Experimental studies on effects of
diet composition (electrolyte contents), litter quality (type, moisture) and
infection (coccidia) on the development and severity of foot pad dermatitis
in young turkeys housed with or without floor heating

Thesis

submitted in partial fulfilment of the requirements for the degree

- Doctor of Veterinary Medicine -
Doctor medicinae veterinariae
(Dr. med. vet.)

by

Amr Abd El-Wahab Hassan Abd El-Wahab

from
Mansoura / Egypt

Hannover, Germany 2011
Academic supervision:        Univ.-Prof. Dr. Josef Kamphues

Institute of Animal Nutrition

University of Veterinary Medicine Hannover, Foundation, Germany

1. Referee                   Univ.-Prof. Dr. Josef Kamphues

Institute of Animal Nutrition

University of Veterinary Medicine Hannover, Foundation, Germany

2. Referee:                  Univ.-Prof. Dr. Dr. h. c. J. Hartung

Institute for Animal Hygiene, Animal Welfare

and Farm Animal Behaviour

University of Veterinary Medicine Hannover, Foundation, Germany

Date of the oral examination: 18.11.2011
Dedicated to

My parents, wife, son and brothers
List of publications and presentations

Publications


**Poster Presentations**

1. Effects of sodium and potassium content in the diet and floor heating on development and severity of foot pad dermatitis (FPD) in young fattening turkeys (2011)
   A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
   Proceedings of the Society of Nutrition Physiology 20, 15-17.03.2011, Göttingen, Germany, p 73

2. Effects of different sodium and potassium contents in the diet and of floor heating on development and severity of foot pad dermatitis in young fattening turkeys (2011)
   A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
   Proceedings of the 18th European Symposium on Poultry Nutrition (ESPN), Çeşme-izmir-Turkey October 31- November 04, 2011, pp. 289-292

**Oral Presentations**

1. Interactive effects of different litter moisture contents and time of exposure on the development and severity of foot pad dermatitis in fattening turkeys (2010)
   A. Abd El-Wahab, A. Beineke, M. Beyerbach and J. Kamphues
   Proceedings of the 14th Congress of the European Society of Veterinary and Comparative Nutrition, Zurich, Switzerland, September 6th–8th, 2010, p. 66

2. Critical moisture content of litter, diet and use of floor heating regarding foot pad dermatitis in growing turkeys (2011)
   A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
Tierernährung für Tierärzte im Fokus: Gesundheit und Leistung des Nutzgeflügels unter dem Einfluss von Futter und Fütterung, Institut für Tierernährung, Stiftung Tierärztliche Hochschule Hannover, 08. 04. 2011

4. Effects of litter type and dietary Na and K contents without/with floor heating on the development and severity of foot pad dermatitis in young turkeys (2011)
A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
Proceedings 6th International Symposium on Turkey Production, 16th–18th June 2011 Berlin, Germany, pp 58-59

5. Effects of litter type, diets and floor heating on the development and severity of foot pad dermatitis in young turkeys (2011)
A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
Proceedings of the XVth International Congress on Animal Hygiene, 3rd-7th July 2011, Vienna, Austria pp 127-130

6. Effects of different dietary sodium and potassium levels and of floor heating on development and severity of foot pad dermatitis in young turkeys (2011)
A. Abd El-Wahab, C. F. Visscher, A. Beineke, M. Beyerbach and J. Kamphues
Proceedings of the 14th Congress of the European Society of Veterinary and Comparative Nutrition, Zaragoza, Spain, September 14th–16th, 2011, p 80
7. Foot pad dermatitis and experimentally induced coccidiosis in young turkeys housed with/without floor heating (accepted 2011)

Abd El-Wahab A., Visscher C. F, Wolken, S., Reperant, J-M., Beineke A., Beyerbach M. and Kamphues J.

International Poultry Scientific Forum, Georgia World Congress Centre, Atlanta, Georgia, USA, January 23-24, 2012
### Table of contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>1.1 Foot pad dermatitis: Implications for bird health and welfare/Food safety</td>
<td>1</td>
</tr>
<tr>
<td>1.2 External foot pad lesions</td>
<td>3</td>
</tr>
<tr>
<td>1.3 Pathology of the foot pad lesions</td>
<td>5</td>
</tr>
<tr>
<td>1.4 Aetiology of the foot pad lesions</td>
<td>6</td>
</tr>
<tr>
<td>1.4.1 Internal factors</td>
<td>6</td>
</tr>
<tr>
<td>1.4.1.1 Body weight and pressure</td>
<td>6</td>
</tr>
<tr>
<td>1.4.1.2 Sex</td>
<td>7</td>
</tr>
<tr>
<td>1.4.1.3 Breed</td>
<td>7</td>
</tr>
<tr>
<td>1.4.2 External factors</td>
<td>8</td>
</tr>
<tr>
<td>1.4.2.1 Diet composition</td>
<td>8</td>
</tr>
<tr>
<td>1.4.2.2 Wet litter</td>
<td>10</td>
</tr>
<tr>
<td>1.4.2.3 Litter type</td>
<td>12</td>
</tr>
<tr>
<td>1.4.2.4 Litter depth</td>
<td>14</td>
</tr>
<tr>
<td>1.4.2.5 Stocking density</td>
<td>14</td>
</tr>
<tr>
<td>1.4.2.6 Drinker design</td>
<td>15</td>
</tr>
<tr>
<td>1.5 Floor heating</td>
<td>15</td>
</tr>
<tr>
<td>1.6 Coccidiosis</td>
<td>15</td>
</tr>
<tr>
<td>1.6.1 E. adenoeides</td>
<td>16</td>
</tr>
<tr>
<td>1.6.2 Sporulation of oocysts</td>
<td>16</td>
</tr>
<tr>
<td>2 AIM OF THE EXPERIMENTAL STUDIES</td>
<td>19</td>
</tr>
<tr>
<td>3 CHAPTER 1: Experimental studies on the effects of different litter moisture contents and exposure time to wet litter on development and severity of foot pad dermatitis in young fattening turkeys</td>
<td>21</td>
</tr>
<tr>
<td>4 CHAPTER 2: Effects of floor heating and litter quality on the development and severity of foot pad dermatitis in young turkeys</td>
<td>44</td>
</tr>
<tr>
<td>5 CHAPTER 3: Effects of high electrolyte contents in the diet and using floor heating on development and severity of foot pad dermatitis in young turkeys</td>
<td>47</td>
</tr>
</tbody>
</table>
CHAPTER 4: Foot pad dermatitis and experimentally induced coccidiosis in young turkeys fed a diet without anticoccidia

GENERAL DISCUSSION

7.1 Experiments in details

7.1.1 Experiment 1

7.1.2 Experiment 2

7.1.3 Experiment 3

7.1.4 Experiment 4

7.2 General aspects

7.2.1 Exposure to wet litter

7.2.2 Floor heating technique

7.2.3 Diet composition

7.2.4 Role of coccidiosis

7.2.5 Type of litter

7.2.6 General critical points

7.3 Recommendations for management

7.4 Conclusions

SUMMARY

ZUSAMMENFASSUNG

REFERENCES

APPENDIX
List of abbreviations

°C  degree celsius  
d  day  
DM  dry matter  
et al.  et alii  
E.  Eimeria  
FCR  feed conversion ratio  
Fig.  figure  
FPD  Foot pad dermatitis  
h  hour  
H&E  hematoxylin and eosin (staining)  
NfE  nitrogen free extract  
NSP  non-starch polysaccharides  
PI  post inoculation  
SBM  soybean meal  
spp.  species  
sq ft  square foot  
vs.  versus

Dimensions and chemical elements were abbreviated according to the rules of the international nomenclature (IUPAC).
1 Introduction

The transition of poultry industry from the backyard flocks of 1950’s to the current commercial form of intensive production system has led to produce poultry meat to supply for the human consumption as well as expanding export markets. The National Chicken Council recommends <30% incidence of foot pad lesions (FPD) in commercial broiler flocks to meet the current animal welfare guidelines. The prevalence of FPD in turkeys is extremely high (BERG 1998; EKSTRAND et al. 1998). FPD can achieve a prevalence of 91-100% at the end of the fattening period in turkeys (HAFEZ et al. 2004). GROSSE LIESNER (2007) emphasised that about 97.2% of turkeys at slaughtering showed FPD lesions. More recently, the condition of poultry feet is used as a production criterion to evaluate the animal welfare programs implemented by commercial poultry companies. Foot pad dermatitis is an important aspect of bird welfare, as in severe cases, the foot pad lesions may cause pain which together with a deteriorated state of health constitutes a welfare issue. The growing demand for least-cost, wholesome and convenient food products has been the driver for the expansion and diversification of the poultry industry. Poultry feet or paws (the portion of the feet cut just below the spur), are one of the new processing by-products that has an intense demand in recent years from the Southeast Asia especially, China and Hong Kong (BERG 1998). The financial incentive and the increasing demand have led to efforts to maximize the yield and quality of the birds feet harvested. This trade is based on a high quality product, i.e. poultry feet without severe lesions or discoloration. Downgrading results in a precipitous drop in the quantity available for sale and the value received for the exported poultry feet. Downgrading of poultry feet due to FPD results in rejects and associated loss in the sale value of the product. There is no doubt that feeding and housing conditions (litter quality) are the main factors in the aetiology of FPD in poultry farms.

1.1. Foot pad dermatitis: Implications for bird health and welfare/Food safety

Although there are various estimates of its prevalence, it is difficult to compare findings because the scoring systems used in different experiments are not the same. In a survey carried out by EKSTRAND and ALGERS (1997) 98% of Swedish turkey poult had
evidence of FPD. BERG (1998) estimated the prevalence of FPD in Swedish turkeys to be 
20 % for severe lesions (ulcers) and 78 % for mild lesions (discolouration, erosion). FPD can 
achieve a prevalence of 91-100 % in fattening turkeys (HAFEZ et al. 2004). Also, GROSSE 
LIESNER (2007) found that about 97.2 % of turkeys at slaughtering showed FPD lesions 
without any negative adverse effects on final body weight.

A greater focus is being put on animal welfare in modern animal husbandry both for ethical 
reasons and consumer desires. DAWKINS (1983) and later on DUNCAN (1996) have 
claimed that animal welfare is mainly related to the subjective feelings of the animals. In 
contrast, there are a number of different indicators for animal welfare can be used too, such as 
health and mortality, ethological measures, productivity, physiological and immunological 
measures (BROOM 1991). However, none of these indicators can give the full picture alone. 
Often ethical and political considerations must also be taken into account (SANDØE and 
SIMONSEN 1992). Thus, for example the Broiler Foot Health Programme was created in 
Sweden, as part of the Animal Welfare Programme (BERG 1998). In a review paper, 
SAVORY (1995) mentioned that poor litter quality is considered as one of the three main 
categories contributing to welfare problems in broilers. Similarly, HOCKING (1993) stated 
that poor litter is recognised as a welfare problem also in turkey production. HARMS and 
SIMPSON (1975) reported that birds with FPD had an unsteady walk. Nevertheless, it is very 
difficult to identify lameness caused by FPD in a commercial flock. As birds with FPD 
usually get the same kind of lesions on both feet severely affected birds are rarely seen 
limping, but are instead less likely to move. A part from animal welfare aspects, FPD is 
relevant to the poultry meat industry for several reasons. It has been indicated that broilers 
with severe FPD show slower weight gain (MARTLAND 1985; EKSTRAND and ALGERS 
1997), which has been suggested to be a result of pain induced inappetance (MARTLAND 
1985). In a paper describing a study on turkey poults, SCHMIDT and LÜDERS (1976) 
suggested that the lesions cause pain, resulting in reluctance to move and thus decreased feed 
consumption. MARTLAND (1984) reported an association between wet litter and a reduction 
in body weight in groups which also had a high incidence of FPD.

If the problem is widespread in a flock, this can lead to substantially reduced profit for the 
producer. As flocks with a high incidence of FPD often also show a high prevalence of other 
types of contact dermatitis, such as breast blisters and hock burns (GREENE et al. 1985;
MARTLAND 1985), in addition to lower body weights, downgrading may adversely affect the profitability of these flocks (WISE 1978). Moreover, lesions on the feet may be a gateway for bacteria which might affect food safety (MAYNE et al. 2006a).

1.2. External foot pad lesions

Foot pad dermatitis, also known as plantar pododermatitis, is basically a type of contact dermatitis affecting the plantar region of the feet, with lesions begin as small scaly brown scabs on the surface of the metatarsal and digital pads, becoming cracked and eroded and progressively larger over the first few weeks of life along with acute inflammation, swelling, hyperplasia and necrosis of the epidermis with deep ulcers occurring in severe cases (GREENE et al, 1985; BREUER et al. 2006). The ulcers are often covered by crusts formed by exudates, faecal material and litter.

The lesions can develop in less than a week and then progress to ulcers (GREENE et al., 1985). MAYNE et al. (2006b) observed that FPD develops at an early age in commercial turkey flocks, where skin discolouration appears at one week-old, fully developed lesions in which the integrity of the epidermis has been lost. Also, CLARK et al. (2002) observed the FPD as early as 3 days of age as a reddening of the foot pads. Foot pad dermatitis in meat-type poultry partly has a similar background to so-called ‘breast blisters’ and ‘hock burns’ in broilers (HARMS and SIMPSON 1975; GREENE et al. 1985; MARTLAND 1985), but these lesions usually develop more slowly and are less frequent (STEPHENSON et al. 1960). Similar types of lesions in turkeys; such as so called ‘breast buttons’ (focal ulcerative dermatitis; FUD), ‘breast blisters’ and ‘scabby hocks’, are also believed to have the same background as the foot pad lesions (MARTLAND 1984).

Several scoring systems for FPD are in use, one of these is the system based on the work by MARTLAND (1984) in turkeys (Table 1). More recent scoring was stated by MAYNE et al. (2007b), which was more accurate and involves all stages of FPD including that were not assessed by the previous system (Table 2).
### Table 1: Scoring system for classifying foot pad lesions (MARTLAND 1984)

<table>
<thead>
<tr>
<th>Score</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>Small scab(s) &lt;5 % pad area</td>
</tr>
<tr>
<td>2</td>
<td>Larger scabs &lt;25 % pad area</td>
</tr>
<tr>
<td>3</td>
<td>Severe, large scab-filled ulcers</td>
</tr>
</tbody>
</table>

### Table 2: External scoring system according to (MAYNE et al. 2007b)

<table>
<thead>
<tr>
<th>Score</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No external signs of FPD. Skin of the footpad and digital pads appears normal, no redness, swelling or necrosis is evident. The skin of the foot pad feels soft to the touch</td>
</tr>
<tr>
<td>1</td>
<td>Slight swelling and/or redness of the skin of the foot pad</td>
</tr>
<tr>
<td>2</td>
<td>The pad feels harder and denser than a non-affected foot. The central part of the pad is raised with swelling and redness and the reticulate scales may be separated. The digital pads may show a similar reaction</td>
</tr>
<tr>
<td>3</td>
<td>The central and digital foot pads are enlarged and swollen with red areas, and as the skin has become compacted, the foot pad is hard. The reticulate scales have become enlarged and separated, and small black necrotic areas may occur</td>
</tr>
<tr>
<td>4</td>
<td>Marked swelling and redness around the margins of lesions occur. Reticulate scales die and turn black, forming scale-shaped necrotic areas. The scales around the outside of the black areas may have turned white. The area of necrosis is less than one-eighth of the total area of the foot pad</td>
</tr>
<tr>
<td>5</td>
<td>Swelling and redness are evident in the central and digital foot pads. The total foot pad size is enlarged. Reticulate scales are pronounced, increased in number and separated from each other. The amount of necrosis extends to a quarter of the foot pad. Small necrotic areas may also appear on the digital pads</td>
</tr>
<tr>
<td>6</td>
<td>As score 5, but with half the foot pad covered by necrotic cells. The digital pads may have up to half of one pad covered with necrotic cells</td>
</tr>
<tr>
<td>7</td>
<td>Over half of the foot pad covered in necrotic scales</td>
</tr>
</tbody>
</table>
1.3. Pathology of the foot pad lesions

The foot pad lesions can develop in less than one week and then progress to ulcers (GREENE et al. 1985; EKSTRAND and ALGERS 1997). The mildest lesions show an infiltration of heterophils into the stratum germinativum, and defects in keratin formation (MARTLAND 1984). GREENE et al. (1985) also noted heterophils in the dermis, sub-epidermis, and epidermis as well as basophilic debris (necrotic cells) in the stratum corneum. Small vacuoles (often containing heterophils) are seen in the epidermis and inside blood vessels (HARMS and SIMPSON 1975; MARTLAND 1984; GREENE et al. 1985). In the center of the lesion, there is complete destruction of the keratin and epidermal layer, exposing necrotic tissue and a mass of inflammatory cells, predominantly heterophils (GREENE et al. 1985). MAYNE et al. (2006b) noted that FPD lesions are associated with massive increases in heterophils and macrophages and the loss of surface keratin. In more severe, ulcerated lesions, all the above observations were evident, but the major finding was acute inflammation. More dense cellular infiltration occurred, and there were more obvious defects in the stratum corneum such as thickening and the formation of ‘horned pegs’ (MARTLAND 1984). In commercial turkeys, the major pathological changes had occurred by 6 weeks and all turkeys with external signs of FPD lesions had fully developed microscopic inflammatory cellular lesions (MAYNE et al., 2006b).

Recently, MAYNE et al. (2007b) stated a histopathological scoring for the foot pads (Table 3). Externally normal foot pads exhibited microscopic evidence of lesions (minor cellular changes) after the turkeys reached an age of 4 weeks, suggesting that a lesion was beginning to develop. Moreover, the cellular and molecular changes associated with FPD were shown to be an inflammatory immune response and there was no evidence for an allergic reaction (MAYNE et al., 2007a).

The lesions may heal, but after healing, the foot pad does not show the normal skin fissure pattern and has a slightly paler colour (GREENE et al. 1985). A rapid healing of the lesions (within 2 weeks) in broilers when the birds were transferred from wet to dry litter was observed by MARTLAND (1985). Similar results were observed in turkeys by MAYNE et al. (2007b) who found that the lesions had virtually healed 15 day after transfer of the turkeys from wet to dry litter although histopathology assessment showed some residual tissue repair.
Table 3. Scoring system for histopathological observations of foot pads according to MAYNE et al. (2007b)

<table>
<thead>
<tr>
<th>Score</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No change, normal</td>
</tr>
<tr>
<td>1</td>
<td>Hyperkeratosis; ‘horned pegs’ of keratin on surface; epithelial hyperplasia; dead keratin on footpad surface</td>
</tr>
<tr>
<td>2</td>
<td>Epidermal acanthosis; increased dermal blood vessel densit</td>
</tr>
<tr>
<td>3</td>
<td>Vacuoles in dermis/epidermis; necrotic debris in keratin/epidermis</td>
</tr>
<tr>
<td>4</td>
<td>Presence of heterophils, macrophages and lymphocytes in dermis</td>
</tr>
<tr>
<td>5</td>
<td>Increased density of heterophils, macrophages and lymphocytes; congested/necrotic blood vessels; necrotic debris of cells in epidermis</td>
</tr>
<tr>
<td>6</td>
<td>Split epidermis: only one lesion</td>
</tr>
<tr>
<td>7</td>
<td>Split epidermis: more than one lesion</td>
</tr>
</tbody>
</table>

Hence, due to high prevalence of FPD in poultry farms and due to FPD could be used as one of the indicators of animal welfare as well as its relation to the food safety therefore, there is a need to minimize the prevalence and/or severity of FPD. There is only one way, by respect the aetiology factors in all stages of poultry production.

1.4. Aetiology of the foot pad lesions

The cause of FPD is complex. Many factors (that will be reviewed below) have been suggested, but it is more likely that the cause is multifactorial. The potential causes of FPD can be categorised into 2 main factors according to MAYNE (2005).

1.4.1. Internal factors

1.4.1.1. Body weight and pressure

Modern turkeys are less active than traditional turkeys, spending a greater amount of time sitting, and so increasing pressure on the breast, hock and foot pads as turkeys sit resting on these areas (WYLIE 1999). When turkeys stand, which they must do to feed, the small area of the foot pad bears the full weight of the bird. On one hand as body weight increases, the force
per area (pressure) of foot pad increases, and hence the pressure on the foot pads in heavier birds will increase. On the other hand, MARTLAND (1984) found no correlation between weights and the incidence or severity of foot and leg abnormalities in Wrolstad White turkeys. About 97.2% of turkeys showed FPD lesions with no marked effects on the body weight of five different strains of male turkeys at the end of the fattening period (GROSSE LIESNER 2007)

1.4.1.2. Sex

The effects of sex on FPD are still till now a state of controversial debate. Female skin contains more fat and less protein and collagen than males. This suggests that female skin may be more likely to tear than male skin, as the protein matrix will be less dense and therefore easier to pull apart (KAMYAB 2001). There is a higher incidence of lesions in males compared with females in some reports (HARMS and SIMPSON 1975; 1977). However, other work has reported no significant difference between the prevalence of FPD in males and females (MARTLAND 1984; EKSTRAND and ALGERS 1997).

