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Vitamin E Ameliorates the Mineralo-Oxidative Stress of Sucking Lice Infestation in Indian Water Buffalo

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A B S T R A C T

Bubaline pediculosis is a common and economically important ectoparasitic condition caused by sucking lice, *Haematopinus tuberculatus.* This condition in buffalo is characterized by anemia, mineral imbalance, and loss of production performance but its pathobiology is explored less. Aim of the present study was to explore the mineralo-oxidaive pathobiology of bubaline pediculosis and its amelioration using the two antioxidants as an adjunct therapeutic modality (N- acetyl cysteine and Vitamin E). Twenty four buffaloes, severely infested with sucking lice (mean total lice count more than 100) were divided into three groups (Group I, II and III) with eight animals per group; another eight animals (Group IV) free from any clinical abnormalities and ectoparasitism were included as healthy control. Lice infested animals (Group I, II and III) animals were given ivermectin therapy (200 µg/kg body weight SC single dose); additionally, group II and group III animals were orally treated with N-acetyl cysteine (12 mg/kg body weight) and vitamin E (Tocopheryl acetate 4000 mg per animal), respectively, once daily for 14 days. The haematological parameters (TEC, Hb, TLC and DLC), oxidant-antioxidant profile (TAC, LPO, GSH, and SOD) and mineral profile (Zinc, Iron, Magnesium, and Copper) were studied on before therapy (Day 0) and post therapy (Day 28). Bubaline pediculosis revealed severe oxidative stress and mineral imbalance along with remarkable anaemia and leukocytosis. Vitamin E given animals demonstrated better recovery from the mineralo-oxidative pathology of bubaline pediculosis and approached post therapeutic normalcy. Vitamin E as an adjunct therapy along with ivermectin alleviated the patho-biological damages in the host system and speed up the clinical recovery, whereas, N-acetyl cysteine was less effective in quenching the mineralo-oxidative response. Future studies on antioxidants therapy may target the influence of mineralo-oxidative response in the host system.

Introduction

Buffaloes considered as “black gold of India”; because buffaloes provide high quality protein, fat rich milk, serve as a source of employment and livelihoods to millions of landless, marginal, and small farmers (Syed Mohmad and Manmohan Singh, 2017).

Ectoparasitism adversely affects the welfare and production performance farm animals. Worldwide buffaloes were commonly infested with sucking lice, *Haematopinus tuberculatus* especially during the winter season can affect cattle also (Bastianetto and Leite, 2005; Egri, 2019; Mamun *et al.,* 2010). Lice infestation in the animal was favored by the risk factors like lack of hygiene, low health status, low temperature-humid weather condition (Taylor *et al.,* 2016). Sucking lice are sebaceous secretion feeders and bloodsuckers, which are less movable and remain tightly attached to the skin of host for a prolonged time. Lice induce hypersensitivity hence, severe *H.tuberculatus* infestations can result in varying levels of scratching, self-excoriation, alopecia, and papulo - crustous dermatitis (Chaudhry, 1978; Egri, 2019; Taylor *et al.,* 2016). Recent findings revealed that inflammation and reactive oxygen species (ROS) induced tissue damage to play an important role in ectoparasitism induced deprivation of host health (Dimri *et al.,* 2010).

Oxidative stress is defined as an imbalance between the production of the oxidants or free radicals and the ability of the body to quenching of their harmful effects through anti-oxidant defense system. The free radicals readily indices oxidative damage to various biomolecules including proteins, lipoprotein, lipids, and DNA and cause a disturbance in normal cellular signaling pathways and homeostasis (Farber, 1994; Kaur *et al.,* 2006; Lykkesfeldt and Svendsen, 2007). Even, ectoparasiticides like cypermethrin, deltamethrin, and flumethrin usage in rats induce a significant oxidative stress (Dubey *et al.,* 2013; Ince *et al.,* 2013; Kanbur *et al.,* 2010). The biomodulators use in the therapeutic management of various diseases is getting universal acceptance because of their vast scope in amelioration of a wide range of diseases conditions by altering the oxidative status. Generally, antioxidants are reducing agents belonging to the class such as thiols, ascorbates and polyphenols that can neutralize ROS and thereby inhibits the oxidation of other biomolecules. The recent finding suggests that antioxidants play a role in immunomodulation (Ajith *et al.,* 2017). N-Acetyl Cysteine (NAC) has been used for the treatment of acute and chronic bronchitis as a mucolytic agent for many years and is an antidote for acetaminophen poisoning. Fat-soluble vitamin E has an antioxidant property through which it can protect the polyunsaturated fatty acids (PUFAs) of the cell membrane from biological oxidation, regulate the production of reactive oxygen species (ROS) and modulate the signal transduction (Lee and Han, 2018).

