Effect of *Lepidium sativum* L. (Garden Cress) Seed and Its Extract on Experimental *Eimeria tenella* Infection in Broiler Chickens

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**ABSTRACT**

The anticoccidial effects of *Lepidium sativum* (LS) seed were assessed on 240, male, broiler chickens aged 1 d following oral inoculation of $1 \times 10^5$ sporulated *E. tenella* oocysts. The experimental study was conducted at the Debre Zeit Agricultural Research Center Poultry Farm. LS was included in the feed of the chickens in the form of seed powder, whole seed or extract. Broiler chickens were assigned to eight treatments: noninfected and unsupplemented (control check), infected and unsupplemented (control), infected and supplemented with seed powder, whole seed, extract or amprolium and noninfected and supplemented with seed powder or whole seed. One g.kg⁻¹ of each seed preparation in feed was applied. Treatments were applied for a period of 2 wk from day 18 of hatching. Treatment efficacy was assessed based on measuring and the analysis of the parameters: 1) performance measures (body weight gain, feed intake, mortality); 2) fecal oocysts measurements; and 3) intestinal lesion scores. The body weight gains of *E. tenella*-challenged chickens fed LS-supplemented diets were significantly ($P < 0.003$) higher than those fed the control diet. Chickens fed the LS-supplemented diet following *E. tenella* infection demonstrated a significantly higher reduction of mortality ($P < 0.0001$), fecal oocyst shed ($P < 0.0007$) and lesion score ($P < 0.0001$) than those fed the unsupplemented or control diet. However, noninfected chickens fed seed powder of the LS-supplemented diet showed lesion scores and mortality. *Lepidium sativum*-supplemented diets demonstrated effective anticoccidial results on *E. tenella*-challenged chickens.

**Keywords:** broiler, experimental *Eimeria tenella* infection, lesion score, *Lepidium sativum*, oocyst

**INTRODUCTION**

Coccidiosis is recognized as the major parasitic disease of poultry and is caused by the apicomplexan protozoan *Eimeria* which seriously impairs the growth and feed utilization of infected animals, resulting in a loss of productivity (McDougal and Fitz-Coy, 2008). Conventional disease control strategies have relied heavily on chemophylaxis and, to a certain extent, live vaccines; combined, these factors inflict tremendous economic losses to the world poultry industry in excess of USD 3 billion annually (Dalloul and Lillemo, 2006). In Ethiopia, the incidence of poultry coccidiosis has been reported to exist in many parts of the country causing losses due to mortality following a severe outbreak; for example, Lobago *et al.* (2005), reported 38.34% losses in the Kombolcha Poultry Multiplication and Research Center while Dinka and Yacob

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(2012) reported 71.7% losses in the Debre Zeit Agricultural Research Center Poultry Farm and the species identified in both places were *E. acervulina*, *E. brunetti*, *E. necatrix* and *E. tenella*.

Antimicrobial compounds produced by microorganisms have been used for decades in poultry diets to increase performance and decrease morbidity, particularly in broiler chickens (McDougald and Fitz-Coy, 2008). However, consumer pressure related to the potential development of antibiotic-resistant bacteria has resulted in the development of nonantibiotic feed additives that may also improve broiler performance (Brenes and Roura, 2010). Nowadays, growth promoting antibiotics are banned in several countries (Casewell et al., 2003). In addition, due to the rapid and continuous development of anticoccidial drug resistance (Chapman, 1998) and the higher costs for medication, there is an increasing interest in the search for alternative products.

*Lepidium sativum* (LS), commonly known as garden cress, belongs to the Brassicaceae family and is an annual erect herbaceous plant, growing up to 20–80 cm. In Europe and many other parts of the world, seedlings of LS are used as a salad or to season salads, because of their pungent taste. In Ethiopia, LS is cultivated for its seeds, which are primarily used medicinally (Jansen, 1981). LS seed possesses varied medicinal properties and is known as a “versatile medicine”; seed has been used in local traditional medicine, for example, in villages around the Debre Libanos monastery and in Butajira district to treat various kinds of human and animal ailments such as diarrhea, dysentery, unidentified gastrointestinal disorders, stomach ache, indigestion, febrile disease and skin disorders (Gedif and Hahn, 2003; Teklehaymanot et al., 2007).

