0.6

*, **: Variables with different superscript in the same column are significantly different at P < 0.05.
T.L.C. = Total leukocytic count.

Table 3 : Different Antioxidants and Other Oxidative Stress Markers (mean values ± SD) in Clinically Healthy Horses and in Those with Acute and Chronic Lower Respiratory Tract Diseases.

Groups	GSH (mg/dL)	GR (U/L)	GST (U/L)	SOD (U/L)	CAT (U/L)
Control *(n = 20)*	2.34 ± 1.84	1442 ± 132	256 ± 72.6	161 ± 22.9	1598 ± 277
Acute lower *(n = 29)*	2.96 ± 1.5	1645 ± 244	190 ± 63.6	206 ± 52.2	938 ± 384
Chronic lower *(n = 61)*	2.11 ± 1.33	1667 ± 228	163 ± 89.2	318 ± 66.3	509 ± 225

*, **: Variables with different superscript in the same column are significantly different at P < 0.05.
GSH; Reduced glutathione. GR; Glutathione reductase, GST; Glutathione-S-transferase, SOD; Superoxide dismutase, CAT; Catalase

Table 4. Mean values ± SD) of TAC, Vitamin C, Uric acid, MDA and LDL in Clinically Healthy Horses and in Those with Acute and Chronic Lower Respiratory Tract Diseases

Groups	TAC (mmol/L)	Vitamin C (mg/L)	Uric acid (mg/dL)	MDA (nmol/mL)	LDL (mg/dL)
Control *(n = 20)*	0.59 ± 0.33	95 ± 27	1.00 ± 0.73	8.7 ± 2.5	21.6 ± 9.8
Acute lower *(n = 29)*	0.65 ± 0.38	71 ± 10*	2.65 ± 1.42*	15.1 ± 3.7*	114 ± 47.2*
Chronic lower *(n = 61)*	0.53 ± 0.33	64 ± 19*	1.99 ± 1.15	32.5 ± 6.9**	139 ± 36.4**

*, **: Variables with different superscript in the same column are significantly different at P < 0.05.
TAC; Total antioxidant capacity, MDA; Malondialdehyde, LDL; Low density lipoprotein

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الملخص العربى

حالة انزيمات مضادات الأكسدة ومؤشرات الأكسدة الدالة على وجود الأكسدة فى الخيول المصابة بأمراض الجهاز التنفسى السفلى الحادة والمزمنة

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قسم الأمراض الباطنية والأمراض المعدية والأسماك

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كان الهدف من هذه الدراسة تقييم مستوى الأكسدة والانزيمات المضادة للأكسدة فى حالات اضطرابات الجهاز التنفسى السفلى الحادة والمزمنة فى الخيول، لهذا الغرض تم الحصول على عينات من الدم الوريدى من عدد (٩٠) من الخيول تم تشخيصها إكلينيكياً وإصابتها باضطرابات الجهاز التنفسى السفلى الحادة والمزمنة وعدد (٢٠) من الخيول السليمة اكلينيكياً وتم اعتبارها كمجموعة ضابطة تم قياس نشاط انزيم الجلوتاثيون المختزل، الجلوتاثيون ريدكتيز، الجلوتاثيون - اس - ترانسفيريز، السوبر أوكسيد ديسموتيز والكاتاليز فى بلازما الدم، وكذلك تم قياس ڤيتامين (ج) واجمالى القدرة المضادة للأكسدة فى بلازما الدم، كما تم قياس مستويات حامض اليوريك والمالوندای ألدهيد ومستويات البروتين الدهنى منخفض الكثافة فى مصل الدم، وجد أن هناك زيادة معنوية فى نشاط انزيم الجلوتاثيون ريدكتيز والسوبر أوكسيد ديسمبوتيز ومستويات حمض اليوريك والمالوندای ألدهيد والبروتين الدهنى منخفض الكثافة فى الخيول المصابة باضطرابات الجهاز التنفسى السفلى الحادة والمزمنة مقارنة مع المجموعة الضابطة، وعلى النقيض فقد لوحظ انخفاض معنوى فى نشاط انزيم الكاتاليز والجلوتاثيون - اس - ترانسيفيريز ومستوى ڤيتامين (ج) فى الخيول المصابة مقارنة مع المجموعة الضابطة وبالتقييم العلمى للنتائج المتحصل عليها من خلال هذه الدراسة تأكد أن الأكسدة المصاحبة مع تغيير نشاط الانزيمات المضادة للأكسدة تعتبر سمة مميزة لأمراض الجهاز التنفسى السفلى فى الخيول والتى يمكن الاعتماد عليها تشخيصياً وعلاجياً فى المستقبل.