Previous studies revealed that Vitamin E and N-acetyl cysteine have good antioxidant with immunomodulatory properties (Dekhuijzen, 2004; Lee and Han, 2018). However, studies on the evaluation of these compounds in the management of mineralo-oxidative damage in bubaline pediculosis are not available. Hence, the focus of the current study was to examine the antioxidant potential of Vitamin E and N-acetylcysteine in the management of pathobiology of bubaline pediculosis.

Materials and Methods

Experimental design

A total of twenty four Indian water buffaloes with “Severe” (cumulative count more than 100) sucking lice infestation were divided into three groups and another eight apparently healthy buffaloes without any parasitic infestation belonging to the 2-6 years age group was kept as healthy control. The severity of sucking lice infestation was carried out by summing of lice counted from different predilection sites; A-cheek (5×10 cm area), ear (5×10 cm area), C-neck and dewlap

(10×20 cm area), D-withers (10×10 cm area), E-foreleg (10×10 cm area), F-back (10×10 cm area), G-hind leg (10×10 cm area), H-tail head and perineum (10×10 cm area) using standard counting technique (Holdsworth *et al.,* 2006; Veneziano *et al.,* 2013, 2003). The *Haematopinus tuberculatus* infested buffaloes (Soulsby, 1982) were allotted three treatment plan; group I (Ivermectin injection @ 200 µg/kg SC single dose), group II (Ivermectin injection @ 200 µg/kg SC single dose and N-acetyl cysteine @ 12 mg/kg SID PO for 14 days) and group III (Ivermectin injection @ 200 µg/kg SC single dose and Vitamin E 4000 mg per animal SID PO for 14 days). The remaining eight healthy animals without any parasitic infestation served as healthy control (group IV). Commercial preparations of Ivermectin (Hitek® injection, 1% w/v; Virbac, India), Vitamin E (Evion® capsule, 400 mg; Merck, India) N-acetyl cysteine (Fluimucil® Tablet, 600 mg; Elder Pharma, India) were used. WAAVP guidelines for evaluation of the ectoparasiticide efficacy was used for clinical evaluation of the different treatment plan (Holdsworth *et al.,* 2006) and was assessed by calculating reduction percentage and assessment of mineralo-oxidative status (Veneziano *et al.,* 2013).

Sample collection and processing

Animals were evaluated for changes in haematological, oxidant-antioxidant and mineral profile on before the start of therapy (day 0) and post therapy (day 28) of the experiment. Blood samples (12 mL) were collected by external jugular venipuncture in sterile EDTA and heparin coated vials.

Serum samples were collected in sterile vials and stored in a deep freezer for the estimation of mineral profile. About two milliliter of blood collected in sterile EDTA coated vials were utilized for haematological analysis. For estimation of oxidative stress parameters 6 mL of blood collected in heparin coated vial was utilized.

Estimation of catalase (CAT), lipid peroxidation (LPO), superoxide dismutase (SOD) were carried out in the hemolysate obtained from the 6 mL of the heparinized blood sample and whereas, reduced glutathione (GSH) estimation was carried out in RBC suspension obtained from the above blood sample. The cyanohemoglobin method described by Tentori and Salvati, (1981) was used for estimation of hemolysate haemoglobin concentration.

Evaluation of oxidant and anti-oxidant profile

The lipid peroxidation (LPO) levels were estimated as per the method described by Placer *et al.,* (1966). DTNB method of Prins and Loos was used for the estimation of reduced glutathione (GSH) concentration in RBCs (Prins and Loos, 1969).

Estimation of superoxide dismutase (SOD) activity was carried out as per the method described by Madesh and Balasubramanian (1998). The method described by Aebi (1974) was used for estimation of Catalase (CAT) activity. Estimation of serum Total antioxidant capacity was carried out as per the manufacturer's instruction using Total Antioxidant Capacity (TAC) assay kit (Sigma-Aldrich, USA) (Miller and Rice-Evans, 1997).