Previous investigations have shown that antioxidant-rich plant extracts and feed diets with high n-3 fatty acids (n-3 FA) have potential benefits in treating cecal coccidiosis in chickens (Allen et al., 1996; 1997). Diwakar et al. (2010) reported that the essential oil derived from LS seed contained tocopherol (a natural antioxidant), carotenoid, oleic acid and α-linolenic acid, while Zia-Ul-Haq et al. (2012) showed that LS extract (Soxhlet extracted), had a good antioxidant capacity that could reduce different types of radicals.

Some studies have shown the protective action of LS seed including an antihypertensive and diuretic effect (Maghrani et al., 2005), hypoglycaemic activity in diabetic rats (Eddouks et al., 2005), improvement in asthmatic attacks (Paranjape and Mehta, 2006) and an inhibitory effect of the LS juice against heterocyclic aromatic amines-induced DNA damage and preneoplastic lesions (Kassie et al., 2002). However, the anticoccidial effects of LS seed against chicken coccidiosis have not been reported. Therefore, the present study aimed to investigate the anticoccidial effect of LS seed against *E. tenella* experimental infection in broiler chickens.

**MATERIALS AND METHODS**

**Experimental birds**

A sample of 240, Hubbard classic, male, broiler chicks aged 1 d were purchased from the Debre Zeit Agricultural Research Center, Ethiopia. Chicks were placed in a brooder house for 18 d. Feed and water were supplemented *ad libitum* without anticoccidial drugs. The starter feed was formulated using maize, wheat bran, soybean, noug cake (*Guizotia abyssinica*), limestone, vitamin premix and salt. All broiler chicks were housed in an intensive deep-litter floor. Chicks were divided by group into separate pens. Experimental chicks were vaccinated against Newcastle and infectious bursal diseases.

**Collection of Lepidium sativum seeds**

*Lepidium sativum* seeds, brownish red in color, were collected during August and September 2011 from the Bench Maji zone, Bebeqa woreda,
southwestern Ethiopia. The seeds were cleaned and prepared as whole seed and powder. Seeds were powdered using a mortar and pestle.

**Ethanols extract preparation using hot continuous Soxhlet extraction method**

Samples of 500 g of LS seed powder were each placed in a thimble made of filter paper and inserted into the wide central tube of the extractor of the Soxhlet apparatus. The solvent (ethanol) was placed in the flask and heated at 78 °C and its vapors condensed in a reflux condenser. The condensed extractant dripped into the thimble containing the crude drug, which was extracted by contact. When the level of the liquid in the chamber had risen to the top of the siphon tube, the liquid contents of the chamber drained off into the flask. This process was continuous and was carried out until a drop of the solvent from the siphon tube did not leave any residue when evaporated (Handa, 2008). One mL aliquots of the filtrate were taken and concentrated. The concentration of LS solution was 27 mg.mL\(^{-1}\) (12.8%). The extract was stored in a refrigerator for 2 wk until used and was mixed with feed before use.

**Isolation and propagation of *Eimeria tenella* oocysts**

*Eimeria tenella* oocysts used in this study were identified by a combination of oocyst size, location in the gut, appearance of the lesions and schizont size (McDougald and Fitz-Coy, 2008). Following evisceration at *post mortem*, the cecal contents were washed into a beaker using tap water and the oocysts were isolated using a flotation procedure (Permin and Hansen, 1998). Oocysts were sporulated by incubating a small amount of feces or concentrated suspension of oocysts in distilled water with one or more percentage potassium dichromate solutions with forced aeration at room temperature for 72 h (Conway and Mckenzie, 2007; Bowman, 2009). The sporulated *E. tenella* oocysts were obtained as described earlier. The sporulated *E. tenella* oocysts were suspended in 2% K\(_2\)Cr\(_2\)O\(_7\) solution and refrigerated at 4 °C until oral administration. The K\(_2\)Cr\(_2\)O\(_7\) solution was removed through centrifugation and the sporulated *E. tenella* oocysts were suspended in distilled water at the time of oral administration.