1.4.1.3. Breed

In some studies a clear difference has been observed in the prevalence of FPD between different commercial breeds at the same age (EKSTRAND et al., 1998; SANOTRA and BERG 2003), while other studies have found no such difference (EKSTRAND et al., 1998). Large White turkey poult s were found to be more susceptible to FPD than Broad Breasted Bronze poult s when reared in the same conditions on wire floors. Using a scoring system of 0 for a normal foot pad, and 4 for a severe lesion, Large White poult s had an average score of 0.60, whilst Bronze poult s had a score of 0.20 (CHAVEZ and KRATZER 1972). However, this may be due to the fact that Large White poult s had a more rapid growth rate than Bronze poult s, resulting in heavier birds. BILGILI et al. (2006) reported that the susceptibility to FPD may vary by strain-cross. However, other researches found no relationship between the body weight of heavy turkey line and the incidence of FPD under commercial conditions (ELLERBROCK 2000; GROSSE LIESNER 2007). Newest experiments of GÜNTER et al. (2011, unpublished data) found marked/significant differences between three different genetic lines of turkeys under identical feeding and housing conditions.
1.4.2. External factors

Litter quality and type may be important in the prevalence of FPD as they are in contact with the foot pad. Litter quality is affected by many variables, e.g. moisture levels, humidity, season, amount and consistency of faeces, and stocking density (MAYNE 2005). After a series of experimental studies of diet composition (macrominerals, high dietary levels of SBM) and litter quality, YOUSSEF (2011) stated that wet litter is the predominating factor in the development of FPD in young turkeys. The type of litter provided is also important, as different substrates may absorb varying amounts of water, and cause varying amounts of friction on the foot pads of the birds. YOUSSEF (2011) observed that both lignocellulose and straw showed a higher water binding capacity compared with other bedding materials (wood shavings and maize silage). However, the water evaporation was higher and faster in lignocellulose but lower in straw. Wet wood shavings and maize silage released more water by pressure, related to their lower water binding ability but small amounts were released from lignocellulose and straw (YOUSSEF 2011). Lesions develop at points of contact between skin and the ground undergoes the cellular changes that characterise FPD (WHITEHEAD 1990). Therefore, GREENE et al. (1985) concluded that FPD was a contact dermatitis and suggested that poor litter conditions may be responsible.

1.4.2.1. Diet composition

Different dietary nutrients have a great impact on the foot pad quality such as unbalanced levels of protein, minerals or vitamins (MAYNE 2005; KAMPHUES et al., 2009; YOUSSEF 2011). Regarding to the protein content in the diet, the most severe lesions were found in birds fed a high protein diet consisting of only vegetable-based proteins. This could be due to increased nitrogen excretion and NH$_3$ formation in the litter (NAGARAJ et al., 2007). Also, EICHNER et al. (2007) found that birds fed all-vegetable diets based exclusively on corn and soybean meal had a higher incidence and severity of FPD when compared with those fed diets containing poultry by-product or corn gluten meal. Feeding higher levels of protein in the diet resulted in poor skin integrity and therefore predisposed the birds to FPD (WHITEHEAD and BANNISTER 1981). This was probably due to deficiency of biotin which was indicated by lower plasma biotin levels when the birds were fed high protein diets. It was found that the excess of crude protein levels in the diet could increase the production and excretion of uric
acid in the urine, leading to wet excreta that are rich in nitrogen, which results in a high prevalence of contact dermatitis (GORDON et al., 2003). Ammonia is produced as a result of microbial activity (in the excreta and litter) on uric acid. A combination of wet litter and high ammonia content in the litter was suggested to cause FPD (MARTLAND 1985). Nevertheless, YOUSSEF (2011) observed that the higher pH, NH$_3$ and uric acid content in the wet litter did not increase the severity of FPD and the high moisture alone 73 % for 8 h/d, without the presence of excreta, was sufficient to cause FPD. MAYNE et al. (2007b) found no direct correlation between NH$_3$ concentrations released from the litter and the incidence of FPD in turkeys.

On the one hand, it is observed that the indigestible carbohydrates levels in plant sources [primarily soybean meal (SBM)] are extremely high. These carbohydrates are reported as non-starch polysaccharides (NSP) and are also found at high concentrations in wheat, barley, triticale, and other grains (CHOCT 1997). The viscosity of digesta increases, as the dietary NSP concentrations increase resulting in “sticky” excreta which adheres more readily to the foot pads of the birds. On the other hand, soybean meal contains also a high amount of K (>20 g/kg DM), which is an electrolyte known to increase water intake (JAMES and WHEELER 1949). YOUSSEF (2011) concluded that the effect of SBM levels on FPD could be related to its content of both K (produce high excreta moisture) and oligosaccharides (with potential to produce viscous/sticky excreta). JENSEN et al. (1970) found a lower incidence of FPD when the poults fed diets containing other protein rich ingredients such as peas, meat meal and fish meal, but a low level of SBM. Also, it was noted that poults fed diets containing 40 % or more of SBM exhibited a high incidence of FPD, while birds fed diets containing low amounts or no SBM had little or no FPD lesions (JENSEN et al., 1970).

Dietary fat quality can also affect litter surface friability. BILGILI et al. (2006) evaluated the effect of low and high density diets on foot pad quality. Broilers reared on the high-density diet had significantly higher incidence of FPD compared with the low-density diet. Till now, little is known whether unabsorbed fat (fatty acids) could play a role in the development of FPD. The influences of macrominerals, on the incidence of FPD are thought to be related to increased water intake, resulting in higher moisture content in excreta and/or litter. Based on the feed composition, electrolytes play a major role in increasing the water intake in poultry. Diets with higher Na and K levels result in an increased water intake (JAMES and
WHEELER 1949 and SMITH et al., 2000) and litter moisture; whereas an increase in Cl did not have the same effect (MURAKAMI et al., 2000). The increased level of K in the diet is related to inclusion of higher levels of plant-protein rich ingredients especially soybean meal. MURAKAMI et al. (2000) observed that higher levels of Na (exceeding 0.15 %) in the broiler diets lead to increased litter wetness. However, YOUSSEF (2011) observed that the dietary Na content up to 2 g/kg and/or excess of Ca, P, Mg or Cl had no marked effect on litter quality and/or the severity of FPD in turkey poults. Moreover, HARMS and SIMPSON (1982) reported that higher levels of dietary NaCl increased the rate of growth of turkey poults and concurrently increased the severity of FPD.

Inadequate amounts of certain amino acids and vitamins as well as trace elements in the diets such as methionine, cystine, biotin, riboflavin and zinc increase the risk of FPD. Vitamins like biotin have a great impact on the prevalence of FPD (CLARK et al., 2002). Also, deficiency of zinc resulted in a higher incidence of foot pad lesions (WHITEHEAD 1990). Additionally, CHAVEZ and KRATZER (1972) found that supplementation of the diet with methionine reduced the incidence of FPD in turkey. Recently, YOUSSEF (2011) concluded that supplementation of the diet with high levels of biotin or organic zinc could reduce the severity of FPD, but only on dry litter and without preventive effects on wet litter.

1.4.2.2. Wet litter

Foot, breast and hock lesions increased in severity when birds’ litter was wet. The severity of lesions increased in broilers and turkeys reared in pens containing wet and sticky litter (HARMS and SIMPSON 1975, 1977 and MARTLAND 1985). These birds were observed with severe skin ulceration on the plantar surface of the foot, the caudal aspect of the intertarsal joint and over the sternum. Turkeys reared on wet litter, especially if particularly deep, were found after 20 weeks, to have a larger mean number of lesions, when compared with those raised on dry litter. In turkeys that did have lesions, a larger mean percentage of the foot pad was ulcerated in birds raised on wet litter when compared with those reared on dry litter (MARTLAND 1984). Moreover, by changing the wet litter for dry litter growth rate recovered (MARTLAND 1985), and lesions began to heal (GREENE et al. 1985 and MARTLAND 1985).
Housing birds on wet litter also increases the chance of faecal adhesion to the feet, which has been hypothesised to induce FPD (JENSEN et al. 1970).

Wet litter was found to contain a higher concentration of nitrogen and higher pH values than dry litter (LERNER 1996; ALCHALABI 2002), accompanied by higher concentrations of volatile ammonia within the litter which may be a causative agent of FPD. It was also reported that wet litter (74% moisture) alone causes similarly severe lesions as wet dirty litter (MAYNE et al., 2007b). YOUSSEF (2011) found that high litter moisture alone (73% moisture, 8 h/d), over 3 weeks without the presence of excreta was sufficient to cause FPD. These findings indicate that the effect of diets is likely to be caused by their effect on litter moisture rather than the contents of the excreta except in so far as the diet increases water intake and excreta- or litter moisture (MAYNE et al., 2007b).

It was reported that moisture content in litter exceeding 35% often result in a higher incidence of FPD (MARTLAND 1984). LYNN and SPECHTER (1987) observed that when the moisture content in the litter exceeds 46%, the litter surface becomes wet. Litter surface friability, along with the moisture, are also considered as predisposing factors to produce a contact dermatitis on the hock in broilers (TUCKER and WALKER 1992). Therefore, poor litter quality is considered a welfare problem in modern poultry production and hence research should focus on developing measures for upgrading litter quality. Therefore, the maintenance of proper litter quality (with moisture content of 25-30%) is likely to be highly effective in reducing the incidence and severity of FPD (JODAS and HAFEZ 2000). Also, YOUSSEF (2011) found that the severity of FPD began to increase markedly at a moisture content exceeding 30%.

In poultry, wet excreta and/or litter are often caused by unbalanced diet composition (will be discussed later) and poor quality of diet and water (JODAS and HAFEZ 2000; KAMPHUES et al., 2009). Thus, all factors that affect the litter quality (moisture content) directly or indirectly are of special interest. The factors contributing in production of wet excreta or litter and consequently predisposing for FPD are shown in Figure 1. A mixture of faeces and litter sticks to the foot pad and dries on, becoming extremely solid. This suggests that the effect of wet litter is stronger than the effect of increased body weight in regards to the development of FPD. Experimental evidences already reported and after a series of experimental studies of
Youssef (2011) suggested that wet litter is the most important factor affecting the development of FPD.

**Forced water intake**
- high barn temperature
- secondary to diet composition
- water quality (e.g. SO₄²⁻content)

**Defects in drinker techniques**
- leakage in valves of drinkers
- water waste by animals

**Diet composition**
- excess of nutrients excreted via urine (protein (nitrogen), Na, Cl, Mg, K)
- laxative dietary constituents (sulfate, amide, pectin,....)
- increased bacterial fermentation of nonstarch polysaccharides (e.g. stachyose, raffinose,....)
- mycotoxins (e.g. ochratoxin) → kidney function disorders

**Specific stress conditions**
- increased diuresis due to fear, panic, forced activity, overcrowding, barn climate (rapid changes of temperature)

**Improper litter material**
- inadequate water binding capacity
- bad loosening of litter (excreta compacted on litter surface)

**Infectious diseases (diarrhea/diuresis)**
- enteritis with subsequent malabsorption and maldigestion due to:
  - Coccidia (different Eimeria species)
  - Bacteria (E. coli, Campyl., Clostridia)
  - Viruses (e.g. Adenovirus)
- kidney diseases with subsequent kidney function disorders (e.g. by Infectious Bronchitis or Gumboro virus,....etc.)

---

**Fig. 1:** Diagram summarizing the possible causes of ‘‘wet litter syndrome’’ (watery/wet excreta) in poultry (modified after KAMPHUES et al., 2009)

1.4.2.3. Litter type

Litter plays an important role in moisture management within the broiler house. It acts as sponge absorbing moisture. Litter must not only be able to absorb moisture but should also
have a reasonable drying time to get rid of that moisture via evaporation as discussed recently of YOUSSEF (2011). The type of litter appears to have a marked effect on the incidence of FPD in turkeys (HESTER et al. 1997). Bedding materials with sharp edges (large particle size wood chips, chopped straw, etc.) may contribute to FPD by opening small puncture wounds on the foot pad which can lead to entry of bacteria and probably to FPD (BILGILI 2009). Therefore, the effects of litter material on FPD are thought to be due to either the physical structure (hard or soft) or the water-binding capacity (high or low) of the litter. EKSTRAND and ALGERS (1997) found that pouls reared on straw in commercial conditions showed a higher prevalence of FPD than those on wood shavings. Straw tends to have higher moisture content as well as higher ‘’caking’’ scores (forming compacted layer of excreta at the litter surface), resulting in a greater incidence of foot pad lesions (BILGILI et al., 2009). However, MCILROY et al. (1987) found no significant difference in the occurrence of hock and breast lesions in broilers reared on straw or wood shavings in commercial flocks.

The most common bedding material used for turkeys is wood shavings but there is currently a further litter type which can also be used namely lignocellulose. Lignocellulose is produced from wood by chopping the wood into fine particles which are then pressed into pellet form using steam and high temperature. It was noted that lignocellulose reduce the severity of FPD significantly which could be attributed to the higher absorbing capacity and also quick release of water from lignocellulose (BERK and HINZ 2010; YOUSSEF 2011).

Moreover, sand was also found to be an acceptable litter alternative to wood shavings, consistently showing a lower prevalence of foot pad lesions in broilers compared with wood shavings (BILGILI et al., 1999). Recently, BILGILI et al. (2009) studied the effect of different litter materials [wood shavings, pine bark, chipped pine, mortar sand, chopped straw, ground hardwood pallets, ground door filler (a wood fibre-based material used in insulating metal doors), and cotton-gin trash] on FPD in broilers. It was found that the ground door filler and the mortar sand had significantly lower incidence of FPD than did the other bedding materials. This could be related to higher moisture binding capacity of ground door filler and quickly release of moisture from mortar sand. GRIMES et al. (2006) found no significant difference in the prevalence of FPD between litter materials made from cotton waste, gypsum, and newspaper in comparison to wood shavings; however, there was more ‘’caking’’ with the cotton waste products.
A really modern trend is the use of artificially dried maize silage as a litter. Dried maize silage is already supplied by some biogas system operators. The low pH value as well as the lactic acid content of maize silage may have a bactericide effect and might result in a reduction in the effects of bacteria in the shed (BOSSE and MEYER 2007). Moreover, YOUSSEF (2011) noted that the external and histopathological FPD scores in young turkeys reared on dry litter were similar for wood shavings and dried maize silage.

1.4.2.4. Litter depth

The prevalence of FPD in broiler flocks raised on thick layers (>5 cm) of litter material was higher than in flocks housed on thinner layers (<5 cm; EKSTRAND et al., 1998). A possible explanation could be that the birds are less prone to peck, scratch and turn the litter particles over if the litter is thick and are thereby less effective in ventilating the litter and keeping it dry. In contrast to these results, MELUZZI et al. (2008) reported that broilers reared on thicker layers of litter (3-4.5 kg/m²) had a lower incidence of FPD than those raised on thin layer (2.3-3 kg litter/m²). TUCKER and WALKER (1999) found lower hock burn scores when the litter was at a depth of 10 cm compared with 2.5 and 5 cm. However, STEPHENSON et al. (1960) found no effect of the litter depth on the prevalence of breast blisters in broilers. Moreover, TUCKER and WALKER (1992) observed variations in the results obtained with different litter materials. This inconsistency may be related to differences in the structure, particle size and other quality properties of the tested litter materials. Presumably the depth has no effect per se, all findings are related to DM or moisture contents of the litter surface, where the foot pad is in contact with.

1.4.2.5. Stocking density

Litter quality deteriorates rapidly and litter moisture increases as stocking density increases (TUCKER and WALKER 1992) by the effect of a ratio determined by a fix mass of litter but increasing the amount of excreta is due to increase the number of birds. Nevertheless, the effect of stocking density on the incidence of FPD is still controversial. Lesions of the foot pad, breast and hock increased in broilers when stocking density increased (SVEDEBERG 1988). This is probably related to poorer litter quality because more cases of poor litter quality were found in highly stocked pens (<0.48 ft²/bird) when compared with low density pens.
[≥0.49 ft²/bird (0.15 m²/bird)]. However, other studies suggested that stocking density has little or no effect on the prevalence of FPD (ALGERS and SVEDBERG 1989) as long as the surface is dry.

1.4.2.6. Drinker design

EKSTRAND and ALGERS (1997) reported that flocks with small cup drinkers showed a lower prevalence of FPD when compared with bell drinkers. In support of this finding, TUCKER and WALKER (1999) found that small cup drinker designs reduced litter wetness, and decreased hock burn which is thought to be exacerbated by sitting in wet litter.

1.5. Floor heating

Recently, floor heating was used in poultry farms. Many forms for application of floor heating were encountered. It has been noted that the prevalence of FPD in floor heating groups was 21.5 % ± 3.7 vs. 45.0 % ± 7.1 for groups housed without floor heating (BERG and ALGERS 2004). Up till now, there was no study to determine the suitable temperature at litter surface that can be reached by using floor heating without any side effects on birds. Also, little is known about the effects of floor heating on airborne dust inside poultry houses as well as on the health of the respiratory system of birds. From economical point of view, the costs of applying floor heating in the poultry farms should be considered however recently many farms are already supplied by some biogas system operators.

1.6. Coccidiosis

The incidence of FPD can be affected by the health condition of birds. One of the major factors causing wet litter is diarrhoea. This can be a result of different infections in the intestinal tract, for-example protozoal *Eimeria spp*, infection (MAYNE 2005). Coccidiosis is one of the most important and common diseases that affect poultry, it results in a great economic loss all over the world (BRAUNIUS 1980).
1.6.1. **E. adenoeides**

According to TREES (1990) the *E. adenoeides* (affecting the caeca) is considered the highest pathogenic parasite among the other major *Eimeria spp.* infecting turkeys (*E. meleagrimitis*, affecting the upper gut; *E. galopavonis*, affecting the lower gut; and *E. dispersa*). In the case of *E. adenoeides*, there may be oedema, white caseaus exudates in the caeca that may be packed with gametocytes and oocysts and petechial haemorrhages (JOYNER 1978). CLARKSON (1959) found that 2.5x10^4 oocysts of *E. adenoeides* given orally caused reduced weight gain in 3-week-old poults; and 10^5 to 2x10^5 oocysts caused 50 % to 100 % mortality. Mortality was 90 % to 100 % when poults were given 8x10^4 oocysts of *E. adenoeides* (CHAPMAN 2008).

1.6.2. **Sporulation of the oocysts**

It is generally believed that moist litter will favour the development of coccidiosis, because of the higher sporulation ability thus induced (CARD and NESHEIM 1972; MATTER and OESTER 1989). The infective form of Eimeria is highly resistant oocyst, which is shed in the excreta of infected birds. The oocyst is excreted from the host as an undifferentiated stage out side of the GIT, and in order to become infective it must sporulate. During sporulation four sporocysts, each containing two sporozoites, are formed within the oocyst (KHEYSIN 1972). The degree and rate of sporulated oocysts are important factors affecting the infection pressure in a flock of birds, thus influencing the epidemiology of the infections (WALDENSTEDT et al. 2001). Sporulation of the oocysts depends mainly on three basic factors: temperature, humidity, and access to oxygen (KHEYSIN 1972). Normal sporulation occurs from about 8 up to 32.5 °C. Below 12 °C the time requires for sporulation is exceedingly long, and at 35 °C sporulation is morphologically abnormal. The optimal temperature for sporulation was about 30 °C at which temperature some organisms completed the process in 23 h. Fifty percent of the oocysts required 65 hours to begin sporulating at 20 °C and 36 h at 25 °C (MAROUARDT et al. 1960).

Coccidial infections in a study may be obtained naturally by placing birds on contaminated litter, but infections established through the use of “seeder birds” or inoculation of oocysts via the feed or water are usually more effective and predictable (GARD et al. 1969). JOHNSON and REID (1970) stated external scoring for caecum infected with coccidia
Table 4. Scoring system for external observations of caecum infected with coccidia according to JOHNSON and REID (1970)

<table>
<thead>
<tr>
<th>Score</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No gross lesions</td>
</tr>
<tr>
<td>1</td>
<td>Scarce petechial haemorrhages on the mucosal surface and slight thickening of the intestinal mucosa</td>
</tr>
<tr>
<td>2</td>
<td>A small number of haemorrhages up to pinhead size on the mucosal surface, oedema and thickening of the intestinal mucosa</td>
</tr>
<tr>
<td>3</td>
<td>Many haemorrhages up to pinhead size on the mucosal surface, oedema and thickening of the intestinal mucosa, degenerative changes in the mucosal epithelium, caeca contain necrotic cheese-like depris</td>
</tr>
<tr>
<td>4</td>
<td>Many haemorrhages up to pinhead size on the mucosal surface, oedema and pronounced thickening of the intestinal mucosa, strong degenerative changes in the mucosal epithelium, caeca are full of necrotic cheese-like depris containing many oocysts and blood traces</td>
</tr>
</tbody>
</table>

Also, for description the histopathological changes occurred in the caecum, a developed scoring system for caecum histopathological assessment was established (Table 5).