Evaluation of hematological profile

The method decribed by (Berman, 1919) was used for the estimation of haemoglobin concentration (gm/dl). Haematological parameters like TLC, TEC, and DLC were estimated as per the method described by (Schalm and Jain, 1986).

Statistical analysis

The data were analyzed by two-way ANOVA with Tukey's post hoc test using IBM SPSS statistics package version 25.0 (Snecdecor and Cochran, 1994). The values were expressed as mean ± S.E.

Results and Discussion

The mean lice count in different body regions for different treatment groups and healthy control on day 0 and day 28 is depicted in table 1. The sucking lice of buffalo were mostly concentrated on withers, back and neck and dewlap regions, followed by cheek, foreleg, hind leg, tail head and perineum, which is not available for animal's self-grooming. The lice infestation was clinically manifested as dermatological lesions related to weakness, hyper-sensitivity reaction, pruritus, alopecia, and seborrhea. However, lice infestation was reduced significantly in all three treated groups on day 28, group III showed better clinical improvement of clinical signs and skin condition.

The hematological profile of sucking lice infested buffaloes had significant ($p<0.05$) anaemia and leukocytosis with neutrophilia, lymphocytopenia, eosinophilia (Table 2). Remarkable improvement of haematological profile (Hb, TEC, TLC, and DLC) by day 28 was observed in all three treatment groups, but, group III values were similar to the healthy control group. Lice infested animals showed significant oxidative stress, with significantly elevated oxidant (LPO) levels and decreased serum TAC, SOD, reduced GSH level, and CAT (Table 3). Moreover, cell membrane lipids oxidative damage biomarker malondialdehyde was significantly ($p<0.05$) reduced in the three treatment groups on day 28 and group II and group III of the treated animals value were closely comparable to the healthy control group. All the treated groups revealed significant improvement of their antioxidant defense statuses like serum TAC, SOD activity, CAT activity and reduced glutathione on day 28, but, the group II and group III showed remarkable improvement closer to the healthy control group.

The mineral profile of lice infested animal revealed a significantly ($p<0.05$) decreased serum iron, zinc, magnesium (Table 4). However, serum copper levels did not differ significantly between infested and healthy control animals and all the three treatments did not alter serum copper levels much. Group 11 and group III of the treated animals iron, zinc, magnesium levels were improved on day 28 and values were almost similar to the healthy control.

In the present study, sucking lice infested buffaloes were found to have marked oxidative stress, severe mineral imbalance, iron deficiency anaemia, leukocytosis with neutrophilia, eosinophilia and lymphocytopenia. Similar mineral imbalance, oxidative stress and hematological changes were observed in other ectoparasitism like ovine pediculosis (Dede *et al.,* 2002), caprine pediculosis (Ajith *et al.,* 2017), bubaline pediculosis(El-Moghazy, 2011), demodicosis in dogs (Dimri *et al.,* 2008), psoroptic mange infection in sheep (Aktas *et al.,* 2017), sarcoptic mange in goats (De and Dey, 2010), dogs (Behera *et al.,* 2011; Singh and Dimri, 2013) and buffaloes (Dimri *et al.,* 2007). Accruing evidence from recent studies is promising the potential use of antioxidants in prophylactic and therapeutic management of the infectious disease. The ameliorative potential of the antioxidants in management of parasitic disease is increasing dramatically, Vitamin C in caprine pediculosis (Ajith *et al.,* 2019), Vitamin E-Selenium for sarcoptic mange in canines (Behera *et al.,* 2011; Singh and Dimri, 2013), Vitamin ADEH for

psoroptic in rabbit (Singh *et al.*, 2012) and herbal essential oils for sarcoptic mange infestation in sheep (Dimri and Sharma, 2004).

N-Acetyl cysteine (NAC), a mucolytic agent, has been used for the treatment of acute and chronic bronchitis in patients for many years and also used as an antidote for acetaminophen toxicity. NAC has been used as a possible chemo-preventive agent is due to its anti-oxidative or detoxifying properties (De Vries and De Flora, 1993). NAC exhibits both direct and indirect antioxidant properties; acts directly by interaction with free electrophilic thiol groups of ROS (Dekhuijzen, 2004). NAC acts indirectly by increasing the intracellular concentrations of cysteine, GSH and oxidant species scavenging. NAC administered orally for seven days in milking goats had quenched the effects of oxidative stress (Jóźwik *et al.*, 2010). Vitamin E, the part of the chain-breaking antioxidant system, scavenges the free radicals and prevents the oxidation of membrane lipids (McDowell, 2000). Vitamin E prevents the peroxidation of phospholipids by acting as the first line of defense against membrane peroxidation. In the present study Vitamin E treated animals had a marked improvement of antioxidant and mineral status compared to N-acetyl cysteine treated animals might be due to NAC mediated activation of Th2 response in lice infestation (Ajith *et al.*, 2019). Vitamin E is an effective antioxidant with anti-inflammatory property might help in improvement of general body condition, mineral balance and oxidative stress in sucking lice infested buffaloes (Tahan *et al.*, 2011).