**Preliminary *in vitro* anti-coccidial efficacy evaluation of *Lepidium sativum* extracts on *Eimeria tenella* oocysts**

To assess the effective dosage, a preliminary anticoccidial efficacy test was conducted by observing the effect of the plant extracts on the sporulation of *E. oocysts*. Various concentrations (500, 1,000 and 1,500 mg.mL\(^{-1}\)) of LS extract in distilled water were incubated with 100 *E. tenella* oocysts at room temperature for 72 h. Oocysts were also incubated in K\(_2\)Cr\(_2\)O\(_7\) solution as a control. Oocysts were monitored for their sporulation for a period of 72 h. The number of sporulated *E. tenella* oocysts was then recorded. The highest sporulation inhibition was observed *in vitro* in the incubation with 1,000 and 1,500 mg.mL\(^{-1}\) concentrations of LS extract.

**Experimental treatments**

The experimental treatments were carried out using a completely randomized design. Two hundred and forty chickens were randomly allotted into eight treatments (n = 30), with three replications per treatment for a period of 2 wk. The experimental study was conducted at the Debre Zeit Agricultural Research Center Poultry Farm. Treatments were applied from day 18 of hatching. The chicks were supplemented with the different treatments throughout the experiment for a period of 2 wk. At age 18 d, chickens in treatments 2, 3, 4, 5 and 6 were orally challenged with 1 × 10^5 sporulated *E. tenella* oocysts in 1mL of distilled water suspension using a calibrated syringe and were monitored daily for the development of clinical coccidiosis and the presence of *Eimeria* oocysts in their feces. Sporulated *E. tenella* oocysts were obtained as described earlier. The sporulated *E. tenella* oocysts were suspended in 2% K\(_2\)Cr\(_2\)O\(_7\) solution and refrigerated at 4 °C until oral administration. The K\(_2\)Cr\(_2\)O\(_7\) solution was removed through centrifugation and the sporulated *E. tenella* oocysts were suspended in distilled water at the time of oral administration.
fed a regular diet. Chickens in treatments 1, 7 and 8 were not challenged. Based on the results of the preliminary anticoccidial efficacy test, 1 g.kg\(^{-1}\) of each seed preparation in feed was applied. Two days after infection, the chickens in treatments 3, 4, 5 and 6 were respectively supplemented with 1 g.kg\(^{-1}\) of seed powder, 1 g.kg\(^{-1}\) of whole seed, 1 g.kg\(^{-1}\) of extract in feed and 0.6 g.L\(^{-1}\) of amprolium in the drinking water. Chickens in treatments 7 and 8 were also respectively supplemented with 1 g.kg\(^{-1}\) of LS seed powder and 1 g.kg\(^{-1}\) of whole seed (Table 1). Chickens were monitored daily for the presence of clinical signs.

**Experimental parameters and data collection**

The efficacy of treatments was assessed on the basis of the following parameters: 1) performance measures (body weight gain, feed intake and mortality); 2) fecal oocyst measurements; and 3) cecal lesion scores. The number of dead chickens was recorded daily until day 7 post challenge. The body weight of all experimental chickens in each group was weighed twice—on day 18 (before challenge) and day 7 post challenge. The feed intake of all experimental chickens in each group was weighed daily from day 18 to day 7 post challenge. Fecal samples from all experimental groups were collected and checked before challenge; no oocysts were detected. Fecal samples in each cage were collected from randomly selected sites and the oocyst count per gram of feces was calculated using the technique described by Permin and Hansen (1998) and recorded from day 4 to day 12 post challenge. On day 7 after challenge, three randomly selected chickens in each group were euthanized by cervical dislocation for cecal lesion scoring according to the method of Johnson and Reid (1970). The scoring scales ranged from 0 to +4, where 0 = no lesion, +1= mild lesion (with few scattered petechia), +2= moderate lesion (with numerous petechia, bleeding and slight thickening), +3= severe lesion (with severe bleeding and clotting) and +4= extremely severe lesion (with severe bleeding, a much thickened or ruptured cecal wall, gangrene or death).

**Data analysis**

The data were analyzed using the analysis of variance procedure of the SAS statistical software package (Statistical Analysis System, 2003). Tukey’s post hoc test was applied to compare the means of body weight gain, feed intake, mortality and fecal oocyst count between treatments. Cecal lesion scores were analyzed using general linear models and the means of cecal lesion scores between treatments were compared using least square means. The difference between treatments was considered significant at the \(P < 0.05\) level.