Table 5. Histopathological scoring system for caecum infected with coccidia

<table>
<thead>
<tr>
<th>Score</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>normal</td>
</tr>
<tr>
<td>0.5</td>
<td>&lt;3 sub-epithelial haemorrhages; mild inflammation</td>
</tr>
<tr>
<td>1</td>
<td>&gt;3 sub-epithelial haemorrhages; mild to moderate inflammation</td>
</tr>
<tr>
<td>1.5</td>
<td>bleeding into the lumen; moderate inflammation</td>
</tr>
<tr>
<td>2</td>
<td>&gt;3 intraluminal haemorrhages and/or ≥10 sub-epithelial haemorrhages; moderate to severe inflammation</td>
</tr>
<tr>
<td>2.5</td>
<td>mild atrophy of the crypts; severe inflammation</td>
</tr>
<tr>
<td>3</td>
<td>moderate atrophy of the crypts; mild fibrinous haemorrhagic exudation</td>
</tr>
<tr>
<td>3.5</td>
<td>small to medium sized ulcer (&lt;10 crypt width)</td>
</tr>
<tr>
<td>4</td>
<td>extensive mucosal ulceration; diphtheriod necrotic inflammation</td>
</tr>
</tbody>
</table>

The appearance of resistance to coccidiostats, consumer demand for reducing amount of feed additives, and European Union Regulations (withdrawal of antibiotic feed additives as a
precautionary measure) might restrict the use of coccidiostats in the future (EUROPEAN COMMISSION REGULATIONS 1997). If this happens, alternative strategies should probably be introduced to restrict the adverse effects of coccidial infection on production. However, testing the diet composition, should not let us neglect the potential role of coccidiosis or – from the feed production point of view – the correct use/adding of an effective coccidiostat. In the diet composition with high efficient coccidiostat, the role of infection could be neglected. However, due to maldosing and/or inefficient anticoccidial additive in the diet, the excreta and bedding material will be markedly influenced by the coccidial infection. The ingredients used and their commercial sources as well as the form of the feed (pellet, crumble or mash) should reflect that to be used in practice, as milling and pelleting process may affect drug stability, growth rate and feed utilization of broilers (ENGBERG et al., 2002).

Summarizing, there is a broad spectrum of interactions among dietary factors, housing and management (including the most important factor, i.e. the litter moisture) and infections of birds that all together predispose or protect poultry from FPD.
2 Aim of the experimental studies

Some scientists actually argue that instead of attempting to “measure” animal welfare, the role of science should be primarily to identify, rectify and prevent welfare problems. Therefore, there is a high need to reduce the severity of FPD and hence to increase the birds’ health and performance. Four experimental studies in young turkeys were performed to demonstrate field relevant interactions between different causative factors that together determine the risk for being affected by FPD. Therefore, the following questions should be investigated and answered:

1. What is the minimum level of moisture in the litter and/or the time of exposure that together result in elevated risks for FPD development?
The answer to this question should have a marked impact on housing (needs for litter treatment), management and feeding (diet composition etc).

2. What is the effect of the litter material per se, i.e. at identical feeding/watering/housing conditions under the influence of a modern management tool, i.e. at housing with and without floor heating?
The answer should have marked consequences for routinely established housing conditions or measures that should be implemented.

3. What are the interactive effects of high electrolyte contents in the diet, when concomitantly the modern technique of floor heating is available or not?
The answer should allow to set up limits regarding diet composition related to housing conditions.

4. What are the consequences regarding FPD, when coccidiosis develops (here due to an experimental infection/in the field caused by missing an effective coccidiostat)?
The answer should sensitize people, not to neglect under those circumstances the role of infections for litter quality and thus for development of FPD.
The own experimental studies were focused the effects of changes in one factor - in the multiaetiological “puzzle” of FPD - on further relevant variables (i.e. feeding/housing/management/infection) in the system.

Therefore, four consecutive trials were conducted to:

- find out the “critical litter moisture content” that results in higher severity of FPD,
- investigate the effects of two litter types “wood shavings or lignocellulose”/with and without floor heating on the development and severity of FPD,
- quantify the impact of dietary factors (surplus levels of electrolytes) with and without floor heating on the development and severity of FPD,
- test the effects of an experimental coccidial infection (wet litter as a consequence of an infection) on the development and severity of FPD in turkeys.
3. Chapter 1

Experimental studies on the effects of different litter moisture contents and exposure time to wet litter on development and severity of foot pad dermatitis in young fattening turkeys

Experimentelle Untersuchungen zu Auswirkungen unterschiedlicher Feuchtegehalte der Einstreu und Dauer der Exposition auf Entwicklung und Schweregrad der Fußballenentzündung von jungen Mastputen
3. Chapter 1

Experimental studies on the effects of different litter moisture contents and exposure time to wet litter on development and severity of foot pad dermatitis in young fattening turkeys

Experimentelle Untersuchungen zu Auswirkungen unterschiedlicher Feuchtegehalte der Einstreu und Dauer der Exposition auf Entwicklung und Schweregrad der Fußballenentzündung von jungen Mastuten

1A. ABD EL-WAHAB, 1C. F. VISSCHER, 2A. BEINEKE, 3M. BEYERBACH, 1J. KAMPHUES

1Institute of Animal Nutrition, 2Institute of Pathology, 3Institute of Biometry and Information Processing, University of Veterinary Medicine Hannover, Foundation, Germany

Accepted for publication in Journal of European Poultry Science, 2010

will be published at January, 2012

Correspondence: Prof. Dr. Josef Kamphues

Institut für Tierernährung, Tierärztliche Hochschule Hannover

Bischofsholer Damm 15, 30173 Hannover, Germany

Tel: +49 (0) 511-856-7301 Fax: +49 (0) 511-856-7698

E-mail:josef.kamphues@tiho-hannover.de
Experimental studies on the effects of different litter moisture contents and exposure time to wet litter on development and severity of foot pad dermatitis in young fattening turkeys

*Experimentelle Untersuchungen zu Auswirkungen unterschiedlicher Feuchtegehalte der Einstreu und Dauer der Exposition auf Entwicklung und Schweregrad der Fußballenentzündung von jungen Mastputen*

A. ABD EL-WAHAB¹, C. F. VISSCHER¹, A. BEINEKE², M. BEYERBACH³, J. KAMPHUES¹

**Introduction**

The incidence and severity of foot pad dermatitis (FPD) is of great concern to the poultry industry. It is basically a type of contact dermatitis affecting the plantar region of the feet, with lesions surrounded by a reddening of the foot pads as a first symptom, then discoloration and hyperkeratosis often in combination with erosions and necrosis of the epidermis, with deep ulcers occurring in severe cases (BREUER et al., 2006). In meat type turkeys this disease can reach a prevalence of 91-100 % at slaughter (HAFEZ et al., 2004). About 97.2 % of turkeys showed FPD lesions with no marked effects on the body weight of five different strains of male turkeys at the end of the fattening period (GROSSE LIESNER, 2007). Feet health is an important aspect of poultry welfare and in recent years the level of FPD has been used to characterise the health and welfare of poultry flocks. Moreover, lesions on the feet may be a gateway for bacteria which might affect carcass quality (MAYNE et al., 2006).

Many factors have been implicated in the prevalence of FPD such as: type of litter, litter management, stocking density and nutrient supply (MAYNE, 2005). Birds spend most of

---

¹ Institute of Animal Nutrition, University of Veterinary Medicine Hannover, Foundation, Germany
² Institute of Pathology, University of Veterinary Medicine Hannover, Foundation, Germany
³ Institute of Biometry and Information Processing, University of Veterinary Medicine Hannover, Foundation, Germany
their productive life in close association with the bedding/litter material and hence the quality of the latter tells a lot about the skin quality of the bird. MARTLAND (1984) concluded that wet litter induces foot ulceration in fattening turkeys as the wetness of the litter may have increased the rate of production of irritants or more probably brought the irritants into contact. Pure water (without excreta) alone is sufficient to produce severe lesions (MAYNE et al., 2007; YOUSSEF et al., 2008). Housing birds on wet litter also increases the chance of fecal adhesion to the feet, which has been hypothesised to induce FPD (JENSEN et al., 1970). Furthermore, wet litter can lead to high ammonia levels in poultry houses because of high bacterial activity (ELLIOTT and COLLINS, 1982). High ammonia levels could be dissolved in high litter moisture resulting in irritant alkaline solution causing FPD (TUCKER and WALKER, 1992). However, in recent studies, it was noted that ammonia and/or uric acid concentration in the litter did not aggravate the negative effects of water on the foot pad, indicating that the high moisture alone in the litter is sufficient to cause FPD in young turkeys (YOUSSEF et al., 2008).

Dietary factors such as proportion of soybean meal and amounts of oligosaccharides, potassium and salt in feed force wet litter conditions (JENSEN et al., 1970; SMITH et al., 2000; EICHNER et al., 2007; BILGILI et al., 2009; YOUSSEF et al., 2009). Therefore, poultry diets may also be indirectly involved in the pathogenesis of this disease. Previous researches showed that moisture contents in litter have some side effects on bird’s health. Litter moisture higher than 35 % often results in a higher incidence of FPD (HARMS et al., 1977; MARTLAND, 1984 and 1985; MAYNE et al., 2006). According to JODAS and HAFEZ (2000) “wet litter condition” develops if moisture content is above 40 %. LYNN and SPECHTER (1987) showed that litter moisture contents higher than 46 % resulted in a wet litter surface and contact dermatitis in broilers. WANG et al. (1998) found that the prevalence of FPD was 49 % when housing layers on wet litter (55 % moisture). Housing turkey poult s on high wet litter (73 % moisture) for only 8 h/d led to imposingly higher prevalence of FPD (YOUSSEF et al., 2010). A previous study had used high litter moisture content (73 %) but had not shown the threshold level of litter moisture which could lead to a higher prevalence of FPD.

Additionally, exposure time of animals to wet litter could be varied in the field conditions during day-light (16 h) in terms of the time spent around feeding or drinking places, which are
particularly “wet litter conditions”. ABD EL-WAHAB et al. (2010) observed that in turkey field conditions, litter moisture contents could reach about 66.0 ± 2.2; 67.7 ± 1.1; 69.1 ± 2.4 around drinking and 50.5 ± 5.3; 57.4 ± 3.3; 61.2 ± 2.6 % around feeding places at day 50, 57 and 64 of fattening period, respectively. This suggests that litter moisture contents increased as the turkeys aged which could be due to the changing ratio of excreta to litter. Against this background, this study aimed to find out “the critical moisture content” or rather the time of exposure to this critical litter quality which results in higher prevalence and severity of FPD in young fattening turkeys.

Material and methods

Housing

Seventy-five female turkey poults (BUT-Big 6), one day old, were obtained from a commercial hatchery, divided into 4 groups, each group being allocated to a floor pen (1.50 m x 1.32 m). Each pen was littered with wood shavings to a depth of approximately 4 cm over the floor (5 kg/m2). During the first 3 days additional feed was offered (about 300 g/d) on paper to accustom birds to the feed. All turkeys were fed ad libitum a commercial diet (pellets) recommended by the breeder (Table 1).

Each pen was equipped with an infrared lamp, to achieve a temperature of about 32-35 °C at the outset in the vicinity of the one-day-old birds. The temperature was lowered by about 1°C every 2 days reaching about 18°C at day 35. The photoperiod from d 4 on was 16 h of light and 8 h of darkness.

Experimental Design

Table (2) summarises the experimental design. At the beginning of the experimental treatments (on day 14), the number of animals was optimised to 12 birds in the control group and 21 birds in each of the three treatment groups. The four groups were kept on dry wood shavings. The litter was kept as clean and dry as possible during the experimental period by regularly removing wet and dirty litter especially the upper layers of the litter and adding fresh clean dry litter (85.5 % DM). The control group was housed in this pen continuously, whereas each of the other groups was divided into two equal subgroups which were exposed daily for 4 or 8 h to different wet litter contents (35 %, 50 % and 65 % moisture) in separate boxes without removing the excreta from the wet litter. The different litter moisture contents
were experimentally maintained by adding water as required. Assessment of foot pads was made at days 14, 21, 28 and 35. After that three birds from each group were slaughtered for histopathological analysis.

**Scoring criteria**

The birds were examined at the beginning of the experiment at day 14, then weekly till day 35. If the feet were dirty, they were gently washed with a wet cloth and dried before scoring; only the central plantar area was scored, signs of foot pad lesions were assessed on a 7-point scale (0 = normal skin; 7 = over half of foot pad is covered with necrotic scales) according to MAYNE et al. (2007).

Due to small cellular changes occurring within the foot pad before any evidence of a lesion is present on the external foot pad surface, both foot pads of three birds from each group were assessed histologically by removing the skin of the foot pad and storing it in 10% buffered neutral formalin in a micro-cassette. Sections were prepared and processed using standard protocols for tissue processing and stained with haematoxylin and eosin. Sections were examined under a light microscope and categorised using the histopathological scoring system according to MAYNE et al. (2007).

**Measurements**

Litter samples for measuring the moisture and pH were collected at days 14, 21, 28 and 35 from 5 sites (4 peripheral and 1 central sample), then thoroughly mixed according to HOSKINS et al. (2003). A subsample of about 100 g was taken to assess the DM content. Samples were oven-dried at 103 °C for the time needed to reach constant mass. Litter pH was measured by using a pH meter. Litter ammonia was tested weekly from day 14 to 35 by using a handheld Dräger meter tube (sample tube: ammonia 5 to 70 ppm) attached to a Dräger pump (Dräger Accuro, Dräger Interservices GmbH, KST.0576, Germany). The glass Dräger tube was broken at both ends and inserted into the Dräger pump. The pump was then held about 2 to 3 cm from the litter in the middle of the pen. The extent of the discolouration within the Dräger tube was then read off the tube and recorded.

Body weight was recorded weekly at the same day of scoring individually. Feed and water intakes were measured daily on group level. Feed conversion rate was estimated on the basis of feed consumed throughout the experimental period and body weight gain of the birds.
Statistical analyses
The data was analysed using the SAS statistical software package (release 9.1, SAS Inst. Inc., Cary, NC, USA). The foot pad scores were evaluated by using the mean of both feet. Nonparametric one-way analysis was used for calculating the differences between treatments (seven independent treatment groups) in external scores and body weight. Therefore, the Kruskal-Wallis-Test for unpaired observations was used. In addition, pairwise differences at the different time points between the groups were calculated using the Wilcoxon two-sample test for unpaired observations within procedure NPAR1WAY [with exception of external scoring at d 35; histopathological scoring (see below)].

For determining the differences in external scores along several time points a single factorial analysis of variance (ANOVA) with repeated readings of the external scores of foot pads of the same animals (n=3; procedure GLM REPEATED) was carried out. Differences of means were compared pair-wise using Tukey adjustments (procedure MEANS). Normal distribution and homogeneity of variance were assumed due to the low number of animals.

Data of histopathological scoring (and comparison of groups for external scoring at d 35) were analysed in a single factorial analysis of variance (procedure GLM) according to groups and age. Differences of means were compared pair-wise (LSMEANS / TDIFF PDIFF). Normal distribution and homogeneity of variance were assumed due to the low numbers of animals.

Differences were considered to be significant when p < 0.05.

Results
The experimental groups were generally healthy and no diseases or mortalities were reported throughout the experimental period. All birds were given a coccidiostat (lasalocid-A-sodium) in the feed. No growth-promoting substances were used in any group, and no birds were otherwise medicated. Additionally, normal feed and water intakes were observed.

Animal performance
Only at day 21 significant differences were observed in body weight of birds according to litter treatment (Table 3). The growth of birds was somewhat reduced with increasing litter moisture content. At the end of the experimental period the differences in body weight
between the groups were small. The highest weight gain (61.0 g/bird/d) was observed for birds housed continuously (24 h/d) on dry litter compared to the other experimental groups (57.5; 56.2 and 55.1 g/bird/d for G2; G3 and G4, respectively). Furthermore, it was noted that birds housed continuously on dry litter showed a better feed conversion ratio (FCR) of about 1.64 compared to wet litter treatments (1.75; 1.80 and 1.84 for G2; G3 and G4, respectively). There were no differences in the feed and water intakes of birds reared on different litter treatments.

**Litter condition**

The mean of the different litter moisture contents is shown in Table (4). The highest DM content (85.5 %) was as planned for the birds reared continuously for 24 h/d on clean dry litter (control group). Conversely in the experimental groups, the wet litter was experimentally maintained by adding water as required to be (35 %, 50 % and 65 % moisture) in adjacent separate boxes without removing the excreta from the wet litter. At the beginning of the experiment there were no significant differences in the pH values of litter for all groups. Nevertheless, throughout the experimental period the pH value of the dry litter was much lower (6.04) compared to the other groups. Furthermore, the pH values were slightly lower for wet litter containing 35 % or 50 % moisture for 4 or 8 h (6.90/7.24 or 7.42/7.56, respectively) compared to high wet litter content (65 % moisture) for 4 or 8 h (7.85/8.08, respectively). In addition, by doubling the time of exposure (8 h) the pH value was slightly higher. Generally, the pH of wet litter increased as the turkeys aged with no difference in the pH of the dry litter.

Table 4 shows the mean measurements of ammonia during the experimental period. Generally, the concentrations of ammonia in the air were significantly lower for dry litter (0.7 ppm) compared to the wet litter treatments (7.18/8.08; 9.13/10.4 and 11.0/12.0 ppm for 35 %, 50 % and 65 % moisture for 4 or 8 h, respectively). In addition, levels of ammonia were far lower for wet litter containing 35 % or 50 % moisture for 4 or 8 h (7.2/8.0 or 9.1/10.4 ppm, respectively) compared to high wet litter contents (65 % moisture) for 4 or 8 h (11.0/12.0 ppm, respectively).
**Foot pad lesions**

Mean external scores are presented in Table 5 and Figure 1. There was no evidence of external lesions at the beginning of the experiment for all groups. It was observed that throughout the experiment the external scores on days 21 and 28 for birds housed continuously for 24 h/d on clean dry litter were significantly lower compared to the other treatment groups. The severity in external scores throughout the experimental period was the highest for birds reared on 65 % moisture for 8 h/d. Moreover, first marked increase of FPD was observed after exposure for 4 h to 35 % litter moisture. In addition, higher values of external FPD lesions were observed by doubling the exposure time (8 h) in most wet litter treatments (G3.1/G3.2 at d 21, d 28 and G4.1/G4.2 at d 21, d 28, d 35).

The histopathological scores were significantly lower for the control group where the birds were housed continuously on dry litter compared to the other experimental groups at days 21, 28 and 35 (Table 6 and Figure 2). At the end of the experiment (d 35) the histopathological scores for birds housed on 65 % litter moisture for 8 h/d were significantly higher compared to those in the other treatment groups. Throughout the experimental period birds exposed for 8 h/d to wet litter within each litter treatment group were markedly higher for histopathological scores compared to those exposed for only 4 h/d. Histopathological scores at d 14 were significantly lower than those on following days for all groups. A significant histopathological score difference was observed for birds housed on 65 % moisture for 4 h/d at d 21 and d 35, too. The significant differences of the histopathological scores have to be interpreted with caution due to the small number of investigated birds.

**Discussion**

No significant differences in birds’ performance were observed at the end of the experimental period. Nevertheless, the highest body weight was recorded for the control group (85.5 % litter DM). MAYNE et al. (2007) found that mean body weights for turkeys housed on dry litter were significantly higher than those on wet litter. Furthermore, there were no marked differences between the experimental groups in feed and water intakes.

Regarding the data of litter pH values, it was observed that dry litter (control group) has a much lower pH value compared to the experimental groups (Table 4). Also, pH values increased as the moisture in the litter rose. Additionally, there were no significant differences
in the DM contents and pH values of the excreta of different experimental groups (data not shown) because of the same type of diet (commercial). Hence, these results indicate that the level of moisture content in the litter plays a role in increasing the litter pH value. Ammonia is produced as a result of microbial activity on uric acid. Wet litter and high pH act like a catalyst in this process. Therefore, pH value and moisture content of the litter are two of the most important factors determining the ammonia concentration, hence influencing ammonia release. Research has demonstrated that ammonia release from litter is negligible at litter pH below 7 (REECE et al., 1985). CARR et al. (1990) has reported that wet litter can lead to high ammonia levels in broiler houses. It is clear from our results that level of NH$_3$ was much lower in dry litter (0.7 ppm) compared to wet litter (10.5 ppm). Overall, the wetter the litter the higher the concentrations of the ammonia released from litter. Already levels of NH$_3$ as low as 10 ppm can impair performance and increase susceptibility to respiratory infections (CARLILE, 1984).

Proper litter conditions have a major impact on the health of foot pads. Many authors have found positive correlations between litter quality, particularly moisture and the incidence of FPD (HARMS et al., 1977; EKSTRAND et al., 1997; YOUSSEF et al. 2009). A significant difference (p < 0.05) was found for FPD scores when comparing birds reared continuously on dry litter for 24 h/d with those housed on wet litter for 4 or 8 h/d (Tables 5 and 6) as wet litter may soften the epithelium of foot pads which results in the skin being more prone to contact dermatitis (GREENE et al., 1985; MAYNE, 2005; MAYNE et al., 2007; YOUSSEF et al., 2010). Moreover, the severity of FPD was extremely high in wet litter containing 65 % moisture. Similarly, MELUZZI et al. (2008) observed that the higher the litter moisture the higher the FPD scores in broiler. Also, MARTLAND (1984) found a positive association between high litter moisture and FPD.

The effect of exposure time was noted in this experiment. By doubling the time of exposure (8 h) the severity of FPD was slightly increased compared to those turkeys exposed to 4 h, primarily for lower litter DM content. This might be expected as prolonged contact of the foot pads to wet litter brings more irritants in the litter and excreta closer to the foot pads. YOUSSEF et al. (2010) observed that housing turkey poults on wet litter (73 % moisture) for 8 h/d results in a higher prevalence of FPD. Nevertheless it was not clear whether the high prevalence of FPD was due to the high moisture content or to the prolonged exposure time.
However, an exposure of birds to wet litter containing 35% moisture for only 4 h/d was definitely enough to develop a significant increase in external and histopathological FPD scores thus indicating that the critical moisture content in the litter may be about 35%. Both factors (moisture content/exposure time) significantly and additively influenced severity of FPD. JODAS and HAFEZ (2000) found that the maintenance of proper litter quality with a moisture content of 25-30% is probably the most important factor to lower the incidence and severity of FPD. The findings of the external lesions were similar to the histopathological findings (Tables 5 and 6). Additionally, in this study the lesions were observed only on the epidermis of the foot pads during histopathological preparation. MAYNE et al. (2006) concluded that FPD is not a response to bacterial invasion as bacteria were usually present on the surface, but not in deeper layers.