Table.1 Mean lice count in different body regions of sucking lice (*Haematopinus tuberculatus*) infested treatment groups on day 0 and day 28

Body Region	Group I(n=8)		Group II (n=8)		Group III (n=8)		Group IV (n=8)	
	Day 0	Day 28	Day 0	Day 28	Day 0	Day 28	Day 0	Day 28
Cheek	10.86	0.25	10.78	0.33	12.58	0	0	0
Ear	2.27	0	2.47	0	2.17	0	0	0
Neck and dewlap	30.52	0.78	30.82	0.54	30.42	0	0	0
Withers	91.68	1.63	92.27	1.43	96.67	0	0	0
Foreleg	8.18	0	8.64	0	8.08	0	0	0
Back	25.22	0.53	24.78	0.45	25.58	0	0	0
Hind leg	2.28	0	2.58	0	2.08	0	0	0
Tail head and perineum	4.77	0	6.12	0	4.17	0	0	0
Cumulative count (Mean)	175.78	3.19	178.46	2.75	181.75	0	0	0
Reduction percentage*	**98.18**		**99.01**		**100**		**-**	

* Calculated by the formula

$$Reduction\ percentage = \frac{lice\ count\ before\ treatment - Lice\ count\ after\ treatment}{Lice\ count\ before\ treatment} \times 100s$$

Table.2 Haematological profile of lice infested goats (Mean ± SE)

Parameter	Day 0	Day 28
Hemoglobin concentration (gm/dl)		
Group I (n=8)	9.02 ± 0.28^{Bb}	12.22 ± 0.23^{Ca}
Group II (n=8)	9.05 ± 0.26^{Bb}	13.52 ± 0.26^{Ba}
Group III (n=8)	9.07 ± 0.25^{Bb}	15.68 ± 0.24^{Aa}
Group IV (n=8)	15.65 ± 0.35^{Aa}	15.72 ± 0.32^{Aa}
Total Erythrocyte count-TEC (million cells/µL)		
Group I (n=8)	3.52 ± 0.24^{Bb}	4.46 ± 0.12^{Ca}
Group II (n=8)	3.63 ± 0.23^{Bb}	6.52 ± 0.13^{Ba}
Group III (n=8)	3.56 ± 0.18^{Bb}	6.58 ± 0.15^{Ba}
Group IV (n=8)	6.59 ± 0.14^{Aa}	6.60 ± 0.12^{Aa}
Total Leukocyte count-TLC (Thousand cells/ µL)		
Group I (n=8)	16.08 ± 0.25^{Ab}	14.63±0.26Aa
Group II (n=8)	16.04 ± 0.22^{Ab}	12.56 ± 0.24^{Ba}
Group III (n=8)	16.07 ± 0.23^{Ab}	12.25 ± 0.22^{Ba}
Group IV (n=8)	11.58 ± 0.22^{Ba}	11.56 ± 0.23^{Ca}
Differential Leukocyte Count-DLC (%)		
Neutrophil (%)		
Group I (n=8)	45.43 ± 0.52^{Ab}	42.43 ± 0.53^{Aa}
Group II (n=8)	44.73 ± 0.52^{Ab}	40.24 ± 0.46^{Ba}
Group III (n=8)	45.43 ± 0.52^{Ab}	38.07 ± 0.55^{Ca}
Group IV (n=8)	37.58 ± 0.38^{Ba}	37.78 ± 0.36^{Ca}
Lymphocyte (%)		
Group I (n=8)	42.11 ± 0.68^{Bb}	52.58 ± 0.45^{Ca}
Group II (n=8)	42.28 ± 0.62^{Bb}	55.52 ± 0.38^{Ba}
Group III (n=8)	42.18 ± 0.63^{Bb}	58.54 ± 0.42^{Aa}
Group IV (n=8)	59.13 ± 1.03^{Aa}	59.13 ± 1.03^{Aa}
Eosinophil (%)		
Group I (n=8)	6.08 ± 0.42^{Aa}	4.58 ± 0.40^{Ab}
Group II (n=8)	6.08 ± 0.42^{Aa}	2.35 ± 0.38^{Bb}
Group III (n=8)	6.08 ± 0.42^{Aa}	1.56 ± 0.42^{Cb}
Group IV (n=8)	1.42 ± 0.19^{Ba}	1.44 ± 0.22^{Ca}
Monocyte (%)		
Group I (n=8)	2.18 ± 0.22^{Aa}	1.42 ± 0.18^{Ab}
Group II (n=8)	2.15 ± 0.23^{Aa}	1.32 ± 0.12^{Bb}
Group III (n=8)	2.16 ± 0.20^{Aa}	1.26 ± 0.16^{Ca}
Group IV (n=8)	1.25 ± 0.13^{Ba}	1.25 ± 0.15^{Ca}
Basophil (%)		
Group I (n=8)	1.08 ± 0.15^{Aa}	0.92 ± 0.18^{Aa}
Group II (n=8)	0.92 ± 0.15^{Aa}	0.83 ± 0.21^{Aa}
Group III (n=8)	0.96 ± 0.13^{Aa}	0.67 ± 0.14^{Aa}
Group IV (n=8)	0.7 ± 0.15^{Aa}	0.7 ± 0.19^{Aa}