**Table 1** Experimental treatment in eight groups, (30 chickens each) to evaluate the effect of *Lepidium sativum* on *Eimeria tenella* infection.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Number of chicks</th>
<th>Oocyst challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Unsupplemented</td>
<td>30</td>
<td>_</td>
</tr>
<tr>
<td>2</td>
<td>Unsupplemented</td>
<td>30</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>1 g of seed powder per kg of feed</td>
<td>30</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>1 g of whole seed per kg of feed</td>
<td>30</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>1 g of extract per kg of feed</td>
<td>30</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>0.6 g of amprolium per liter in drinking water</td>
<td>30</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>1 g of seed powder per kg of feed</td>
<td>30</td>
<td>_</td>
</tr>
<tr>
<td>8</td>
<td>1 g of whole seed per kg of feed</td>
<td>30</td>
<td>_</td>
</tr>
</tbody>
</table>

Oocyst challenge involved challenge with \(1 \times 10^5\) of sporulated *E. tenella* oocysts, (–) = No infection with sporulated *E. tenella* oocysts, (+) = Infection with sporulated *E. tenella* oocysts.
RESULTS

Oocyst excretion

After the peak of oocyst shed, chickens fed the diet supplemented with whole seed demonstrated a slow reduction of oocyst shed until day 9 post challenge and later a fast reduction of oocyst shed was observed on day 9 post challenge. However, chickens fed a diet supplemented with either seed powder, extract or amprolium showed a fast reduction of oocyst shed on day 8 post challenge (Figure 1).

Average daily gain and average daily feed intake

The average daily gain of *E. tenella*-infected chickens fed the diet supplemented with either LS seed powder, whole seed or extract, was 11.08 ± 1.29, 11.24 ± 1.27, 10.55 ± 0.76 g, respectively, and significantly (*P* < 0.003) higher than in those fed the unsupplemented control diet (6.80 ± 0.45 g). The average daily feed intake of chickens fed the diet supplemented with either LS seed powder, whole seed or extract was significantly greater (*P* < 0.0001) than in those fed the unsupplemented control diet (Figure 2).

Oocysts per gram, mortality and lesion score

Chickens fed a diet supplemented with either seed powder, whole seed or extract following *E. tenella* infection demonstrated significantly reduced oocysts counts of 88 ± 10.5, 100 ± 10 and 3.67± 2.51 (*P* < 0.0007), mortality 3.33 ± 5.77, 0 and 0 % (*P* < 0.0001) and lesion score 0.89 ± 0. 27, 0.56 ± 0.27 and 0.67 ± 0.27 (*P* < 0.0001), respectively, compared with those fed the unsupplemented or control diets with 370 ± 225, 16.67 ± 5.77% and 2.67 ± 0.27, respectively. Infected chickens fed a diet supplemented with seed powder showed slightly increased lesion scores and mortality when compared to those fed the whole seed or extract-supplemented diet. Noninfected chickens fed the diet supplemented with seed powder also showed levels of cecal lesion and mortality similar to those in the infected chickens (Tables 2 and 3).

DISCUSSION

After the peak of oocyst shed (day 7 post challenge), chickens fed a diet supplemented with either seed powder, extract or amprolium rapidly reduced oocyst shed on day 8 post challenge (Figure 1). However, the chickens fed the diet supplemented with whole seed gradually reduced oocyst shed until day 9 post challenge. The delay in the reduction of oocyst shed might have been related to the retardation of parasite development.

![Figure 1](image-url) Oocyst fecal count of *Eimeria tenella*-challenged chickens fed diet supplemented either with or without *Lepidium sativum*, or with or without amprolium, 6-12 d post challenge.
This could have been due to the whole seed coat, which may contain different active ingredients of different amounts which are capable of preventing the intra cellular development of *E. tenella* or the enhancement of the host immunity. Delaquis *et al.* (2002) reported that different active ingredients could be found in different parts of the plant. *Lepidium sativum* seed oil is rich in tocopherol (a natural antioxidant), carotenoid and fatty acids such as oleic and α-linolenic acids (Diwakar *et al.*, 2010). Tocopherols are lipid-soluble antioxidants they may have an effect on the intracellular development of the parasites. A diet supplemented with whole seed might affect the development of *E. tenella* similar to n-3 fatty acid- supplemented diets. Allen *et al.* (1996) reported reduced parasite

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**Table 2** Oocyst fecal count and mortality rate in *Eimeria tenella*-challenged chickens fed diet supplemented either with or without *Lepidium sativum*, or with or without amprolium, 7 days post challenge. Treatments were: noninfected unsupplemented (1), infected unsupplemented (2), infected + seed powder (3), infected + whole seed (4), infected + extract (5), infected + amprolium (6), noninfected + seed powder (7) and noninfected + whole seed (8). (Vertical error bars indicate ± SD.)