Conclusions
Generally, high litter moisture content and daily exposure to wet litter could lead to some side effects such as a decreased final body weight and hence increased FCR as well as an increased pH value of the litter and higher levels of ammonia in the poultry houses. Therefore, controlling the level of ammonia in the poultry houses could be achieved by maintaining the litter moisture content within a range of 35%. High ammonia emission was associated with high wet litter and high pH compared to dry litter (LERNER, 1996; ALCHALABI, 2002; NAGARAJ et al., 2007).

Nevertheless, the key point is that the prevalence and severity of FPD were clearly affected by the litter quality. The first marked increase of FPD lesions was observed after one week of exposure for 4 h/d at 35% litter moisture with increasing severity of FPD for higher moisture contents. Exposure of the birds to wet litter for 8 h/d in each subgroup was sufficient to worsen the FPD scores slightly compared to those exposed for 4 h/d. This increase was only significant for high litter moisture contents (65%). Nevertheless, both factors (moisture content/exposure time) significantly and additively influenced severity of FPD. However, even short exposure to wet litter around feeding or drinking places may result in a markedly increased prevalence and severity of FPD. The significant differences in most cases in external and histopathological scores between the control group and an exposure to the different wet litter types for only 4 h emphasise the superior position of the first 4 h of
exposition for the development of lesions. After all, feed intake alone takes a few hours a day (MASIC et al., 1974) and therefore exposure to higher litter moisture for less than 4 h around feeding and drinking places cannot be avoided. Thus, testing the effect of a shorter exposure time to wet litter is invalid.

Out of this observation, it can be concluded that the critical litter moisture content for the development of marked changes in external and histological scores of food pads is about 35 % or even lower. According to clinical signs of disease this level has to be interpreted with caution. MAYNE et al. (2007) suggested that birds with medium to severe histopathological lesions (score > 4) of foot pads may experience discomfort during locomotion. In severe cases, ulcers (score 6-7) can impair birds’ gaits (MARTRENCHAR et al. 2002). In this study even for higher moisture contents there were no clinically obvious symptoms for the impairment of birds’ health. According to the increased FPD scores at longer exposure time on wet litter, it has to be emphasised that higher litter moisture contents (>35 %) have to be avoided. In addition, it has to be mentioned that depending on the severity of FPD, the lesions are either limited to the epidermis or reach deep into the dermis and subcutis. In this study, the lesions were observed only on the epidermis of the foot pads. This may also be a reason why under clinical aspects no health impairments in the different treatment groups were observed.

**Summary**

This experimental study aimed to find out the “critical moisture content” of litter which results in higher development and severity of FPD in young fattening turkeys. Four groups of two weeks old ♀ turkeys were reared on dry wood shavings during three weeks. The control group was housed in this pen continuously, whereas each other group was divided into two equal subgroups and exposed daily for 4 or 8 h to different wet litter contents (35 %, 50 % and 65 % moisture) in adjacent separated boxes. Foot pads were assessed weekly for external and for histopathological scoring (MAYNE et al. 2007). The results revealed that FPD severity was much higher for wet litter compared to dry litter. The severity of FPD rose slightly with an increased moisture content in the litter. External FPD scores were lower about 1.3 and 1.6 for birds exposed to wet litter (35 % moisture for 4 or 8 h, respectively) than for those exposed to 50 % or 65 % moisture for 4 or 8 h (1.83/2.33 and 2.66/4.16, respectively).
Furthermore doubling exposure time (4\rightarrow 8 \text{ h}) in each wet litter treatment led to only slightly increased severity of FPD for the lower litter moisture (35 and 50 \% moisture) and a higher rise for the wettest litter treatment (65 \% moisture) at the end of the trial. Predominantly even an exposure of \leq 4 \text{ h} per day might result in forced severity of FPD. Moreover, at the end of the experiment no significant difference was observed for the body weight between the experimental groups.

As a result of this study it is assumed that the critical moisture content for the development of FPD lesions is about 35 \% moisture content in litter or even slightly less. However, for clinical signs of FPD even higher moisture contents of litter higher pressure load on birds’ food pads at the end of a fattening period are necessary.

**Keywords**

Litter moisture; exposure time; foot pad dermatitis; turkey

**Zusammenfassung**

Ziel der vorliegenden Untersuchungen war es, den kritischen Feuchtegehalt in der Einstreu für Puten zu finden, bei dessen Überschreiten mit einer erhöhten Entwicklung und Intensität einer Fußballenentzündung zu rechnen ist. Insgesamt wurden 75 weibliche Puten in einem Alter von 14 Tagen auf eine Kontroll- und drei Versuchsgruppen aufgeteilt. Letztere wiederum wurden in jeweils zwei Untergruppen aufgeteilt, die täglich entweder für 4 \text{ h} oder 8 \text{ h} unterschiedlichen Feuchtegehalten der Einstreu ausgesetzt waren (35 \%, 50 \%, 65 \% Feuchtigkeit). Sowohl makroskopische als auch histologische Untersuchungen der Fussballen wurden nach MAYNE et al. (2007) in wöchentlichen Abständen, beginnend am Tag 14 und endend am Tag 35, durchgeführt. Aus den Untersuchungen geht klar hervor, dass der Schweregrad der Fußballendermatitis unter dem Einfluss steigender Feuchtegehalte der Einstreu kontinuierlich zunimmt. Zum Versuchsende hatten die Tiere in der Kontrollgruppe die geringsten makroskopischen Scorewerte (0,50), während in den Versuchsgruppen der Anstieg in Abhängigkeit von Feuchtegehalt und Expositionsdauer (4 h/8 h) deutlich hervortrat (35 \% Feuchtigkeit: 1,33 bzw. 1,66; 50 \% Feuchtigkeit: 1,83 bzw. 2,33; 65 \% Feuchtigkeit: 2,66 bzw. 4,16). In der Körpermassenentwicklung waren am Versuchsende keine Unterschiede erkennbar. Aus den vorliegenden Untersuchungen kann geschlossen werden,

**Stichworte**
Feuchtigkeit der Einstreu; Expositionszeit; Fußballenentzündung; Puten

**References**


GROSSE LIESNER, B. B., 2007: Comparative investigations on the performance and appearance (incidence and manner) of primarily non-infectious health problems in male fattening turkeys of five different strains. Dissertation, University of Veterinary Medicine, Hannover, Germany.


MARTLAND, M. F., 1984: Wet litter as a cause of plantar pododermatitis, leading to foot ulceration and lameness in fattening turkeys. Avian Pathology 13, 241-252.


Correspondence: Prof. Dr. J. Kamphues, Institut für Tierernährung, Stiftung Tierärztliche Hochschule Hannover, Bischofsholer Damm 15, D-30173 Hannover, Germany; E-Mail: josef.kamphues@tiho-hannover.de
Table 1. Composition (%) and chemical analyses (g/kg as fed) of the experimental diet fed to turkeys during the experimental period (d14 – d35)

<table>
<thead>
<tr>
<th>Ingredients (%)</th>
<th>Chemical Composition (g/kg)</th>
<th>Macrominerals (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat</td>
<td>39.8 DM 875</td>
<td>Ca 12.8</td>
</tr>
<tr>
<td>Soybean meal (heat treated)</td>
<td>35.2 crude ash 65.9</td>
<td>Ca 12.8</td>
</tr>
<tr>
<td>Yellow corn</td>
<td>11.0 crude protein 272 P 7.15</td>
<td></td>
</tr>
<tr>
<td>Soybean (full fat, heat treated)</td>
<td>5.00 crude fat 53.8 Mg 1.69</td>
<td></td>
</tr>
<tr>
<td>Fish meal</td>
<td>3.00 crude fibre 21.4 Na 1.40</td>
<td></td>
</tr>
<tr>
<td>Mixed fat1</td>
<td>1.60 NfE 462 Cl 1.86</td>
<td></td>
</tr>
<tr>
<td>CaCO3</td>
<td>1.90 starch 313 K 10.2</td>
<td></td>
</tr>
<tr>
<td>Monocalciumphosphate</td>
<td>1.90 sugar 46.5 S 3.32</td>
<td></td>
</tr>
<tr>
<td>NaCl</td>
<td>0.200 ME4 (MJ/kg) 11.9 Microminerals (mg/kg)</td>
<td></td>
</tr>
<tr>
<td>Methionine</td>
<td>0.070 threonine 9.06 Cu 21.7</td>
<td></td>
</tr>
<tr>
<td>Lysine</td>
<td>0.140 tyrosine 9.43 Zn 116</td>
<td></td>
</tr>
<tr>
<td>Vitamin mixture2</td>
<td>0.050 cystine 5.54 Fe 257</td>
<td></td>
</tr>
<tr>
<td>Trace elements mixture3</td>
<td>0.090 methionine 5.55 Mn 104</td>
<td></td>
</tr>
<tr>
<td>Coccidiostat premix</td>
<td>0.050 lysine 16.5 Se 0.320</td>
<td></td>
</tr>
</tbody>
</table>

1 Soybean, palm, canola, sunflower oil
2 Vitamin mixture supplies the following per kilogram of the diet: vitamin A, 13000 IU; vitamin D3, 4000 IU; 25-hydroxycholecalciferol, 0.0250 mg; vitamin E, 100 mg
3 Trace elements supplies the following per kilogram of diet: copper, 12 mg; iron, 75 mg; zinc, 75 mg; manganese, 90 mg; iodine, 1.8 mg; selenium, 0.3 mg; cobalt, 0.04 mg
4 ME calculated by using the official formula for complete diets: MEn (MJ/kg) = 0.01551 crude protein + 0.03431 crude fat + 0.01669 starch + 0.01301 sugar (nutrients in g/kg diet; FMVO, annex 4)
Table 2. Experimental design regarding exposure time to different litter moisture contents

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>animals (n)</th>
<th>exposure to dry litter (h)</th>
<th>DM %1)</th>
<th>exposure to wet litter (h)</th>
<th>moisture %1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td></td>
<td>12</td>
<td>24</td>
<td>85.5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>G2</td>
<td>G2.1</td>
<td>21</td>
<td>20</td>
<td>85.5</td>
<td>4</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>G3</td>
<td>G3.1</td>
<td>21</td>
<td>20</td>
<td>85.5</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>G4</td>
<td>G4.1</td>
<td>21</td>
<td>20</td>
<td>85.5</td>
<td>4</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td>8</td>
</tr>
</tbody>
</table>

1) as planned

Table 3. Comparison of turkey’s body weight (g) at different times (Mean ± SD)

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>day of life (duration of treatments)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>14 (0) (n=12; n*=21)</td>
</tr>
<tr>
<td>G1</td>
<td></td>
<td>433 ± 56.3</td>
</tr>
<tr>
<td>*G 2</td>
<td>G2.1</td>
<td>454± 53.9</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td></td>
</tr>
<tr>
<td>*G 3</td>
<td>G3.1</td>
<td>458 ± 40.4</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td></td>
</tr>
<tr>
<td>*G 4</td>
<td>G4.1</td>
<td>451 ± 42.3</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td></td>
</tr>
<tr>
<td>p-value Kruskal-Wallis</td>
<td>0.8164</td>
<td>0.0376</td>
</tr>
</tbody>
</table>

for calculating differences in body weight at the beginning of the trial, the animals sacrificed were considered; in G2, G3 and G4 added to both subgroups

A,B Means in the same column with different superscripts are significantly different (p < 0.05)
Table 4. DM content (%), pH values of the litter and NH$_3$ (ppm) in the air (3 cm above floor) during the experiment (Mean ± SD)

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>DM</th>
<th>pH</th>
<th>NH$_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 1</td>
<td></td>
<td>85.5 ± 2.03</td>
<td>6.04 ± 0.081</td>
<td>0.725 ± 0.35</td>
</tr>
<tr>
<td>G 2</td>
<td>G2.1</td>
<td>64.8 ± 0.559</td>
<td>6.90 ± 0.875</td>
<td>7.18 ± 5.57</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td>65.2 ± 0.487</td>
<td>7.24 ± 1.22</td>
<td>8.08 ± 6.36</td>
</tr>
<tr>
<td>G 3</td>
<td>G3.1</td>
<td>49.7 ± 0.399</td>
<td>7.42 ± 1.04</td>
<td>9.13 ± 6.96</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td>50.1 ± 0.537</td>
<td>7.56 ± 1.02</td>
<td>10.4 ± 7.49</td>
</tr>
<tr>
<td>G 4</td>
<td>G4.1</td>
<td>34.8 ± 0.366</td>
<td>7.85 ± 1.26</td>
<td>11.0 ± 8.02</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td>35.2 ± 0.385</td>
<td>8.08 ± 1.32</td>
<td>12.0 ± 8.55</td>
</tr>
</tbody>
</table>

no statistical analysis due to small number of samples

Table 5. External foot pad scores of young turkeys throughout the experiment (Mean ± SD)

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>14 (0) (n=12; n*=21)</th>
<th>21 (7) (n=9)</th>
<th>28 (14) (n=6)</th>
<th>35 (21) (n=3)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 1</td>
<td></td>
<td>0.000 ±0.000</td>
<td>0.222 ±0.363</td>
<td>0.500 ±0.447</td>
<td>0.500 ±0.500</td>
<td>0.2853</td>
</tr>
<tr>
<td>*G 2</td>
<td>G2.1</td>
<td>0.000 ±0.000</td>
<td>0.944 ±0.300</td>
<td>1.13 ±0.204</td>
<td>1.33 ±0.288</td>
<td>0.0017</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td>1.22 ±0.441</td>
<td>1.25 ±0.273</td>
<td>1.66 ±0.288</td>
<td></td>
<td>0.0185</td>
</tr>
<tr>
<td>*G 3</td>
<td>G3.1</td>
<td>0.000 ±0.000</td>
<td>1.27 ±0.363</td>
<td>1.33 ±0.408</td>
<td>1.83 ±0.288</td>
<td>0.0010</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td>2.05 ±0.463</td>
<td>2.16 ±0.258</td>
<td>2.33 ±0.288</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>*G 4</td>
<td>G4.1</td>
<td>0.000 ±0.000</td>
<td>2.11 ±0.416</td>
<td>2.42 ±0.376</td>
<td>2.66 ±0.288</td>
<td>0.0010</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td>3.05 ±0.583</td>
<td>3.75 ±0.689</td>
<td>4.16 ±1.04</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
</tbody>
</table>

** Means in the same row with different superscripts are significantly different (p < 0.05). Comparison between the different time points only made for animals surviving to the end of the trials (repeated measurements of the same animals)

A,B Means in the same column with different superscripts are significantly different (p < 0.05)
Table 6. Histopathological foot pad scores of young turkeys throughout the experiment (Mean ± SD)

_Histopathologische Scores der Fußballen der jungen Puten im Versuchsverlauf_

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>day (duration of treatments)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>14 (0) (n=3)</td>
<td>21 (7) (n=3)</td>
</tr>
<tr>
<td>G 1</td>
<td></td>
<td>0.000±0.000</td>
<td></td>
</tr>
<tr>
<td>G 2</td>
<td>G2.1</td>
<td>0.000±0.000</td>
<td>3.66±1.15</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td>4.16±0.763</td>
<td>4.50±0.500</td>
</tr>
<tr>
<td>G 3</td>
<td>G3.1</td>
<td>0.000±0.000</td>
<td>4.50±0.500</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td>5.00±0.000</td>
<td>5.33±0.577</td>
</tr>
<tr>
<td>G 4</td>
<td>G4.1</td>
<td>0.000±0.000</td>
<td>5.16±0.288</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td>5.50±0.500</td>
<td>6.00±0.500</td>
</tr>
</tbody>
</table>

\[a,b\] Means in the same row with different superscripts are significantly different (p < 0.05)

\[A,B\] Means in the same column with different superscripts are significantly different (p < 0.05)
Figure 1. External foot pads scores of young turkeys at the end of the experimental period (d 35) in relation to different exposure times to varying litter moisture (Mean ± SD); lowercase letters indicate significant differences with p < 0.05 between the groups.

*Makroskopische Scores der Fußballen der jungen Puten am Versuchsende (Tag 35) in Abhängigkeit von der Einstreufeuchte und Expositionsduer*
Figure 2. Histopathological foot pads scores of young turkeys at the end of the experimental period (d 35) in relation to different exposure times to varying litter moisture (Mean ± SD); lowercase letters indicate significant differences with p < 0.05 between the groups.

Histopathologische Scores der Fußballen der jungen Puten am Versuchsende (Tag 35) in Abhängigkeit von der Einstreufeuchte und Expositionsduauer
4. Chapter 2

Effects of floor heating and litter quality on the development and severity of foot pad dermatitis in young turkeys
4. Chapter 2

*Floor heating-Foot pad dermatitis*

**Effects of floor heating and litter quality on**

**the development and severity of foot pad dermatitis in young turkeys**

A. ABD EL-WAHAB¹, C. F. VISSCHER¹, A. BEINEKE², M. BEYERBACH³, J. KAMPHUES¹

¹ Institute of Animal Nutrition, University of Veterinary Medicine Hannover, Foundation, Germany
² Institute of Pathology, University of Veterinary Medicine Hannover, Foundation, Germany
³ Institute of Biometry and Information Processing, University of Veterinary Medicine Hannover, Germany

*Published in Journal of Avian Diseases 55:429-434, 2011*

Corresponding author:

Prof. Dr. J. Kamphues, Institut für Tierernährung, Stiftung Tierärztliche Hochschule Hannover, Bischofsholer Damm 15, D-30173 Hannover, Germany
Tel./Fax:+49 511 8567301   Fax: +49 511 7698
E-Mail: josef.kamphues@tiho-hannover.de
Running title: Floor heating-Foot pad dermatitis
SUMMARY

Actions concerning animal health in the turkey production are coming more and more to the fore. Litter quality has a great impact on the bird’s health and welfare. This study aimed at evaluating effects of using floor heating, different litter materials and exposure to litter with “critical moisture content” of 35 % for 16 or 24 hr/d on the severity of foot pad dermatitis (FPD), a widespread disease in fattening turkeys. Four groups of 2-wk-old ♀ turkeys were reared during 3 wk with 20 birds in each. All turkeys were fed a commercial pellet diet ad libitum. The first 2 groups were kept on wood shavings (35 % moisture) without and with floor heating. The other 2 groups were housed on lignocellulose (Soft Cell®) of 35 % moisture without and with floor heating. In each group half of birds were housed for 8 h/d in adjacent separate boxes where the litter was kept clean and dry throughout the experimental period. Foot pads were assessed weekly for external and at d 35 for histopathological scoring (scores: 0=healthy/7=ulcer). At d 14 each bird had normal/healthy foot pads. The results indicates that using floor heating resulted in significantly lower FPD scores (0.8 ± 0.2) compared to groups without floor heating (2.0 ± 0.8). Using lignocellulose as a litter material resulted in significantly lower histopathological FPD scores (1.4 ± 0.7) compared with wood shavings (1.7 ± 0.8). In all birds housed on dry litter for 8 h/d, significantly lower FPD scores were found compared with birds housed on wet litter for 24 hr. In conclusion, using floor heating even with wet litter (35 % moisture), independent of the litter type resulted in reduced severity of FPD compared with those birds housed in pens without using floor heating. Additionally, using lignocellulose as a litter material resulted in lower FPD compared to wood shavings. Keeping litter dry and “warm” could be achieved by using floor heating which considered as a practical step to enhance animal health and welfare.