Values with different superscripts A, B, C, D differ significantly ($p < 0.05$) in the same column
Values with different superscripts a, b differ significantly ($p < 0.05$) in the same row

Table.3 Variation in oxidant-antioxidant profile of treatment groups on day 0 and day 28 (Mean±SE)

Parameter	Day 0	Day 28
Lipid per oxidation-LPO (nM MDA/mg Hb)		
Group I (n=8)	8.26 ± 0.12^{Aa}	7.58 ± 0.16^{Ab}
Group II (n=8)	8.34 ± 0.18^{Aa}	6.62 ± 0.15^{Bb}
Group III (n=8)	8.56 ± 0.16^{Aa}	6.52 ± 0.13^{Bb}
Group IV (n=8)	6.36 ± 0.18^{Ba}	6.42 ± 0.15^{Ba}
Reduced glutathione-GSH (µMol/ml of packed RBC)		
Group I (n=8)	0.63 ± 0.04^{Bb}	0.68 ± 0.02^{Ca}
Group II (n=8)	0.65 ± 0.03^{Bb}	0.78 ± 0.04^{Ba}
Group III (n=8)	0.64 ± 0.02^{Bb}	0.81 ± 0.03^{Ba}
Group IV (n=8)	0.84 ± 0.03^{Aa}	0.85 ± 0.04^{Aa}
Super Oxide Dismutase-SOD (µmol/mg Hb)		
Group I (n=8)	8.06 ± 0.25^{Bb}	9.28 ± 0.18^{Ca}
Group II (n=8)	8.07 ± 0.26^{Bb}	9.68 ± 0.24^{Ba}
Group III (n=8)	8.08 ± 0.27^{Bb}	9.75 ± 0.25^{Ba}
Group IV (n=8)	10.22 ± 0.16^{Aa}	10.19 ± 0.14^{Aa}
Catalase activity-CAT (µmol H_2O_2 decomposed/min/mg Hb)		
Group I (n=8)	4.69 ± 0.12^{Bb}	6.12 ± 0.12^{Ca}
Group II (n=8)	4.73 ± 0.15^{Bb}	6.75 ± 0.14^{Ba}
Group III (n=8)	4.75 ± 0.14^{Bb}	6.79 ± 0.14^{Ba}
Group IV (n=8)	6.85 ± 0.23^{Aa}	6.82 ± 0.25^{Aa}
Total Antioxidant Capacity-TAC (mM/L of serum)		
Group I (n=8)	0.80 ± 0.23^{Bb}	1.25 ± 0.17^{Ba}
Group II (n=8)	0.81 ± 0.22^{Bb}	1.73 ± 0.13^{Aa}
Group III (n=8)	0.82 ± 0.26^{Bb}	1.76 ± 0.15^{Aa}
Group IV (n=8)	1.78 ± 0.08^{Aa}	1.79 ± 0.10^{Aa}