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>OPG (at day12)</th>
<th>Mortality (%) ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control check</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>2</td>
<td>Control (infected + unsupplemented)</td>
<td>370 ± 225&lt;sup&gt;a&lt;/sup&gt;</td>
<td>16.7 ± 5.77&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>3</td>
<td>Infected + seed powder</td>
<td>88.0 ± 10.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.33 ± 5.77&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>4</td>
<td>Infected + whole seed</td>
<td>100 ± 10.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>5</td>
<td>Infected + extract</td>
<td>3.67 ± 2.51&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>6</td>
<td>Infected + amprolium</td>
<td>37.3 ± 14.2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>7</td>
<td>Noninfected + seed powder</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.67 ± 5.77&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>8</td>
<td>Noninfected + whole seed</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.00 ± 0.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

OPG = Oocysts per gram of feces (× 100).

<sup>a,b</sup> Means in a column with different lowercase superscript letters are significantly different (*P* < 0.05).
(E. tenella) invasion and development in chickens that consumed diets supplemented with high levels of n-3 fatty acids from fish (menhaden) oil and expressed flaxseed oil, and Danforth et al. (1997) reported ultra structural changes in both the asexual and sexual stages induced by n-3 fatty acids.

At sufficiently high intakes, long-chain n-3 polyunsaturated fatty acids (PUFAs), as found in oily fish and fish oils, decrease the production of inflammatory eicosanoids, cytokines and reactive oxygen species and the expression of adhesion molecules. Long-chain n-3 PUFAs act both directly (for example, by replacing arachidonic acid as an eicosanoid substrate and inhibiting arachidonic acid metabolism) and indirectly (for example, by altering the expression of inflammatory genes through effects on transcription factor activation). Long-chain n-3 PUFAs also give rise to a family of anti-inflammatory mediators termed resolvins (Calder, 2006). The decrease in cecal lesions might have been associated with a reduction in the inflammatory process and the development of the parasite within the cecal mucosa. Diets rich in n-3 PUFAs have anti-inflammatory and immunosuppressive activities (Calder, 1997). Such n-3 fatty acid-induced effects may be of use as a therapy for acute and chronic inflammation and for disorders which involve an inappropriately activated immune response (Calder and Grimble, 2002).

Eimeria tenella-challenged chickens fed a diet supplemented with either seed powder, whole seed or extract showed improved body weight gain. This might have been associated with the antioxidant capacity of LS seed. Antioxidants are an important part of the defense system of the host body and help to cope with oxidative stress caused by reactive oxygen species. Colnago et al. (1984) reported significantly reduced mortality and increased body weight gain in nonimmunized chickens challenged with E. tenella and fed diets supplemented with selenium or vitamin E. A Lepidium sativum-supplemented diet showed similar anticoccidial effect to these previous results.

The nutrient composition of LS seed was reported to have high amounts of all essential amino acids, except S-containing types and tryptophan with appreciable amounts of protein, fiber, lipids, ash, moisture and carbohydrates (Zia-Ul-Haq et al., 2012). These may have contributed to the reduction in growth retardation. Apart from diet, the body also has several antioxidant

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>LS means lesion score ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control check (noninfected + unsupplemented)</td>
<td>0.00 ± 0.27c</td>
</tr>
<tr>
<td>2</td>
<td>Control (infected + unsupplemented)</td>
<td>2.67 ± 0.27a</td>
</tr>
<tr>
<td>3</td>
<td>Infected + seed powder</td>
<td>0.89 ± 0.27b</td>
</tr>
<tr>
<td>4</td>
<td>Infected + whole seed</td>
<td>0.56 ± 0.27bc</td>
</tr>
<tr>
<td>5</td>
<td>Infected + extract</td>
<td>0.67 ± 0.27bc</td>
</tr>
<tr>
<td>6</td>
<td>Infected + amprolium</td>
<td>0.56 ± 0.27bc</td>
</tr>
<tr>
<td>7</td>
<td>Noninfected + seed powder</td>
<td>0.11 ± 0.27c</td>
</tr>
<tr>
<td>8</td>
<td>Noninfected + whole seed</td>
<td>0.00 ± 0.27c</td>
</tr>
</tbody>
</table>

LS = Least squares means.