Key words: foot pad dermatitis; floor heating; litter type; young turkeys
Abbreviations: B.U.T. = British United Turkeys; DM = dry matter; FCR = feed conversion ratio; FPD = foot pad dermatitis
5. Chapter 3

Effects of high electrolyte contents in the diet and using floor heating on development and severity of foot pad dermatitis in young turkeys
Chapter 3

5. Chapter 3

Effects of high electrolyte contents in the diet and using floor heating on development and severity of foot pad dermatitis in young turkeys

Short title: Dietary electrolyte and foot pad dermatitis in young turkeys

A. Abd El-Wahab*, C. F. Visscher*, A. Beineke#, M. Beyerbach§, J. Kamphues*

* Institute of Animal Nutrition, University of Veterinary Medicine Hannover, Foundation, Germany
# Institute of Pathology, University of Veterinary Medicine Hannover, Foundation, Germany
§ Institute of Biometry and Information Processing, University of Veterinary Medicine Hannover, Foundation, Germany

Accepted for publication in Journal of Animal Physiology and Animal Nutrition, 2011
(Officially accepted at 30.08.2011, in press)

Correspondence: Prof. Dr. J. Kamphues, Institut für Tierernährung, Stiftung Tierärztliche Hochschule Hannover, Bischofsholer Damm 15, D-30173 Hannover, Germany; Tel.: 0049 511 856-7301 Fax: 0049 511 856-7698
E-Mail: josef.kamphues@tiho-hannover.de
Introduction

Sodium and potassium are essential for all animals (Suttle, 2010). The effects of dietary sodium and potassium levels on water intake and excreta moisture are well documented and there is wide agreement among authors that an excess of these nutrients in poultry diets increases excreta moisture (Francesch and Brufau, 2004) resulting in “wet litter conditions”. It was observed that a higher water intake occurred in birds fed diets containing 8.00-9.00 g K/kg and 2.00 g Na/kg compared with diets containing 7.00 g K/kg and 2.00 g Na/kg and the excreta moisture was highly correlated with dietary K content (Eichner et al., 2007). In diet formulation it is easy to achieve a low Na content but using normal protein sources often results in K levels >10 g/kg diet. Therefore, dietary factors such as the proportion of soybean meal and amounts of oligosaccharides have to be considered. Potassium itself and salt in feed force “wet litter conditions”(Youssef et al., 2011). Consequently, a higher incidence of foot pad dermatitis (FPD) may indirectly depend on definite dietary factors (Smith et al., 2000; Eichner et al., 2007; Bilgili, 2009). Nevertheless, an increase in Cl did not have the same effect as sodium and potassium on water intake and excreta moisture (Murakami et al., 2001). The low prevalence and severity of FPD are of great concern regarding animal welfare, the birds’ performance and product quality. FPD is a type of contact dermatitis with hyperkeratosis in the early stage followed by necrosis and ulcers of the foot pads in the late stage (Ekstrand et al., 1997). At the end of the fattening period this disease can reach a prevalence of 91-100 % in turkeys (Hafez et al., 2004). About 97.2 % of turkeys showed FPD lesions with no marked effects on the body weight of five different strains of male turkeys at the end of the fattening period (Grosse Liesner, 2007). The rate of FPD incidence per farm is gaining recognition as a well-being indicator (Bradshaw et al., 2002; Martrenchar et al., 2002). The aetiology of FPD is a complex interaction of different factors. Some of these are related to dietary factors which may affect water consumption and excretion. Other factors are related to management and housing (litter quality, type of litter, stocking density and drinking system). Finally, there are factors related to diseases caused by various infections (Mayne, 2005). Moreover, Youssef et al. (2010) noted that housing turkey poult s on high wet litter (73 % moisture) for only 8 h/d (over 3 weeks) led to imposingly high severity of external FPD scores. Furthermore, Abd El-Wahab et al. (2011a) observed that the first marked increase of FPD lesion in turkey poult s occured after exposure for only 4 h/d to a “critical litter moisture”
of 35 %. It might be that water softens the epidermis of the foot pad, resulting in more susceptibility to FPD (Jensen et al., 1970). Furthermore, Berg and Algers (2004) noted that the prevalence of FPD for floor heating groups was 21.5 % ± 3.70 vs. 45.0 % ± 7.10 for groups without floor heating. Similarly, Abd El-Wahab et al. (2011b) observed that using floor heating resulted in significantly lower FPD scores (0.860 ± 0.273) vs. (2.00 ± 0.872) for groups not using floor heating. Therefore, this study aimed to test the effects of normal vs. high Na and K contents in the diet with or without floor heating as well as exposure to wet litter with 35 % moisture on the development and severity of FPD in young turkeys. (Keywords: electrolytes; FPD; floor heating; young turkeys)

Materials and methods

Housing

Ninety one-day-old female turkey poults (BUT-Big 6) were obtained from a commercial hatchery. The birds were housed in a floor pen littered with wood shavings kept dry and clean before the beginning of the experiment by daily removal of the upper layers of the litter replacing it with fresh dry litter. During the first 3 days additional feed was offered (about 300 g/d) on paper to accustom the birds to the feed. All turkeys were fed commercial diets (pellets) ad libitum during the first 2 weeks of life (starter/grower diet: 1st/2nd week). The pen were equipped with an infra red lamps, to achieve a temperature of about 34-36 °C at the outset in the vicinity of the one-day-old birds. The temperature was lowered by about 1 °C every 2 days, reaching about 20 °C at d 35. The photoperiod from d 4 was 16 h of light and 8 h of darkness.

Experimental design

The experimental design of this study is summarised in Table 1. At the beginning of the experimental period (d 14) ten birds were killed for foot pad histopathological assessment. The remaining birds, 80 in total, were labelled and then divided into 4 equal groups housed in floor pens (1.50 m x 1.32 m). Each floor pen was littered with wood shavings (1.00 kg/m²; 86.8 % ± 0.340 DM). The experiment was conducted over a period of 3 weeks. Throughout this period, the first 2 groups were fed normal levels of Na and K (1.60 and 7.80 g/kg diet, respectively), being with and without floor heating. The
other two groups were fed a surplus level of Na and K (3.10 and 15.3 g/kg diet, respectively), being with and without floor heating. The composition of the experimental diets fed to the turkey poults and the chemical analysis of each according to the groups are shown in Tables 2 and 3. Bolus alba (kaolin) was added to the control diet to equalise the crude ash, other nutrients and energy content. Samples of both diets were analysed by standardised laboratory methods according to VDLUFA (2004). The temperature at the litter surface was about 35 °C in groups with floor heating vs. 25 °C in groups without floor heating. Electrical floor heating was used to achieve the required litter surface temperature. The room temperature was nearly the same for all groups (25.3 °C). Half of the birds in each group (n=10) were additionally exposed to wet litter (35 % moisture) for 4 h/d in adjacent separate boxes, experimentally maintained by adding water as required (every 2 days). External assessment of foot pads was done at d 14, 21, 28 and 35. At d 35 all birds were killed for histopathological scoring.

**Scoring criteria**

The external examination was performed on all birds at the beginning of the experiment at d 14, then weekly till d 35. If the feet were dirty, they were gently washed with a wet cloth and dried before scoring; only the central plantar was scored, signs of foot pad lesions being recorded on a 7-point scale (0 = normal skin; 7 = over half of the foot pad is covered with necrotic scales) according to Mayne et al. (2007).

Due to small cellular changes occurring within the foot pad before any evidence of a lesion was present on the external foot pad surface, both foot pads of all sacrificed birds were assessed histologically by removing the skin from the foot pad and storing it in 10 % buffered neutral formalin in a micro-cassette. Sections were prepared and processed using standard protocols for tissue processing and stained with haematoxylin and eosin. Sections were examined under a light microscope and categorised using the histopathological scoring system on a 7-point scale (0 = normal epidermis; 7 = more than one rupture or “ulcer” of the epidermis) according to Mayne et al. (2007).

**Measurements**

Litter samples for measurement (moisture and pH) were collected at d 14, 21, 28 and 35 from 5 sites (the 4 corners and the middle of the pen), then thoroughly mixed. Sub-samples of about 100 g were taken to assess moisture content by drying at 103 °C until a constant mass
was obtained (VDLUFA, 2004). The litter pH was measured by making a suspension (1 part of material: 9 parts of water) then by using a pH meter (WTW, Weilheim, Germany). The fresh excreta of the birds were collected from each pen once a week by putting a plastic sheet in each pen for about 1 hour until about 70-100 g fresh pure excreta per pen had been obtained. The collected excreta were then removed from each pen, thoroughly mixed and divided into two parts: the first part for measuring the pH, the second part being dried at 103 °C to determine the DM content. After drying the excreta, the samples were ground and analysed for crude ash and macrominerals (Na and K: flame photometer, Unicam, Dreieich, Germany; Cl: using a chloride analyser-coulombic titration, IMA, Giessen, Germany).

Individual body weight was recorded weekly on the day of scoring. It has to be emphasised that each bird in all groups were marked throughout the experimental period. Feed and water intakes were measured daily at group level. Feed conversion ratio (FCR) was estimated on the basis of feed consumed throughout the experimental period as well as body weight gain of the birds. At the end of the experiment, samples of the whole litter material were freeze-dried, ground and analysed for uric acid content with chemical kits using the enzymatic-photometric method by (UA Plus, Roche, Mannheim, Germany) and for nitrogen content using vario MAX CNS (Elementar Analysensysteme GmbH, Hanau, Germany).

Statistical analyses

The foot pad scores were evaluated by using the mean of both feet. The external and histopathological foot pad scoring and body weight data were analysed separately for each sampling point (d 14, 21, 28 and 35) using the GLM procedure of the SAS® statistical software package (SAS, 2002). For external and histopathological scores the RANK procedure was used beforehand to compute ranks. Treatment means for all measurements were tested pair-wise using t-distributed statistics for independent samples generated by the TDIFF-Option in the LSMEANS statement from SAS procedure GLM.

For calculating differences in external FPD scores at group level between two time points, for normal distribution the t-test for paired observations was used. Otherwise the Wilcoxon’s signed-rank test within procedure UNIVARIATE was implemented. All statements of statistical significance are based upon p < 0.05. However, it has to be stressed that feed and
water intakes were estimated on group level, and therefore FCR can not be statistically analysed because n=1.

**Results**

The experimental diet contained about twice the concentration of electrolytes of the control diet. Regarding other nutrients (crude ash, protein, amino acids and trace elements) as well as energy density both diets were almost identical (Table 3).

**Animal performance**

Using floor heating resulted in higher water:feed intake ratio (2.91 and 3.62) vs. (2.33 and 2.84) for groups without floor heating (Table 4). Birds fed a normal diet and exposed to wet litter with 35 % moisture for 4 h/d had a significantly higher (1857 g ± 167) end body weight compared with those fed a high electrolytes diet and housed for 24 h/d without floor heating (1693 g ± 216). Nevertheless, no significant differences were found in the end body weight at d 35 among the other experimental groups (Table 5).

**Excreta analysis**

The dietary factors (surplus of electrolytes) markedly changed the composition of excreta especially that related to stimulated water intake as described previously. The marked differences in diet composition (Na, K levels) were reflected to the same degree in the excreta (Table 6).

**Litter conditions**

Feeding the high electrolytes diet in absence of floor heating resulted in much higher litter moisture (37.5 % DM) compared with the normal dietary Na and K levels (64.5 % DM content of litter) at d 35. By using floor heating the lowest litter moisture content during the experimental period for birds fed normal or high electrolytes diets (85.6; 69.7 % DM, respectively) at d 35 was observed (Figure 1). Additionally, using floor heating resulted in lower total litter weights at the end of the experiment (d 35) for birds fed normal or high electrolytes diets compared with those groups without floor heating (12.2 and 14.6 kg vs. 16.6; 25.8 kg, respectively).
Feeding the surplus of electrolytes diet showed the highest litter pH value (7.74) in comparison with those groups fed the normal electrolytes diet (6.50). Using floor heating led to the lowest levels of litter pH values (6.16 and 6.25) regardless of the type of diet consumed (normal or high electrolytes). In addition, feeding the high electrolytes diet and the absence of floor heating resulted in the lowest litter contents of nitrogen and uric acid (17.8; 10.9 g/kg DM for nitrogen and uric acid, respectively). Floor heating led to the highest litter content for nitrogen and uric acid regardless of the type of diet consumed (44.3; 36.3 g/kg DM for nitrogen and 26.9; 24.5 g/kg DM for uric acid, respectively).

Foot pad lesions
The development of foot pad lesions (external and histopathology) was markedly influenced by two factors: diet and floor heating (Table 7). Feeding the high electrolytes diet resulted in significantly higher external (3.65 ± 1.03) and histopathological (4.26 ± 1.25) FPD scores in comparison with those for birds being fed normal electrolyte levels. Furthermore, using floor heating resulted in significantly lower external (2.36 ± 0.588) and histopathological (2.76 ± 0.575) FPD scores compared with groups without floor heating. It should be emphasised that at the beginning of the experiment (d 14) there were no alterations in foot pads for all birds.

Feeding normal electrolyte levels in absence of floor heating led to significantly higher histopathological FPD scores for birds exposed to wet litter for 4 h/d compared with birds housed continuously without any exposure to wet litter (3.70 ± 0.632 vs. 3.15 ± 0.412, respectively). Similarly, by feeding normal electrolyte levels with floor heating showed significantly higher histopathological FPD scores for birds exposed to wet litter for 4 h/d compared with birds housed continuously without any exposure to wet litter (2.65 ± 0.412 vs. 2.10 ± 0.218, respectively) (Table 8). Generally, daily exposure to wet litter for 4 h tended to lead to increased FPD scores (except for group 3, Table 8).

In pens where birds were housed continuously, using floor heating resulted in significantly lower external and histopathological FPD scores compared with those groups without any floor heating and fed either normal or surplus electrolyte levels.

Generally, feeding high electrolyte levels and an absence of floor heating showed significantly higher FPD scores compared with the other experimental groups. In contrast,
feeding a normal electrolytes diet and using floor heating resulted in significantly lower external FPD scores compared with the other experimental groups.

Discussion
With further intensification and high performance goals to be achieved in the modern poultry production, improving the excreta/litter quality may increase not only the health and well-being of birds but also the economic profits in the commercial poultry industry (Francesch and Brufau, 2004). For poultry to be able to perform their growth capacities to the full, they should be well looked after and kept in good environmental conditions including the litter that is affected by a number of dietary, management and housing measures. Bad quality of litter increased the prevalence and severity of FPD and hence could be used as one of the indicators for animal welfare, one of the important aspects of animal production (Berg, 1998).

In this study, feeding normal dietary electrolytes seems to favor the end body weight. In contrast, feeding a high electrolytes diet or using floor heating did not result to marked differences in the final body weights. Also, Grosse Liesner (2007) observed no marked effects of FPD on the body weight of turkeys at the end of the fattening period. However, Mayne et al. (2007) observed that mean body weights for turkeys housed on dry litter were significantly higher than those on wet litter. As expected the addition of kaolin to the normal diet did not result in marked changes of body weight compared with groups where no kaolin was added. Katouli et al. (2010) observed that a diet with 1.5 % kaolin resulted in no significant differences in weight gain or FCR in broilers till wk 5 of rearing period in broiler compared with the control group.

Litter quality
Litter moisture is a predominant factor that characterizes litter quality (Mayne, 2005; Youssef et al., 2010). Many factors impact water intake in poultry, but for feed formulations based on common ingredients, electrolytes play a major role. The dietary concentrations of Na and K are of special interest as they are absorbed efficiently, and have to be excreted via urine resulting in an increase in the water intake. Therefore, the high moisture content of excreta may be caused by high electrolytes intake. Oviedo-Rondon et al. (2001) reported that higher levels of Na in the diet might increase litter moisture, whereas an increase in Cl did not have
the same effect (Murakami et al., 2001). Water intake increases linearly with the dietary electrolyte balance (DEB) increases and the increase of water intake was also reflected in a progressive increase in litter moisture. This principle supports the litter moisture responses obtained in this study. Absence of floor heating showed much higher litter moisture content, reflected the highest total weight of the litter at the end of the experimental period compared with groups using floor heating, which could be associated with many environmental and management problems. Furthermore, wet litter was associated with a higher pH compared with dry litter (Lerner, 1996; Nagaraj et al., 2007). Litter pH is an interesting parameter regarding environmental aspects (Hooge, 1995), but not FPD aspects. It is well known that feeding high electrolytes levels render an alcalogenic diet, that increases urinary pH (hence excreta pH), results in alkaline litter pH (7.31±0.920 vs. 6.50±0.480 for feeding normal diet), as expected due to markedly increased dietary electrolyte balance (DEB). On the contrary, Martland (1985) found a lower pH in the wet litter which had a higher prevalence of FPD than in the dry litter.

**Severity of FPD**

Feed formulation can affect the incidence and severity of FPD. However, litter moisture is considered to be a leading factor causing FPD (Jensen et al., 1970). Thus, FPD can be kept at a minimum if proper litter management is practised. The high prevalence and severity of FPD were correlated to high litter moisture (Bilgili, 2009). Based on the feed composition, Na and K levels play a major role due to increased electrolytes intake and moisture in the litter predisposing the birds to FPD. Feeding a high electrolytes diet and the absence of floor heating resulted in significantly higher FPD scores. Therefore, factors that increase water excretion in birds are expected to increase the incidence and severity of FPD. It is possible that wet litter softens the epithelium of foot pads, this resulting in the skin being more prone to contact dermatitis (Greene et al., 1985; Mayne, 2005). Thus, the wet litter appears to be the major factor leading to FPD (Mayne et al., 2007). Our findings tally with Youssef et al. (2010) who observed that high potassium levels (12 g/kg diet) resulted in a marked increase in the severity of FPD. Also, Youssef et al. (2011) found that the high macromineral intake (17.1 Ca, 7.73 P, 2.79 Mg, 2.32 Na, 4.58 Cl g/kg diet) did not influence foot pad scores on dry litter compared with the low levels (6.65 Ca, 4.43 P, 1.40 Mg, 1.12 Na, 3.16 Cl g/kg diet).
Nevertheless in this study, using floor heating for birds fed high electrolytes levels resulted in significantly lower external FPD scores compared with birds fed a high electrolytes diet in absence of floor heating. Despite elevated water intake by feeding high electrolyte levels, the litter became drier when floor heating was in use. Therefore, floor heating is likely to be highly effective in significantly reducing the development and severity of FPD. Abd El-Wahab et al. (2011b) concluded that using floor heating resulted in a decrease in the severity of FPD (0.950 ± 0.158) despite of exposure to wet litter (35% moisture) for 24 h/d vs. (2.55 ± 0.832) for the group without floor heating. The significant effect of using floor heating on FPD scores could be due not only to the litter becoming dry like fresh litter but also might be due to the effect of temperature. On the one hand, floor heating might lead to “warm foot pad” causing vasodilatation of the blood vessels, increasing the blood flow and promoting healing. The principle of warming effect on blood flow in the legs was also stated by Nisha (2003) causing vasodilatation of the blood vessels with increasing the blood flow in humans. On the other hand with the absence of floor heating the litter is quite cool and this might lead to blood vessel constrictions in the foot pad with a “cold wet foot pad”. The source of warming in turkey houses hangs above the pens; so the upper surface of litter is warm, but the bottom can have a reduced temperature. Our findings tally with those of Berg and Algiers (2004) who found that using floor heating had a significant effect on FPD with a prevalence of 21.5 % ± 3.70 for floor heating groups vs. 45.0 % ± 7.10 for groups not using floor heating.

In addition, exposure of birds to wet litter (35 % moisture) for 4 h/d resulted in higher FPD scores compared with those housed continuously on a litter without any exposure to wet litter (except for group 3, Table 8). Abd El-Wahab et al. (2011a) concluded that exposure of birds for only 4 h/d to wet litter (35 % moisture) was sufficient to markedly increase the severity of FPD (1.33 ± 0.288). This suggests that even brief exposure to wet litter (for example, around feeding or drinking places) might result in a markedly increasing the development and severity of FPD.

Summary

Foot pad dermatitis (FPD) is a very common disease affecting poultry and is mostly caused by bad litter condition. This study aimed to test the effects of poultry diets with normal levels of electrolytes compared with a surplus level of electrolytes with and without using floor heating. Eighty 2-week-old ♀ turkey poultts aged were reared over 3 weeks on wood shavings,
divided randomly into 4 groups. Two groups were fed normal levels of electrolytes (1.60 g Na; 7.80 g K/kg diet), and the other two groups surplus levels of electrolytes (3.10; 15.3 g/kg diet). In each dietary treatment, half of the birds were exposed to floor heating. Half of the birds in each group were exposed for 4 h/d to wet litter (35 % water) in adjacent separate boxes. External assessment of foot pads was done weekly. High dietary electrolytes increased the severity of FPD significantly (3.65 ± 1.03). Floor heating is likely to be highly effective in significantly reducing the severity of FPD (2.36 ± 0.588). Despite forced water intake the litter became drier when floor heating was in use. Combining low Na and K levels with a floor heating system reduced the scores of FPD by about 60 %, compared with high electrolyte levels without floor heating. Therefore, both dietary electrolyte levels and floor heating markedly affected FPD via litter moisture.

References


Greene, J. A.; McCracken, R. M.; Evans, R. T., 1985: A contact dermatitis of broilers-clinical and pathological findings. Avian Pathology 14, 23–38.

Grosse Liesner, B. B., 2007: Comparative investigations on the performance and the appearance (incidence and manner) of primarily non-infectious health problems in male fattening turkeys of five different strains. Doctorate thesis, University of Veterinary Medicine, Hannover, Germany.


Oviedo-Rondón, E.O.; Murakami, A. E.; Furlan, A. C.; Moreira, I.; Macari, M., 2001: Sodium and chloride requirements of young broiler chickens fed corn-soybean diets (one to twenty-one days of age). Poultry Science 80, 592-598.


Suttle, N. F., 2010: Mineral Nutrition of Livestock, 4th edn. CABI publishing, USA.