Values with different superscripts A, B, C, D differ significantly ($p < 0.05$) in the same Column
Values with different superscripts a, b differ significantly ($p < 0.05$) in the same row

Table.4 Mineral profile of lice infested buffaloes (Mean ± SE)

Parameter	Day 0	Day 28
Serum Iron (µg/dL)		
Group I (n=8)	113.30±1.23^{Ba}	145.23±1.12^{Ca}
Group II (n=8)	115.25±1.22^{Ba}	155.44±1.16^{Ba}
Group III (n=8)	114.28±1.25^{Ba}	158.58±1.13^{Ba}
Group IV (n=8)	158.09±2.30^{Aa}	160.09±2.35^{Aa}
Serum Zinc (µg/dL)		
Group I (n=8)	97.28±8.42^{Bb}	122.32±3.14^{Ca}
Group II (n=8)	96.30±8.46^{Bb}	136.22±3.16^{Ba}
Group III (n=8)	98.32±8.48^{Bb}	139.36±3.21^{Ba}
Group IV (n=8)	143.52±1.61^{Aa}	143.48±1.53^{Aa}
Serum Copper (µg/dL)		
Group I (n=8)	68.65±5.43^{Aa}	73.65±1.23^{Aa}
Group II (n=8)	68.58±5.42^{Aa}	72.45±1.28^{Aa}
Group III (n=8)	68.53±5.42^{Aa}	74.45±1.23^{Aa}
Group IV (n=8)	75.33±1.28^{Aa}	75.38±1.23^{Aa}
Serum Magnesium (mEq/L)		
Group I (n=8)	0.52±0.03^{Bb}	1.45±0.06^{Ba}
Group II (n=8)	0.54±0.02^{Bb}	1.63±0.05^{Aa}
Group III (n=8)	0.52±0.05^{Bb}	1.65±0.03^{Aa}
Group IV (n=8)	1.66±0.09^{Aa}	1.67±0.07^{Aa}

Values with different superscripts A, B, C, D differ significantly ($p < 0.05$) in the same column
Values with different superscripts a, b differ significantly ($p < 0.05$) in the same row

The proper functioning of the antioxidant defense needs several trace minerals, thus mineral imbalance contributes to the development of oxidative stress. Magnesium act as an antioxidant by participating as a cofactor for several enzymes, maintaining the stability of cell membranes and mitigating the effects of oxidative stress (Morais *et al.*, 2017). Magnesium deficiency enhances the generation of ROS, free radicals and increases the substrates available for radical oxidation (Zheltova *et al.*, 2016). The normal functioning of the enzymes involved in antioxidant defense system requires zinc as a cofactor and zinc also protects cells by stabilization of cell membranes, inhibition of nicotinamide adenine dinucleotide phosphate oxidase (NADPH-Oxidase) enzyme and synthesis of metallothionein proteins that are effective in sequestration of ROS and reduction of hydroxyl radicals (Chasapis *et al.*, 2012; Ruz *et al.*, 2013).

Increased oxidative stress has been associated with magnesium and zinc deficiencies; magnesium deficiency increases ROS level and simultaneously decreasing the expression of antioxidant enzyme whereas zinc plays an indirect antioxidant role by serving as a structural cofactor and essential catalytic for superoxide dismutase and other enzymes (Eide, 2011). For Cu–Zn superoxide dismutase, the antioxidant enzyme, both zinc and copper are act as essential components.

Therefore, utilization or sequestration of Zn and Cu to neutralize the excessively produced ROS and the general poor condition of affected animals might be responsible for their lower level in the blood of pediculosis affected animals (Dede *et al.,* 2002; Dimri *et al.,* 2010). Anaemia in sucking lice infested buffaloes might have resulted from prolonged blood loss and iron deficiency. The loss of essential nutrients, iron deficiency and oxidative stress induced erythrocyte damage might have attributed to anaemia of the lice infested buffaloes. For iron deficiency anaemia development, oxidative stress plays a crucial role (Yoo *et al.,* 2009).

In conclusion, ubaline pediculosis is associated with severe anaemia, oxidative stress, mineral imbalance and leukocytosis with neutrophilia, lymphocytopenia, and eosinophilia. N-Acetyl cysteine as an adjunct antioxidant is less effective than Vitamin E in reducing mineralo-oxidative pathology of bubaline pediculosis. Future studies on antioxidants therapy may target the influence of mineralo-oxidative response in the host system.

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