Table 3 Lesion score results in Eimeria tenella-challenged chickens fed diet supplemented either with or without Lepidium sativum, or with or without amprolium.

a,b,c = Means in a column with different lowercase superscript letters are significantly different (P < 0.05).

Three randomly selected chickens from each group (a total of 9 chickens per treatment) were euthanized for cecal lesion scoring on day 7 after challenge.
mechanisms that it can use to protect itself from damage mediated by reactive oxygen species. The antioxidant enzymes glutathione peroxidase, catalase and superoxide dismutase are such enzymes. However, an imbalance between oxidants and antioxidants in favor of the oxidants, potentially leading to damage, is termed “oxidative stress” (Sies, 1997).

*Lepidium sativum* seed extract has good antioxidant capacity, which can reduce different types of radicals, such as the phenol content, the tetraethylammonium chloride content, the ferric-reducing antioxidant power and the total radical-trapping antioxidant parameter (Zia-Ul-Haq *et al*., 2012). Diwakar *et al.* (2010) reported that the essential oil derived from LS seeds contained fatty acids such as oleic acid and α-linolenic acid. The current results demonstrated that LS seed-supplemented diets could significantly reduce the oocyst count, mortality and lesion score of *E. tenella*-infected chickens similar to those supplemented with amprolium when compared to the control chickens. This might have been associated with the antioxidant capacity of LS seed.

Sies (1997), described that oxidants are formed as a normal product of aerobic metabolism but can be produced at elevated rates under pathophysiological conditions. Intestinal mucosal cells are exposed to a variety of reactive intermediates and xenobiotics and the rate of accumulation of products of oxidative damage in these cells is high. The intake of plant-based exogenous antioxidant capable of scavenging free radicals may help to limit the oxidative stress and to prevent the damage caused by free radicals. Antioxidant defense involves several strategies, both enzymatic and non-enzymatic. In the lipid phase, tocopherols and carotenoids as well as oxy-carotenoids are of interest, as are vitamin A and ubiquinols.

*Lepidium sativum*-supplemented diets showed similar anticoccidial effect to those supplemented with n-3 fatty acids. Allen *et al.* (1997) reported significantly reduced cecal lesion in chickens challenged with *E. tenella* and fed diets supplemented with 5% menhaden oil and 15% flaxseed. Allen *et al.* (1996) reported significantly decreased cecal lesions and maintained weight gains in chickens challenged with *E. tenella* and fed diets supplemented with 2.5 to 10% fish oil, 10% flax seed oil or 10% linseed oil when they were compared to those fed unsupplemented diets.

Infected chickens fed a diet supplemented with seed powder showed slightly increased lesion scores and mortality when compared to those fed whole seed or extract-supplemented diets. Noninfected chickens fed a diet supplemented with seed powder also showed cecal lesions and mortality similar to those infected chickens. However, the difference between treatments was not statistically significant. In contrast noninfected chickens fed a diet supplemented with whole seed did not show any cecal lesions or mortality. Although Datta *et al.* (2011) showed that LS seed powder did not induce acute or subchronic toxic effects in LS seeds powder-fed Wistar rats, chickens fed a supplement with infected/noninfected LS seed powder showed toxic-like effects. Therefore, further investigation should be done regarding the toxicity of LS seed powder in chickens. A possible reason for the different results of LS to *E. tenella* infection could have been due to the preparation of the medicinal plant and the different active ingredients found in different sites of LS seeds. The composition of essential oils from different parts of the same plant differs widely. Essential oils obtained from the seeds of coriander (*Coriandrum sativum* L.) have a different composition to cilantro, which is obtained from the immature leaves of the same plant (Delaquis *et al*., 2002).

In conclusion, diets supplemented with *Lepidium sativum* seeds demonstrated effective anticoccidial results on *E. tenella* infection in broiler chickens. A diet supplemented with either LS whole seed, powder or extract was effective
in improving bodyweight gain and in reducing cecal lesions, mortality and the numbers of oocysts shed. However, noninfected chickens fed a diet supplemented with seed powder of LS showed cecal lesions and mortality. This might have been due to the toxic effects of the seed powder.

**LITERATURE CITED**