Table 1: Experimental design for treatments without and with floor heating

<table>
<thead>
<tr>
<th>group</th>
<th>sub-group</th>
<th>birds (n)*</th>
<th>dietary electrolytes</th>
<th>floor heating **</th>
<th>dry/wet litter</th>
<th>exposure time (h/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>G1.1</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>24/0</td>
</tr>
<tr>
<td></td>
<td>G1.2</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>20/4***</td>
</tr>
<tr>
<td>G2</td>
<td>G2.1</td>
<td>10</td>
<td>normal</td>
<td></td>
<td></td>
<td>24/0</td>
</tr>
<tr>
<td></td>
<td>G2.2</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>20/4***</td>
</tr>
<tr>
<td>G3</td>
<td>G3.1</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>24/0</td>
</tr>
<tr>
<td></td>
<td>G3.2</td>
<td>10</td>
<td>high</td>
<td></td>
<td></td>
<td>20/4***</td>
</tr>
<tr>
<td>G4</td>
<td>G4.1</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>24/0</td>
</tr>
<tr>
<td></td>
<td>G4.2</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>20/4***</td>
</tr>
</tbody>
</table>

ten birds were killed for histopathological analysis at d 14, before the residual eighty birds were divided into the different groups
** temperature at litter surface was about 35 °C
*** half of birds were exposed to wet litter (35 % moisture) for 4 h/d
Table 2: Composition of the experimental diets (%) fed to turkeys during experimental period (d 14 –d 35)

<table>
<thead>
<tr>
<th>ingredients</th>
<th>normal dietary electrolytes</th>
<th>high dietary electrolytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>wheat grain</td>
<td>25.6</td>
<td>26.0</td>
</tr>
<tr>
<td>soybean meal (toasted)</td>
<td>27.6</td>
<td>28.5</td>
</tr>
<tr>
<td>yellow corn</td>
<td>22.4</td>
<td>21.0</td>
</tr>
<tr>
<td>corn gluten</td>
<td>14.5</td>
<td>14.0</td>
</tr>
<tr>
<td>fish meal (60-65 %)</td>
<td>2.00</td>
<td>1.80</td>
</tr>
<tr>
<td>fat (soybean oil)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>CaCO₃</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>NaH₂PO₄</td>
<td>0.400</td>
<td>0.400</td>
</tr>
<tr>
<td>NaCl</td>
<td>0.100</td>
<td>0.500</td>
</tr>
<tr>
<td>KHCO₃</td>
<td>0.100</td>
<td>2.00</td>
</tr>
<tr>
<td>CaHPO₄</td>
<td>3.00</td>
<td>3.00</td>
</tr>
<tr>
<td>DL-methionine</td>
<td>0.140</td>
<td>0.140</td>
</tr>
<tr>
<td>L-lysine</td>
<td>0.500</td>
<td>0.500</td>
</tr>
<tr>
<td>vitamin mixture*</td>
<td>0.040</td>
<td>0.040</td>
</tr>
<tr>
<td>vitamin E 50 %</td>
<td>0.020</td>
<td>0.020</td>
</tr>
<tr>
<td>trace elements mixture**</td>
<td>0.090</td>
<td>0.090</td>
</tr>
<tr>
<td>bolus alba</td>
<td>1.40</td>
<td>0.000</td>
</tr>
</tbody>
</table>

*vitamin mixture supplies the following per kilogram of diet: vitamin A, 13000 IU; vitamin D₃, 4000 IU; 25-hydroxycholecalciferol, 0.0250 mg; vitamin E, 60 mg; vitamin K₃, 5 mg; vitamin B₁, 4 mg. vitamin B₂, 10 mg; vitamin B₆, 7 mg. vitamin B₁₂, 0.035 mg; folic acid, 4 mg

**trace elements supply the following per kilogram of diet: copper, 13.36 mg; iron, 80 mg; zinc, 80 mg; manganese, 96 mg; iodine, 2 mg; selenium, 0.36 mg; cobalt, 0.04 mg
Table 3: Chemical analysis of experimental diet (g/kg as fed)

<table>
<thead>
<tr>
<th>Chemical composition (g/kg)</th>
<th>dietary electrolytes</th>
<th>macrominerals (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>normal</td>
<td>high</td>
</tr>
<tr>
<td>DM</td>
<td>904</td>
<td>906</td>
</tr>
<tr>
<td>crude ash</td>
<td>80.2</td>
<td>84.7</td>
</tr>
<tr>
<td>crude protein</td>
<td>282</td>
<td>280</td>
</tr>
<tr>
<td>crude fat</td>
<td>44.2</td>
<td>43.9</td>
</tr>
<tr>
<td>crude fibre</td>
<td>27.2</td>
<td>29.7</td>
</tr>
<tr>
<td>NfE</td>
<td>470</td>
<td>468</td>
</tr>
<tr>
<td>starch</td>
<td>303</td>
<td>310</td>
</tr>
<tr>
<td>sugar</td>
<td>42.8</td>
<td>44.5</td>
</tr>
<tr>
<td>ME' (MJ/kg)</td>
<td>11.5</td>
<td>11.6</td>
</tr>
<tr>
<td>amino acids (g/kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>threonine</td>
<td>9.10</td>
<td>9.11</td>
</tr>
<tr>
<td>arginine</td>
<td>15.0</td>
<td>14.6</td>
</tr>
<tr>
<td>cystine</td>
<td>4.73</td>
<td>4.57</td>
</tr>
<tr>
<td>methionine</td>
<td>6.50</td>
<td>5.44</td>
</tr>
<tr>
<td>lysine</td>
<td>15.8</td>
<td>16.1</td>
</tr>
<tr>
<td>Ca</td>
<td>15.6</td>
<td>15.5</td>
</tr>
<tr>
<td>P</td>
<td>10.6</td>
<td>10.7</td>
</tr>
<tr>
<td>Mg</td>
<td>1.95</td>
<td>1.88</td>
</tr>
<tr>
<td>Na</td>
<td>1.60</td>
<td>3.10</td>
</tr>
<tr>
<td>Cl</td>
<td>1.50</td>
<td>3.20</td>
</tr>
<tr>
<td>K</td>
<td>7.80</td>
<td>15.3</td>
</tr>
<tr>
<td>S</td>
<td>3.88</td>
<td>4.30</td>
</tr>
<tr>
<td>Cu</td>
<td>30.8</td>
<td>25.4</td>
</tr>
<tr>
<td>Zn</td>
<td>129</td>
<td>109</td>
</tr>
<tr>
<td>Fe</td>
<td>292</td>
<td>282</td>
</tr>
<tr>
<td>Mn</td>
<td>110</td>
<td>100</td>
</tr>
<tr>
<td>Se</td>
<td>0.550</td>
<td>0.470</td>
</tr>
<tr>
<td>dietary electrolyte balance (mEq/kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lysine</td>
<td>227</td>
<td>436</td>
</tr>
</tbody>
</table>

ME' calculated by using the official formula for complete diets: ME' (MJ/kg) = 0.01551 crude protein + 0.03431 crude fat + 0.01669 starch + 0.01301 sugar (nutrients in g/kg diet; FMVO, 2007)

**dietary electrolyte balance (DEB) was calculated by using the factors reported by Hooge (1995)**
Table 4: Parameters of feed and water intakes as well as young turkeys’ performance during experiment

<table>
<thead>
<tr>
<th></th>
<th>dietary electrolytes</th>
<th>floor heating</th>
<th>feed intake (g/bird/day)</th>
<th>water intake (ml/bird/day)</th>
<th>water intake ratio</th>
<th>weight gain* (g/bird/day)</th>
<th>FCR (kg feed/kg gain)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 1</td>
<td>normal</td>
<td>-</td>
<td>104</td>
<td>242</td>
<td>2.33</td>
<td>69.4 ± 5.60</td>
<td>1.49</td>
</tr>
<tr>
<td>G 2</td>
<td>normal</td>
<td>+</td>
<td>94.5</td>
<td>275</td>
<td>2.91</td>
<td>65.1 ± 6.60</td>
<td>1.45</td>
</tr>
<tr>
<td>G 3</td>
<td>high</td>
<td>-</td>
<td>101</td>
<td>287</td>
<td>2.84</td>
<td>65.6 ± 7.50</td>
<td>1.54</td>
</tr>
<tr>
<td>G 4</td>
<td>high</td>
<td>+</td>
<td>92.5</td>
<td>335</td>
<td>3.62</td>
<td>64.7 ± 7.90</td>
<td>1.43</td>
</tr>
</tbody>
</table>

individual measurements

Table 5: Comparison of young turkeys’ body weights (g) at different times

<table>
<thead>
<tr>
<th>sub-group</th>
<th>dietary electrolytes</th>
<th>floor heating</th>
<th>exposure to dry/wet litter (h/d)</th>
<th>duration of treatments</th>
<th>day (14) (n=10)</th>
<th>day (21) (n=10)</th>
<th>day (28) (n=10)</th>
<th>day (35) (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1.1</td>
<td>normal</td>
<td>-</td>
<td>24/0</td>
<td></td>
<td>367 ±24.7</td>
<td>732 ±53.8</td>
<td>1213 ±82.6</td>
<td>1812 ±108</td>
</tr>
<tr>
<td>G1.2</td>
<td>normal</td>
<td>+</td>
<td>20/4</td>
<td></td>
<td>388 ±27.1</td>
<td>761 ±62.5</td>
<td>1254 ±110</td>
<td>1857 ±167</td>
</tr>
<tr>
<td>G2.1</td>
<td>normal</td>
<td>+</td>
<td>24/0</td>
<td></td>
<td>342 ±44.0</td>
<td>693 ±63.8</td>
<td>1155 ±86.0</td>
<td>1708 ±111</td>
</tr>
<tr>
<td>G2.2</td>
<td>normal</td>
<td>+</td>
<td>20/4</td>
<td></td>
<td>369 ±42.9</td>
<td>729 ±87.3</td>
<td>1198 ±144</td>
<td>1738 ±200</td>
</tr>
<tr>
<td>G3.1</td>
<td>high</td>
<td>-</td>
<td>24/0</td>
<td></td>
<td>327 ±47.0</td>
<td>679 ±90.0</td>
<td>1171 ±152</td>
<td>1693 ±216</td>
</tr>
<tr>
<td>G3.2</td>
<td>high</td>
<td>-</td>
<td>20/4</td>
<td></td>
<td>363 ±48.0</td>
<td>728 ±86.2</td>
<td>1215 ±130</td>
<td>1751 ±167</td>
</tr>
<tr>
<td>G4.1</td>
<td>high</td>
<td>+</td>
<td>24/0</td>
<td></td>
<td>339 ±34.4</td>
<td>687 ±71.0</td>
<td>1139 ±133</td>
<td>1701 ±179</td>
</tr>
<tr>
<td>G4.2</td>
<td>high</td>
<td>+</td>
<td>20/4</td>
<td></td>
<td>347 ±59.6</td>
<td>703 ±93.1</td>
<td>1155 ±170</td>
<td>1701 ±227</td>
</tr>
</tbody>
</table>

*AB means in the same column with different superscripts are significantly different (p < 0.05)
*temperature at litter surface was about 35 °C
**birds were additionally exposed to litter with moisture content of about 35 %
Table 6: DM contents (%), crude ash, Na, K and Cl concentrations (g/kg DM) in excreta of turkeys fed normal/high dietary electrolytes during experimental period

<table>
<thead>
<tr>
<th>group</th>
<th>dietary electrolytes</th>
<th>floor heating</th>
<th>DM</th>
<th>crude ash</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 1</td>
<td>normal</td>
<td>-</td>
<td>22.3 ± 2.59</td>
<td>172 ± 15.7</td>
<td>1.77 ± 0.670</td>
<td>19.9 ± 4.20</td>
<td>4.94 ± 2.35</td>
</tr>
<tr>
<td>G 2</td>
<td>normal</td>
<td>+</td>
<td>22.0 ± 2.73</td>
<td>174 ± 16.8</td>
<td>1.41 ± 0.440</td>
<td>20.7 ± 4.79</td>
<td>4.63 ± 2.57</td>
</tr>
<tr>
<td>G 3</td>
<td>high</td>
<td>-</td>
<td>17.6 ± 5.55</td>
<td>176 ± 16.9</td>
<td>4.81 ± 2.91</td>
<td>35.0 ± 13.9</td>
<td>9.22 ± 4.93</td>
</tr>
<tr>
<td>G 4</td>
<td>high</td>
<td>+</td>
<td>17.2 ± 5.87</td>
<td>175 ± 15.9</td>
<td>4.31 ± 2.44</td>
<td>34.4 ± 13.9</td>
<td>8.80 ± 4.84</td>
</tr>
</tbody>
</table>

Table 7: External and histopathological foot pad scores of young turkeys (results of two factors variance analyses, mean ± SD) during experimental period

<table>
<thead>
<tr>
<th>factor*</th>
<th>treatment</th>
<th>group</th>
<th>n</th>
<th>FPD scores at day (duration of treatments)</th>
<th>external</th>
<th>histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>21 (7)</td>
<td>28 (14)</td>
<td>35 (21)</td>
</tr>
<tr>
<td>dietary</td>
<td>normal</td>
<td>G1/G2</td>
<td>40</td>
<td>1.31 ±0.613</td>
<td>1.71 ±0.534</td>
<td>2.48 ±0.782</td>
</tr>
<tr>
<td>electrolytes</td>
<td>high</td>
<td>G3/G4</td>
<td>40</td>
<td>1.70 ±0.882</td>
<td>2.68 ±1.14</td>
<td>3.65 ±1.03</td>
</tr>
<tr>
<td>floor heating**</td>
<td></td>
<td>-</td>
<td>G1/G3</td>
<td>40</td>
<td>1.91 ±0.807</td>
<td>2.81 ±1.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+</td>
<td>G2/G4</td>
<td>40</td>
<td>1.10 ±0.483</td>
<td>1.57 ±0.460</td>
</tr>
</tbody>
</table>

*AB means in the same column within each factor with different superscripts are significantly different (p < 0.05)
* third factor (exposure to wet litter for 4 h/d) was neglected, as it resulted in no significant differences compared with birds housed for 24 h/d
**temperature at litter surface was about 35 °C
Table 8: Development of external and histopathological foot pad scores of young turkeys throughout experiment (mean ± SD)

<table>
<thead>
<tr>
<th>subgroup</th>
<th>dietary electrolytes</th>
<th>floor heating*</th>
<th>exposure to dry/wet litter (h/d)</th>
<th>FPD scores at day (duration of treatments)</th>
<th>histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>external (n=10)</td>
<td></td>
</tr>
<tr>
<td>G1.1</td>
<td>normal</td>
<td>-</td>
<td>24/0</td>
<td>1.50 ±0.577 AD ±0.316</td>
<td>3.15 ±0.412</td>
</tr>
<tr>
<td>G1.2</td>
<td>normal</td>
<td>+</td>
<td>20/4**</td>
<td>0.80 ±0.422 AB ±0.349</td>
<td>2.10 ±0.218</td>
</tr>
<tr>
<td>G2.1</td>
<td>high</td>
<td>-</td>
<td>24/0</td>
<td>2.30 ±0.483 AB ±0.316</td>
<td>4.70 ±0.537</td>
</tr>
<tr>
<td>G2.2</td>
<td>high</td>
<td>+</td>
<td>20/4**</td>
<td>1.30 ±0.483 AB ±0.349</td>
<td>4.40 ±0.516</td>
</tr>
<tr>
<td>G3.1</td>
<td>high</td>
<td>-</td>
<td>24/0</td>
<td>1.10 ±0.19 ±0.516 AB ±0.459</td>
<td>2.90 ±0.394</td>
</tr>
<tr>
<td>G3.2</td>
<td>high</td>
<td>+</td>
<td>20/4**</td>
<td>1.75 ±0.425 AB ±0.349</td>
<td>2.90 ±0.394</td>
</tr>
</tbody>
</table>

\* indicates differences among treatment groups (p < 0.05)

\** indicates additional exposure to litter with moisture content of about 35%
6. Chapter 4

Foot pad dermatitis and experimentally induced coccidiosis in young turkeys fed a diet without anticoccidia
6. Chapter 4

Health and Disease

FOOT PAD DERMATITIS AND COCCIDIOSIS IN TURKEYS

Foot pad dermatitis and experimentally induced coccidiosis in young turkeys fed a diet without anticoccidia

A. Abd El-Wahab*, C. F. Visscher*, S. Wolken§, J-M. Reperant†,
A. Beineke#, M. Beyerbach‡ and J. Kamphues*¹

*Institute of Animal Nutrition, University of Veterinary Medicine Hannover, Foundation, Germany
†Institute of Parasitology, University of Veterinary Medicine Hannover, Foundation, Germany
‡Institute of Biometry and Information Processing, University of Veterinary Medicine Hannover, Germany
§French Agency for Food, Environmental and Occupational Health & Safety, ANSES, France
#
Institute of Pathology, University of Veterinary Medicine Hannover, Foundation, Germany

Submitted to Poultry Science Journal, at 04.09.2011, undergoing review

¹Corresponding author: josef.kamphues@tiho-hannover.de

Prof. Dr. J. Kamphues, Institut für Tierernährung, Stiftung Tierärztliche Hochschule Hannover, Bischofsholer Damm 15, D-30173 Hannover, Germany
Tel./Fax:+49 511 8567301 Fax: +49 511 8567698
ABSTRACT

Foot pad dermatitis (FPD) is a widespread challenge to turkey production. This study aimed at evaluating the effects of using floor heating, exposure to litter with critical moisture content (35 %) under experimental infection with *E. adenoeides* on the severity of FPD in turkeys. Two trials were done, in each trial four groups of 2-week-old ♀ turkeys being reared during 4 weeks. At the experiment start (d 14) each bird had normal foot pads. All birds were fed ad libitum on identical pelleted diets without any anticoccidial additive. The first 2 groups were kept on dry wood shavings with and without floor heating; the other 2 groups were housed on wet wood shavings of 35 % moisture with and without floor heating. Two birds in each of the 4 groups were experimentally infected with *E. adenoeides* via crop intubation (~50,000 oocysts/bird). Foot pads were assessed weekly for external scoring and at d 42 for histopathological scoring. The number of oocysts eliminated via excreta was determined.

In both trials using floor heating resulted in significantly decreased FPD scores (2.06 ± 0.735; 1.47 ± 0.734) compared with groups housed without floor heating (3.88 ± 0.812; 2.73 ± 1.25). Birds exposed continuously to wet litter (35 % moisture) showed significantly increased FPD scores (3.41 ± 1.23; 2.69 ± 1.34) compared with the group not exposed to wet litter (2.53 ± 1.00; 1.53 ± 0.683). The coccidial infection in both trials resulted in markedly lowered DM contents of excreta (14.8 and 15.1 %) and litter (58.0 and 57.6 %) in the groups exposed to wet litter without using floor heating. In both trials using floor heating resulted in the highest mean DM content of litter (85.1 and 85.0 %) and highest body weights (2693 and 2559 g). The results suggest that induced diarrhea caused by coccidial infection led to a bad litter quality and hence increased the severity of FPD which can be overcome by using floor heating.

**Keywords**: Foot pad dermatitis, litter quality, floor heating, coccidiosis

INTRODUCTION

A low prevalence and severity of foot pad dermatitis (FPD) are highly desirable regarding the health of birds and product quality. FPD is a type of contact dermatitis affecting the plantar region of the feet, with lesions surrounded by a reddening of the foot pads as a first symptom, followed by discoloration and hyperkeratosis often in combination with erosions and necrosis of the epidermis, with deep ulcers occurring in severe cases (Greene et al., 1985).
Furthermore, the lesions can be a gateway for bacteria which may spread hematogenously and impair product quality (Schulze, 1996). FPD can achieve a prevalence of approximately 20 % for severe lesions and 78 % for mild lesions in fattening turkeys (Berg, 1998). At the end of the turkeys' fattening period, this disease could reach a prevalence of 91-100 % (Hafez et al., 2004). About 97.2 % of turkeys showed FPD lesions with no marked effects on the BW of five different strains of male turkeys at the end of the fattening period (Grosse Liesner, 2007). The prevalence of FPD per farm is gaining recognition as a well-being indicator (Berg, 1998; Bradshaw et al., 2002; Martrenchar et al., 2002).

Different authors found positive correlations between litter quality (particularly moisture) and the incidence of FPD (Martland, 1984; Ekstrand et al., 1997; Buda et al., 2002; Youssef et al., 2010). Several factors, which include but are not limited to stocking density, ventilation, and drinker design, can affect litter moisture. One common aspect in most previous studies is that litter moisture is a significant factor in the onset of FPD (Martland, 1985; Shepherd and Fairchild, 2010). Similar to findings in broilers, turkeys raised on wet litter have higher rates of FPD than those raised on dry litter (Martland, 1984). Drying out the litter and moving birds from wet litter to dry litter was observed to reverse the severity of FPD (Greene et al., 1985; Martland, 1985).

Standing on wet litter brings the feet in constant contact with moisture and has been suggested to cause the foot pad to soften and become more prone to damage, predisposing the birds to develop FPD (Jensen et al., 1970). Housing turkey poultls in high moisture (73 %) for 8 h/d led to an imposingly higher prevalence of FPD (Youssef et al., 2010). However, the first marked increase of FPD lesion was observed after exposure for only 4 h/d to 35 % moisture which was nominated as “critical moisture content” (Abd El-Wahab et al., 2012). Previous studies stated that dietary constituents (soybean meal, macrominerals, electrolytes) have a great impact on the litter quality and hence on the severity of FPD (Youssef et al., 2010; Abd El-Wahab et al., 2011b). Finally, it has to be emphasised that also disease affecting excreta quality, such as coccidiosis might be a neglected factor related to FPD.

Coccidiosis is one of the most important and common diseases affecting poultry, resulting in great economic losses all over the world (Chapman, 2008). One of the major factors causing wet litter is diarrhea. This can be a result of different infections in the intestinal tract, for example protozoal Eimeria spp. infection (Mayne, 2005). In turkeys seven species of Eimeria
have been reported. However, *E. adenoeides* is considered the most pathogenic, infecting the caecum of birds (Hafez, 2008). Clinical signs of coccidiosis in turkeys are not pathognomonic and include loss of appetite, listlessness, huddling, diarrhea, drooping wings and ruffled feathers (Reid, 1972, Chapman, 2008). Challenge recommended doses for *E. adenoeides* are 25,000-100,000 sporulated oocysts per bird (Holdsworth et al., 2004).

The emergence of resistance to coccidiostats, consumer demand for using fewer feed additives, and European Union Regulations (withdrawal of antibiotic feed additives as a precautionary measure) might restrict the use of coccidiostats (European Commission Regulations, 1997). If this happens, alternative strategies should probably be introduced to minimize the adverse effects of coccidia on animal health and production.

Testing the diet composition, should not let us neglect the potential role of coccidiosis or – from the feed production point of view – the correct use/adding of an effective coccidiostat. In the diet composition with high efficient coccidiostat, the role of infection could be neglected. However, due to misdosing and/or inefficient anticoccidial additive in the diet, the excreta and bedding material will be markedly influenced by the coccidial infection. The ingredients used and their commercial sources as well as the form of the feed (pellet, crumble or mash) should reflect those to be used in practice. For example, the milling and pelleting process may affect drug stability, growth rate and feed utilization of broilers (Engberg et al., 2002).

Regarding floor heating, it has been noted that using floor heating has a significant effect on FPD (Berg and Algers, 2004; Abd El-Wahab et al., 2011a,b). The aim of the present study was therefore to simulate a condition which could occur in the field when feeding a diet without any anticoccidial additive and to consider the effects on litter quality and FPD in young turkeys challenged by experimental infection with *E. adenoeides*. Furthermore, the study set out to test the effects of using floor heating despite the presence of two additive factors: exposure to wet litter and coccidiosis. Finally, this study continued a series of previous experimental studies on the effects of diet, housing and management on FPD.

**MATERIALS AND METHODS**

The experiments were performed in accordance with the German Animal Welfare Legislation (33.12-42502-04-09/1776).

**Housing**
One hundred and eighty female turkey poults (BUT-Big 6), one-d old, were allotted to two trials. Birds were housed in a floor pen prepared with wood shavings, kept dry and clean before the experiment by removing the upper layers of the litter daily and replacing them with fresh dry litter. During the first 3 d additional feed was offered (about 400 g/d) on paper to accustom the birds to the diet. All turkeys were fed ad libitum with a commercial pelleted diet (containing lasalocid-A-sodium, 110 mg/kg diet) as fed for the first 7 d to keep them free from accidental infection. Afterwards, the birds were shifted to the experimental diet without anticoccidial additive (Table 1) until the end of the rearing period. Each pen was equipped with one infra-red lamp, to achieve a temperature of about 34-36 °C at the outset of brooding of the one-d-old birds. The temperature was lowered by about 1 °C every 2 d. The photoperiod from d 4 onwards was 16 h of light and 8 h of darkness.

**Experimental Design**

Two experimental trials were performed. In each trial, ten birds were chosen randomly for necropsy at the beginning of the experimental period (d 14) for foot pad histopathological assessment. The remaining birds, 80 in total in each trial, were individually identified and then divided into 4 equal groups, each housed in a floor pen (1.50 m x 1.32 m). The first 2 groups were kept on “dry” wood shavings with and without floor heating; the other 2 groups were housed on wood shavings with a moisture content of 35 %, with and without floor heating. The electrical floor heating system (QUATTTEC GmbH, Jessen, Germany) supplied with adjuster to control the temperature was used. In each group, the depth of the litter material was approximately 4 cm (5 kg/m$^2$ of wood shavings). The wet litter was experimentally maintained by adding water as required (every 2 d). The added amount of water was estimated in pre-experimental studies, and then modified during this experiment by measure the DM content of litter. The mean temperature during the experimental period at litter surface was about 35 °C in the groups using floor heating vs. 25 °C in the groups without floor heating. Furthermore, in both trials the relative humidity in each group was measured by using Data Logger (ebro, EBI 20, ebro Electronic GmbH & Co.KG, Ingolstadt, Germany) every hour daily all over the experimental period.

Coccidial infections were established by means of seeder birds. Thus, only two birds in each group (nominated as primary infected birds) were experimentally infected with a pure isolate containing *E. adenoeides* via crop intubation with 1 ml of ~50,000 sporulated oocysts/bird.
The number of oocysts produced in the excreta of primary infected birds was determined after each 4 d post inoculation (PI) until the end of the experiment. In each group, the two primary infected birds were taken out of the pen during the time of collection pooled excreta of the other ones (nominated as secondary infected birds) at d 8, 12, 16, 20 and 24 PI, then those two primary infected birds returned back to the pen. A clean polyethylene sheet covering the litter was used for collecting pooled excreta ~100 g/group for oocyst counting according to Hodgson (1970). Briefly, a sample of 4 g excreta was diluted in saturated NaCl to 60 ml volume. After homogenization and filtration, the sample was loaded into a McMaster counting chamber and the oocysts were allowed to float for 5 min before enumeration. The number of oocysts within three ruled areas, multiplied by 33.33 represents the number of excreta per g.

**Measurements**

Litter samples for measuring moisture and pH were collected weekly from 5 sites (4 peripheral samples and 1 central one) in each pen. At each area a sample (~50 g) over the whole bedding height was punched out using a tin with a diameter of 6.5 cm and then, mixed as one sample. A sub-sample of about 100 g was taken to measure moisture content. Samples were oven-dried at 103 °C for the time needed to reach constant weight. Litter pH was measured by making a suspension (1 part material: 9 parts water) then measured using a pH meter. Dry matter content of excreta was estimated at the day of collecting pooled excreta samples. Part of the fresh pooled excreta was microbiological examined for detection of clostridia, salmonella and campylobacter micro-organisms.

It has to be emphasized that each bird in all groups was marked individually throughout the experimental period. Individual body weight was recorded weekly at the day of scoring. Feed and water intakes were measured daily at group level. Feed conversion ratio (FCR) was estimated and corrected for mortalities on the basis of feed consumed (data from groups) and weight gain of the birds (individual data) throughout the experimental period.

**FPD Scoring Criteria**

External assessment of foot pads was made at d 14, 21, 28, 35 and 42. At d 42 all birds were sacrificed for microscopic evaluation of foot pads. During the external examination, if the feet were dirty, they were gently washed with a wet cloth and dried before scoring; only the central plantar was scored, signs of foot pad lesions were recorded on a 7-point scale.
(0 = normal skin; 7 = over half of the foot pad is covered with necrotic scales) according to Mayne et al. (2007).

Due to small cellular changes occurring within the foot pad before any evidence of a lesion is present on the external foot pad surface, both foot pads of all sacrificed birds were assessed histologically by removing the skin from the foot pad and fixing it in 10 % buffered neutral formalin in a micro-cassette. One cross section of skin was evaluated from the center of each foot pad. Sections were prepared and processed using standard protocols for tissue processing. Paraffin-embedded tissues were cut into 2-3 µm sections using a microtome and stained with hematoxylin and eosin. Sections were examined under a light microscope and categorized using the histopathological scoring system on a 7-point scale (0 = normal epidermis; 1 = hyperkeratosis; 2 = epidermal acanthosis; 3 = vacuoles in dermis and epidermis; 4 = presence of heterophils, macrophages and lymphocytes in dermis; 5 = increased density of heterophils, macrophages and lymphocytes; 6 = ulcer of the epidermis with only one lesion; 7 = more than one rupture or “ulcer” of the epidermis) according to Mayne et al. (2007).

**Statistical Analyses**

The foot pad scores were evaluated by using the mean of both feet. The data from the external and histopathological foot pad scoring, body weight and primary oocyst counting (Log 10) were analyzed separately for each sampling point using the GLM procedure of the SAS Institute Inc. (2005) software. For body weight, external and histopathological FPD scores Tukey test for pair-wise multiple means comparison of the GLM procedure of SAS Institute Inc. (2005) software was used. All statements of statistical significance are based upon p < 0.05. To test potential effects of time (during the experimental diverse time points) with normally distributed differences the t-test for paired observations was used. Otherwise, the Wilcoxon signed-rank test within procedure UNIVARIATE was used. It has to be emphasized that the F-factor was significant between the both trials; hence it is not allowed to pool the data of both trials for statistical analysis.

**RESULTS**

A typical ventilation system in this study was not used (no need for ventilation) but might be under field conditions there is a most important factor “ventilation”. Thus, might be air movement will target all the tested factors that already done. Also, the incoming air in the
farms plays a role in the FPD prevalence. For example, if the incoming air is moist so it will worsen the condition of the litter and consequently increase the severity of FPD. But if the incoming air is warm, it could lead to dryness of the litter surface and hence decrease the severity of FPD. In this study for both trials, the mean relative humidity all over the experimental period was about 47.4 % ± 8.62 and 47.1 % ± 8.07 for groups housed without using floor heating vs. 46.7 % ± 8.12 and 46.3 % ± 8.10 for groups used floor heating. Moreover, two birds died (from groups exposed daily to wet litter with and without floor heating) during the experimental period (4 wk) in both trials. No growth promoting substances were used in any group, and no birds were otherwise treated throughout the whole experimental period. Also, no birds shed oocysts prior to inoculation. Also, it must be stressed that because of significant effect between treatment and experiment interaction (F-factor), the results of the two present trials can not be pooled from a statistical point of view.

**Animal Performance**

Table 2 shows no significant differences were observed between the experimental groups at d 42 in the first trial. However in second trial, daily exposure to wet litter without using floor heating led to a significantly decrease final body weight (2313 g ± 292) compared with other experimental groups except in the case of the group exposed to wet litter using floor heating. In both trials, using floor heating resulted in slightly higher water: feed ratio (2.59 ± 0.035; 3.02 ± 0.028) compared with those birds housed without floor heating (2.49 ± 0.028; 2.75 ± 0.007). Moreover, using floor heating in the absence of wet litter was accompanied by a favourable FCR (1.54 and 1.46). Also, in both trials the groups housed without daily exposure to wet litter either with or without floor heating showed the highest weight gain (80.9 and 80.2 g/bird/day, respectively for first trial; 78.8 and 78.8 for second trial) compared with the other experimental treatments.

**Oocyst Counting**

first of all, it should be emphasized that inoculation of oocysts was performed successfully. Moreover, excreta samples were proved microbiologically for absence of clostridia, salmonella and campylobacter micro-organisms for all groups in both trials. The two birds infected with *E. adenoeides* in each pen showed depression, weakness and dullness. In addition, there were traces of blood in the watery excreta of the turkey poults. At necropsy, there were many hemorrhages on the mucosal surface of the cecum with pronounced
thickening. However, using floor heating without exposure to wet litter in both trials reduced the oocyst numbers in the excreta (1.52/1.21; 0/1.82 and 1.69/0 at d 16, 20, 24 PI, respectively). However, daily exposure to wet litter either with or without floor heating resulted in higher oocyst numbers in the excreta of both trials (3.34/3.11; 3.42/3.77 at d 20 PI and 3.18/3.76; 3.08/2.73 at d 24 PI, respectively).

Regarding the secondary infected birds, it was noted that the coccidial infection was established successfully in the secondary infected birds by natural means (Table 4). It was observed that using floor heating without exposure to wet litter led to a marked decrease in oocyst numbers in the excreta compared with groups not using floor heating throughout experimental period. Nevertheless, in both trials using floor heating with exposure to wet litter resulted in a higher oocyst count in the excreta (3.72/3.92) at d 24 PI compared with the other groups.

**Litter Quality and Coccidiosis**

The mean DM content of litter in both trials during the entire experimental period were the highest for groups used floor heating without exposed to wet litter (85.1 % ± 5.55 and 85.0 % ± 5.17) compared with the other treatments. However, groups exposed to wet litter without using floor heating in both trials resulted in the lowest mean DM content of litter (58.0 ± 7.69 and 57.6 ± 7.89). Regarding excreta DM content, it was noted that the absence of floor heating with daily exposure to wet litter resulted in lowest mean DM content of excreta in both trials (14.8 % ± 2.03 and 15.1 % ± 2.17) compared with the other experimental groups. By using floor heating in both trials the mean DM content of excreta was the highest (17.2 % ± 1.06 and 17.3 % ± 0.49) compared with the other experimental groups. Furthermore, Figure 1 provides more details on the effects of the severity of coccidial infection on mean DM content of excreta, the oocyst counting “Log 10/g excreta” being able to be classified into 3 categories (numbers 0-2 = low; numbers 2-3.5 = medium and numbers 3.5-5 = high). Accordingly, it was observed that in both trials the low oocyst numbers in excreta led to significantly increased DM content of excreta (17.4 % ± 1.11 and 17.5 % ± 0.568) vs. (14.5 % ± 0.900 and 14.6 % ± 1.10) for the high oocyst counting in excreta.

Additionally, in both trials daily exposure to wet litter without using floor heating was accompanied with the highest pH value (7.38 ± 1.10 and 7.57 ± 1.25) compared with the other
experimental groups. While, using floor heating without daily exposure to wet litter resulted in the lowest pH values in both trials (6.40 ± 0.250 vs. 6.38 ± 0.280) compared with the other experimental groups.

**Foot Pad Lesions**

At the beginning of the experiment (d 14) there was no evidence of external or histopathological FPD lesions. Table 5 shows that in both trials using floor heating resulted in significantly decreased external FPD scores (2.06 ± 0.735 and 1.47 ± 0.734) and significantly decreased histopathological FPD scores (2.06 ± 0.662 and 1.51 ± 0.493) in comparison to groups without floor heating (3.88 ± 0.812 and 2.73 ± 1.25 for external scores; 3.53 ± 1.07 and 2.24 ± 0.841 for histopathological scores). Furthermore, in both trials daily exposure to wet litter (35 % moisture) was accompanied by significantly increased external FPD scores (3.41 ± 1.23 and 2.69 ± 1.34) and increased histopathological FPD scores (3.18 ± 1.20 and 2.21 ± 0.827) compared with groups housed without continuously exposed to wet litter (2.53 ± 1.00 and 1.53 ± 0.683, for external scores; 2.41 ± 0.979 and 1.56 ± 0.579 for histopathological scores).

For providing greater details on lesion assessment, the severity of foot pad lesions could be classified into 3 categories (low scores = 0-3.5, medium scores = 4-5.5 and high scores = 6-7). Accordingly, on d 42 it was observed on the one hand that using floor heating in both trials showed 97.5 and 100 % low scores, while the absence of floor heating resulted in 30 and 66.6 % low; 70 and 33.3 % medium scores. On the other hand in both trials, daily exposure to wet litter led to 47.5 and 65.8 % low; 52.5 and 34.2 % medium scores vs. 80 and 100 % low; 20 and 0 % medium scores in the absence of exposure to wet litter.

Duration of treatment (time) resulted in significantly increased FPD scores each week in both trials and for all tested factors (Table 5).

At the end of the experimental period (d 42), using floor heating without exposure to wet litter in both trials resulted in significantly decreased histopathological FPD scores (1.70 ± 0.410 and 1.30 ± 0.299) compared with the other experimental groups despite coccidial infection (Table 6). In the groups exposed daily to wet litter for both trials, using floor heating showed significantly decreased external and histopathological FPD scores (2.37 ± 0.775 and 2.42 ± 0.674, respectively for first trial; 1.68 ± 0.820 and 1.73 ± 0.562, respectively for second trial) compared with the group not using floor heating in spite of induced diarrhea.
caused by coccidial infection (4.45 ± 0.483 and 3.95 ± 1.13, respectively for first trial; 3.71 ± 0.932 and 2.68 ± 0.785, respectively for second trial). Furthermore, in groups housed without floor heating for both trials the daily exposure to wet litter led to significantly increased external FPD scores (4.45 ± 0.483 and 3.71 ± 0.932) vs. (3.32 ± 0.674 and 1.80 ± 0.676), for the group not exposed to wet litter. Table 6 showed that weekly examination of foot pads (duration of treatment) was accompanied with increase severity of FPD scores significantly in both trials and for all experimental groups.

**DISCUSSION**

Infections with coccidia are often associated with severe economic losses, thus more attention should be given to improved housing conditions (Jordan, 1995). For poultry to be able to perform their growth rate potential to the fullest, they should be well looked after and kept in good environmental conditions including the litter quality which is affected by a number of dietary, management and housing measures (Abd El-Wahab et al., 2011b). In fact, the occurrence of FPD is now used as an audit criterion in welfare assessments of poultry production systems in Europe and the United States (Berg and Algers, 2004). Another point of interest was the influence of floor heating, causing drier litter or higher temperature, on the oocyst counts in excreta and also on coccidial lesions in the cecum, which will be discussed in a further publication (specially focused on the coccidial infection under the influence of floor heating).

**Coccidiosis and Litter Quality**

Judging by the results of the oocyst counts in excreta of both primary and secondary infected birds, the experimental infection was successful. One of the most important signs characterizing a coccidial infection is watery excreta (Hafez, 2008), which was reflected in the markedly reduced DM of excreta; especially in groups with higher oocyst counts in excreta. Thus, oocyst numbers in the excreta were closely correlated with the changes in DM content of excreta. Additionally, combination of floor heating and dry litter resulted in markedly reduced oocyst counts in primary and secondary infected birds.

Moisture is the key factor influencing litter quality and managing litter is a crucial step in promoting flock health and well-being. Using floor heating without daily exposure to wet litter resulted in drier litter (85.1 and 85.0 % DM in both trials) during the experimental
period despite coccidial infection and induced diarrhea. It was stated that wet litter was associated with a higher pH compared with dry litter (Lerner, 1996). Similarly, in this study daily exposure to wet litter in the absence of floor heating resulted in a higher litter pH value. On the contrary, using floor heating without daily exposure to wet litter produced the lowest litter pH value even with coccidial infection, which could be due to drier litter.

**Severity of FPD**

Litter moisture is considered to be an important factor predisposing to FPD (Jensen et al., 1970). Thus, FPD can be kept at a minimum with proper litter management. High prevalence and severity of FPD were correlated with high litter moisture (Hafez et al., 2005; Bilgili et al., 2009 and Shepherd and Fairchild, 2010). FPD lesions have been found to become more severe as litter moisture increases.

Although most of the literature suggests that litter moisture is a critical component in the development of contact dermatitis, other studies have found no significant correlation between litter moisture and the incidence and severity of FPD (Eichner et al., 2007). Coccidiosis plays a major role, predisposing the birds to FPD due to diarrhea and subsequent increased moisture in the litter. Thus, with increasing prevalence and severity of FPD on farms, intestinal infections, such as coccidiosis should not be neglected. Excreta quality was markedly influenced by the coccidial infection and consequently led to a decreased litter DM content and increased severity of FPD. Using floor heating for birds resulted in significantly decreased FPD scores compared with groups not using floor heating. Despite induced diarrhea due to coccidial infection, the litter became drier when floor heating was used. Therefore, floor heating is likely to be highly effective in reducing the development and severity of FPD. Abd El-Wahab et al. (2011a,b) observed that the significant effect of using floor heating on FPD scores could be due to the litter becoming dry as fresh litter or could be due to floor heating leading to warm foot pads causing vasodilatation of the blood vessels, increasing the blood flow to promote healing. The principle of the warming effect on blood flow in humans was stated by Nisha (2003). On the other hand, with the absence of floor heating the litter is quite cool and might lead to blood vessel constriction resulting in a cold-wet foot pad. The heat source in turkey houses hangs above the pens; so the upper surface of litter becomes warm but the colder, deeper litter eventually moves to the top (Abd El-Wahab et al., 2011a,b). Our findings agree with the studies of Berg and Algiers (2004) who found that
using floor heating had a significantly beneficial effect on FPD with a prevalence of 21.5 % ± 3.7 for floor heating groups vs. 45.0 % ± 7.1 for groups not using floor heating. Similarly, Abd El-Wahab et al. (2011a) stated that using floor heating led to significantly decrease FPD scores (0.950 ± 0.150) even with daily exposure to wet litter (35 % moisture) with a group not using floor heating (2.55 ± 0.830) in young turkeys at d 35.

Furthermore, in groups not using floor heating, daily exposure to wet litter resulted in significantly increase FPD scores. This could be explained by the fact that standing on wet litter brings the feet in constant contact with moisture and has been suggested to cause the foot pad to soften and become more prone to damage, predisposing the bird to developing FPD (Jensen et al., 1970). Also, in this study showed that the duration of treatment (time factor) plays a significant role in increasing the severity of foot lesions for all experimental treatments. It means that each week (may be less) in all experimental groups was enough to increase the severity of FPD scores significantly. Previous research (Abd El-Wahab et al., 2012) has shown that the first significant increase in FPD lesion was observed after exposure for only 4 h/d to “critical moisture content” (35 %) and the severity of FPD increased with increasing litter moisture. Daily exposure to wet litter (either wood shavings or lignocellulose) with 35 % moisture content resulted in significantly higher FPD scores, 2.55 ± 0.830 or 2.30 ± 0.880, respectively compared with groups housed only for 16 h/d on wet litter, 1.60 ± 0.450 or 1.55 ± 0.860, respectively (Abd El-Wahab et al., 2011a).

REFERENCES


Abd El-Wahab, A., C. F. Visscher, A. Beineke, M. Beyerbach, and J. Kamphues. 2012. Experimental studies on the effects of different litter moisture contents and exposure time to


Grosse Liesner, B. B. 2007. Comparative investigations on the performance and the appearance (incidence and manner) of primarily non-infectious health problems in male fattening turkeys of five different strains. Doctorate Dissertation, University of Veterinary Medicine, Hannover, Germany.


Schulze, K. 1996. Investigations on quality of litter and performance of broilers during fattening depending on stocking density. Doctorate Dissertation, University of Veterinary Medicine, Hannover, Germany.


TABLE 1. Composition (%) and chemical analysis (g/kg as fed) of the experimental diet fed to turkeys during experimental period (d 14 – d 42)

<table>
<thead>
<tr>
<th>ingredients (%)</th>
<th>chemical composition (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ingredients (%)</td>
</tr>
<tr>
<td>wheat</td>
<td>25.6</td>
</tr>
<tr>
<td>soybean meal (toasted)</td>
<td>35.8</td>
</tr>
<tr>
<td>yellow corn</td>
<td>19.5</td>
</tr>
<tr>
<td>DAN Pro A (^1)</td>
<td>6.75</td>
</tr>
<tr>
<td>fish meal</td>
<td>3.00</td>
</tr>
<tr>
<td>soybean oil</td>
<td>2.50</td>
</tr>
<tr>
<td>CaCO(_3)</td>
<td>1.00</td>
</tr>
<tr>
<td>Dicalciumphosphate</td>
<td>3.00</td>
</tr>
<tr>
<td>NaCl</td>
<td>0.100</td>
</tr>
<tr>
<td>NaH(_2)PO(_4) (\times) 2H(_2)O</td>
<td>0.400</td>
</tr>
<tr>
<td>KHCO(_3)</td>
<td>0.100</td>
</tr>
<tr>
<td>DL-methionine</td>
<td>0.190</td>
</tr>
<tr>
<td>L-lysine</td>
<td>0.500</td>
</tr>
<tr>
<td>vitamin mixture (^2)</td>
<td>0.050</td>
</tr>
<tr>
<td>vitamin E 50%</td>
<td>0.030</td>
</tr>
<tr>
<td>trace elements mixture (^3)</td>
<td>0.100</td>
</tr>
<tr>
<td>bolus alba</td>
<td>1.38</td>
</tr>
</tbody>
</table>

\(^1\)soybean protein concentrate (62.5 % crude protein)
\(^2\)vitamin mixture supplies the following per kilogram of diet: vitamin A, 13000 IU; vitamin D\(_3\), 4000 IU; 25-hydroxycholecalciferol, 0.0250 mg; vitamin E, 100 mg
\(^3\)trace elements supply the following per kilogram of diet: copper, 12 mg; iron, 75 mg; zinc, 75 mg; manganese, 90 mg; iodine, 1.8 mg; selenium, 0.3 mg; cobalt, 0.04 mg
\(^4\)ME calculated by using the official formula for complete diets in poultry: ME\(_n\) (MJ/kg) = 0.01551 crude protein + 0.03431 crude fat + 0.01669 starch + 0.01301 sugar (nutrients in g/kg diet; FMVO, 2007).
### TABLE 2. Comparison of young turkey’s body weight (g) at different times

<table>
<thead>
<tr>
<th>floor heating</th>
<th>exposure</th>
<th>day (duration of treatments)</th>
<th>trial 1</th>
<th>trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>to wet</td>
<td>14 (0) (n=20)</td>
<td>21 (7) (n=20)</td>
<td>28 (14) (n=20)</td>
</tr>
<tr>
<td>-</td>
<td>0</td>
<td>418 ±32.5</td>
<td>779 ±75.9</td>
<td>1322 ±103</td>
</tr>
<tr>
<td>-</td>
<td>24</td>
<td>412 ±30.3</td>
<td>798 ±55.7</td>
<td>1336 ±111</td>
</tr>
<tr>
<td>+</td>
<td>0</td>
<td>426 ±22.7</td>
<td>809 ±63.5</td>
<td>1371 ±83.2</td>
</tr>
<tr>
<td>+</td>
<td>24</td>
<td>412 ±34.4</td>
<td>798 ±65.0</td>
<td>1339 ±117</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+1) 24</td>
<td>+1) 24</td>
<td>+1) 24</td>
</tr>
</tbody>
</table>

*a,b* Means in the same column with different superscripts in each trial are significantly different (p < 0.05)

1) n=19 from d 21 old till end of the experimental period

### TABLE 3. Oocysts counting (Log 10/g excreta) of primary individual infected birds during experimental period for both trials

<table>
<thead>
<tr>
<th>floor heating</th>
<th>exposure</th>
<th>day (post inoculation)/primarily</th>
<th>4 (n=2)</th>
<th>6 (n=2)</th>
<th>8 (n=2)</th>
<th>12 (n=2)</th>
<th>16 (n=2)</th>
<th>20 (n=2)</th>
<th>24 (n=2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>to wet</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>0</td>
<td>2.63/0</td>
<td>4.75/4.12</td>
<td>2.26/4.05</td>
<td>4.47/3.05</td>
<td>1.82/2.69</td>
<td>2.39/2.65</td>
<td>2.36/2.56</td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>24</td>
<td>2.60/1.22</td>
<td>4.79/5.02</td>
<td>2.36/1.52</td>
<td>3.49/4.66</td>
<td>1.99/4.10</td>
<td>3.42/3.77</td>
<td>3.08/2.73</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>0</td>
<td>2.58/0</td>
<td>4.86/4.90</td>
<td>3.23/3.48</td>
<td>3.69/0</td>
<td>1.52/1.21</td>
<td>0/1.82</td>
<td>1.69/0</td>
<td></td>
</tr>
<tr>
<td>+</td>
<td>24</td>
<td>2.50/1.22</td>
<td>4.19/5.94</td>
<td>2.79/4.56</td>
<td>3.45/4.44</td>
<td>1.52/1.51</td>
<td>3.34/3.11</td>
<td>3.18/3.76</td>
<td></td>
</tr>
</tbody>
</table>

No statistical analysis due to small number of samples

1) n=1
TABLE 4. Oocysts counting (Log 10/g pooled excreta) of secondary infected birds during experimental period for both trials

<table>
<thead>
<tr>
<th>Floor heating</th>
<th>Litter (h/d)</th>
<th>Exposure to Wet</th>
<th>8 (n=2)</th>
<th>12 (n=2)</th>
<th>16 (n=2)</th>
<th>20 (n=2)</th>
<th>24 (n=2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>0</td>
<td></td>
<td>2.00/0.000</td>
<td>2.22/2.56</td>
<td>4.89/4.01</td>
<td>3.39/3.86</td>
<td>3.57/2.73</td>
</tr>
<tr>
<td>-</td>
<td>24</td>
<td></td>
<td>2.97/2.60</td>
<td>5.15/4.28</td>
<td>5.15/3.96</td>
<td>3.54/2.69</td>
<td>3.66/2.92</td>
</tr>
<tr>
<td>+</td>
<td>0</td>
<td></td>
<td>1.52/0.000</td>
<td>1.82/0.000</td>
<td>1.52/1.00</td>
<td>1.52/1.82</td>
<td>2.82/0.000</td>
</tr>
<tr>
<td>+</td>
<td>24</td>
<td></td>
<td>1.82/3.25</td>
<td>3.41/4.26</td>
<td>3.76/3.60</td>
<td>2.12/2.12</td>
<td>3.72/3.92</td>
</tr>
</tbody>
</table>

No statistical analysis due to small number of samples

TABLE 5. Development of external and histopathological foot pad scores (two factor variance analyses; mean ± SD)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Factor</th>
<th>Treatment</th>
<th>Day (Duration of Treatments)/FPD Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>21 (7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(n=40)</td>
</tr>
<tr>
<td>Trial 1</td>
<td>Floor Heating</td>
<td>-</td>
<td>1.38$^a$±0.711</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+</td>
<td>0.825$^{b,c}$±0.572</td>
</tr>
<tr>
<td></td>
<td>Exposure to Wet</td>
<td>0</td>
<td>0.837$^{d,e}$±0.485</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24</td>
<td>1.37$^{a,c}$±0.782</td>
</tr>
<tr>
<td>Trial 2</td>
<td>Floor Heating</td>
<td>-</td>
<td>0.935$^{a}$±0.400</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+1</td>
<td>0.410$^{a}$±0.427</td>
</tr>
<tr>
<td></td>
<td>Exposure to Wet</td>
<td>0</td>
<td>0.512$^{a}$±0.486</td>
</tr>
<tr>
<td></td>
<td></td>
<td>241</td>
<td>0.842$^{a}$±0.436</td>
</tr>
</tbody>
</table>

$^{a,b,c,d,e}$ Means in the same column within each criteria in each trial with different superscripts are significantly different (p < 0.05)

$^u$,$^w$,$^x$,$^y$,$^z$ Means in the same row within each criteria in each trial with different superscripts are significantly different (p < 0.05)

1) n=39
TABLE 6. Development of external and histopathological foot pad scores during experimental period (mean ± SD)

<table>
<thead>
<tr>
<th>exposure to wet heating (h/d)</th>
<th>floor</th>
<th>day (duration of treatments)/FPD scores</th>
<th>external</th>
<th>histopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>trial 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>0</td>
<td>1.05±0.426</td>
<td>2.10±0.994</td>
<td>3.12±0.856</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>1.72±0.785</td>
<td>3.95±0.723</td>
<td>4.45±0.483</td>
</tr>
<tr>
<td>+</td>
<td>0</td>
<td>0.625±0.455</td>
<td>1.50±0.458</td>
<td>1.75±0.550</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>1.02±0.617</td>
<td>2.37±0.775</td>
<td>2.42±0.674</td>
</tr>
<tr>
<td>trial 2</td>
<td>24(\text{a,b,c,d})</td>
<td>1.02±0.389</td>
<td>2.78±0.115</td>
<td>3.71±0.932</td>
</tr>
<tr>
<td>+</td>
<td>0</td>
<td>0.175±0.493</td>
<td>1.27±0.595</td>
<td>1.30±0.299</td>
</tr>
<tr>
<td></td>
<td>24(\text{a,b,c,d})</td>
<td>0.657±0.410</td>
<td>1.68±0.820</td>
<td>1.73±0.562</td>
</tr>
</tbody>
</table>

\(\text{a,b,c,d}\) Means in the same column in each trial with same superscripts are not significantly different (\(p < 0.05\))

\(\text{a,b,c,d}\) Means in the same row in each trial with different superscripts are significantly different concerning external scores (\(p < 0.05\))

\(n=19\)

FIGURE 1. Intensity of coccidial infection on DM content of excreta (%, mean ± SD)
7 General discussion

A low prevalence and severity of foot pad dermatitis (FPD) are of great concern for many producers regarding both the bird’s performance and product quality. The cause of FPD – as demonstrated here in each experiment - seems to be very complex. Many factors have been implicated in the prevalence of FPD such as: nutrient supply, type of litter, litter management and stocking density (MAYNE 2005). Nevertheless, pure water (without excreta) alone in the litter is sufficient to produce severe lesions (YOUSSEF 2011). Therefore, distinct trials were conducted to find out the critical litter moisture content that results in higher severity of FPD; to investigate the effects of litter type/floor heating; to quantify the impact of the dietary factors (surplus of electrolytes) and finally to test the effects of a coccidial infection (wet litter as a consequence of an experimental infection) on the development and severity of FPD in young turkeys housed with/without floor heating.

7.1. Experiments in details

7.1.1. Experiment 1

Only proper litter conditions can guarantee the health of foot pads. Many authors have found positive correlations between litter quality, particularly moisture and the incidence of FPD (HARMS and SIMPSON 1977; EKSTRAND et al. 1997; YOUSSEF 2011). A significant difference (p < 0.05) was found for FPD scores when comparing birds reared continuously on dry litter for 24 h/d with those housed on wet litter for 4 or 8 h/d as wet litter may soften the epithelium of foot pads which results in a skin being more prone to contact dermatitis (GREENE et al. 1985; MAYNE 2005; MAYNE et al. 2007b; YOUSSEF 2011). Moreover, the severity of FPD was extremely high for birds housed on wet litter containing 65% moisture. Similarly, MELUZZI et al. (2008) observed that the higher the litter moisture was the higher the FPD scores were in broiler. Also, MARTLAND (1984) found a positive association between high litter moisture and FPD. The effect of exposure time was on focus for the first time in this experiment. By doubling the time of exposure (8 h) the severity of FPD was only slightly increased compared with those birds exposed to only 4 h, primarily for lower litter DM content. This might be expected as prolonged contact of the foot pads to wet litter brings more irritants in the litter and excreta closer to the foot pads. Nevertheless, YOUSSEF (2011) observed that the higher pH, NH₃ and uric acid content in the wet litter did
not increase the severity of FPD and the high moisture alone 73 % for 8 h/d, without the presence of excreta, was sufficient to cause FPD.

Nevertheless it was not clear whether the high prevalence of FPD was due to the high moisture content or to the prolonged exposure time. However, an exposure of birds to wet litter containing 35 % moisture for only 4 h/d was definitely enough to induce a significant increase in external and histopathological FPD scores thus indicating that the critical moisture content in the litter may be at least 35 %. Presumably, even at shorter exposure time (1-2 h) would lead to the same result. Both factors (moisture content/exposure time) significantly and additively influenced severity of FPD. JODAS and HAFEZ (2000) found that the maintenance of proper litter quality with a moisture content of 25-30 % is probably the most important factor to lower the incidence and severity of FPD. Also, YOUSSEF (2011) found that the severity of FPD began to increase significantly at a moisture content exceeding 30 %. The question that should be tested in further studies is whether the moisture of the whole litter depth needs to be > 30 % (as it was measure here). Presumably, the water content at the surface of the litter is the dominant factor (and not the moisture of the whole litter). Thus, in future experimental studies a method should be developed that gives primarily an information on the “surface moisture”.

7.1.2. Experiment 2

The birds’ health and welfare could be influenced by direct contact of foot pads with the litter and hence the development of FPD. Using floor heating reduced the severity of FPD significantly. These results raise the question why the reduction of the severity of FPD in the floor heating groups was significantly compared with groups housed without floor heating. Two possible explanations are that the significant effect of using floor heating on FPD scores could be due to the litter becomes dry as fresh litter. Second explanation could be due to effects of floor heating leading to “warm foot pad” causing vasodilatation of the blood vessels, increasing the blood flow and promotion healing. The principle of warming effect on blood flow was also stated by NISHA (2003) causing vasodilatation of the blood vessels and increasing the blood flow in human. On the other hand with the absence of floor heating the litter is quite cool and might lead to blood vessel constrictions in the foot pad with a “cold wet foot pad”. The source of warming in turkey houses is hanging from above the pens; so the
upper surface of litter will be warm but cold from the bottom of the floor is creeping “upstairs”. Our findings accord with the previous ones (BERG and ALGERS 2004) who found that using floor heating has a significant positive effect on FPD with a prevalence of 21.5 % ± 3.7 for floor heating groups vs. 45.0 % ± 7.1 for groups not using floor heating. Furthermore, litter type has a great influence on the severity of FPD. Using lignocellulose resulted in lower FPD scores than wood shavings thus indicating that the physical form of litter either soft (lignocellulose) or sharp edges (wood shavings), may contribute to decreasing or increasing incidence of FPD. Our findings tally with those of BILGILI et al. (2009); BERK and HINZ (2010) and YOUSSEF (2011) who observed that turkeys housed on lignocellulose had a lower incidence of FPD than those housed on hard wood shavings which could be attributed to the higher absorbing capacity and also fast release of water. Providing dry clean litter 8 h/d resulted in markedly decreasing the severity of FPD in groups without floor heating. On the contrary, using floor heating resulted in similar effects on foot pads as those experienced when, providing dry litter 8 h/d. These results are consistent with the findings of GREENE et al. (1985) in a field study in broilers, observing a rapid healing of lesions when the litter became drier. Up to now it is an open question whether the positive effects of floor heating are related to the fast drying of litter surface (in spite of adding water on each day) or to the higher temperature which come in contact with foot pads’ skin. To differentiate between these two interesting hypothesis new experimental studies are needed that allow a warming and also a cooling of the ground where foot pads are in contact with (at comparable moisture contents).

7.1.3. Experiment 3
Nutritional factors that increase water intake and excretion may contribute to FPD. Additionally, the high prevalence and severity of FPD were correlated to high litter moisture (BILGILI et al. 2009). Based on the feed composition, electrolyte levels play a major role, due to increased water intake and moisture in the litter predisposing the birds to FPD. Feeding high electrolytes diet and the absence of floor heating resulted in significantly higher FPD scores. This could be explained as feeding a high Na and K diet that leads to high water intakes and consequently high litter moisture (SMITH et al. 2000). However, YOUSSEF (2011) observed that the dietary Na content up to 2 g/kg had no marked effect on the severity
of FPD in turkey poult. Also, YOUSSEF (2011) indicated that the litter quality was not affected by dietary excess of Ca, P, Mg or Cl but markedly affected by high K contents. Nevertheless, using floor heating for birds fed a diet with high electrolyte levels resulted in significantly lower external FPD scores compared to those fed high electrolytes diet and in absence of floor heating. Despite of forced water intake by feeding high electrolyte levels, the litter became drier when floor heating was in use. Therefore, floor heating is likely to be highly effective in reducing the prevalence and severity of FPD significantly. In addition, exposure of birds to wet litter (35 % moisture) for 4 h/d resulted in higher FPD scores compared to those housed continuously on dry litter (except for G3). Suggesting that even brief exposure to wet litter (for example: around feeding or drinking places) might result in markedly increasing prevalence and severity of FPD. From veterinary point of view it is worth to underline that in spite of high Na and K levels in the diet (for example due to high proportion of potassium rich soybean products) there were no detrimental effects on foot pad health when the young birds were housed in floor heated boxes. Especially in very young birds high protein contents in the diet are necessary, here we find the highest proportion of soybean meal and at this early stage the chicken need the highest temperature. All these aspects could be achieved or tolerated by the animals when a floor heating system would be implemented. May be that in the future special barns with a floor heating system come in use and the fattening is done in barns without this expensive technique.

7.1.4. Experiment 4

Litter moisture is considered to be a leading factor predisposing to FPD (JENSEN et al. 1970). Thus, FPD can be kept at a minimum if proper litter management is practised. Based on intestinal infections, coccidiosis plays a major role, due to diarrhoea and increased moisture in the litter predisposing the birds to FPD. Using floor heating for birds resulted in significantly lower external FPD scores compared with groups without floor heating. Despite of forced watery excreta by coccidia infection, the litter became drier when floor heating was in use. Thus, with increasing prevalence and severity of FPD on farms, intestinal infections, such as coccidiosis should not be neglected. Excreta quality was markedly influenced by the coccidial infection and consequently led to a decreased litter DM content and increased severity of FPD. Furthermore, in groups housed without using floor heating, daily exposure to
wet litter resulted in significantly higher FPD scores. This could be explained by the fact that standing on wet litter brings the feet in constant contact with moisture and has been suggested to cause soften of the foot pad and become more prone to damage, predisposing the bird to developing FPD (JENSEN et al., 1970). It is well known since decades that coccidiosis results in watery excrerta and wet litter conditions. With effective coccidiostats as feed additives those problems can be avoided or at least minimized. But this experiment was performed to demonstrate the relevance of coccidiosis as a causative factor for FPD, in recent times, too. For example due to mixing errors, confounding of diets (with/without coccidiostats) or development of resistance in some strains of coccidian, there could be chances for an out break for a sub clinical coccidiosis that can be controlled by counting oocysts in birds’ excreta, whenever FPD occurs without an explanation (for example normal K levels in the diet). But the most interesting question is whether a more dry and warm surface of litter might affect the infection as process (for example the development of oocysts) per se. This was not on focus in these experiments but it is worth to think about the chances of the floor heating technique from parasitological point of view.

7.2. General aspects

7.2.1. Exposure to wet litter
There is no doubt that wet litter will increase the prevalence and severity of FPD, as it was demonstrated in a lot of field but also experimental studies that were achieved in the own investigations presented here. But a critical question should be allowed: is the litter really the right focus? The litter is a mixture of bedding materials with increasing amounts and proportion of excreta (KAMPHUES et al., 2011). At the end of the fattening period the proportion of excreta will exceed 90 %, it means that less than 10 % of the litter material. Thus, it is presumably that the excreta (faeces and urine) release the water, which is transferred to the air and by the latter out of the barn. Under this aspect the ventilation in the barn is worth to be underlined. Then the question will arise on all factors that could influence the water release from the excreta and the mixture of litter with excreta and the transfer to the air over the litter surface. It is well known that “layer of excreta” on the litter surface impair the process of drying markedly. Thus in future experimental studies, it should be tested what are the main influences that impair/prevent the water release from fresh excreta. In the own
General discussion

experiments there was no need for an artificial ventilation (small number of birds in the barn) but for the future experimental studies it should be tested what these effects and reasons are. There are some ideas on mechanisms that could be involved such as undigested carbohydrates (that bind water) or electrolytes contents in excreta that could act on osmotic substances which could also interact with the release of water, as it was discussed by KAMPHUES et al. (2011). Further factors might be the “physical structure” of excreta; it means the particles size and proportion of rough particles that present surfaces on which drying process could occur. Thus, there is no doubt that wet litter induces FPD but the most important question is: why does not the litter getting dry, instead of artificial ventilation established in the field?

7.2.2. Floor heating technique

Independent of further factors (type of litter, diet composition, artificial infection) the use of floor heating resulted in desirable changes regarding prevalence and severity of FPD. Up to now, these effects are supposed to be related to the drier surface of the litter (as it was demonstrated here in different trials), but this should be tested critically. May be the temperature (or the differences of temperature between bird and the floor) plays a special role, that is related to the birds’ efforts to minimize energy losses due to direct contact between the skin of foot pads and the ground. Those aspects should be elucidated in further future experiments. By using floor heating in the own experiments, distinct interesting interactions could be demonstrated. For example, in spite of high sodium and potassium levels in the diet no detrimental effects occurred regarding litter quality and/or foot pad health. Although the water intake increased – when birds were housed with floor heating – the litter had higher DM content at the end of the trial (d 35), indicating that the floor heating favoured markedly the release of water (transfer to the air). But in general for the first weeks of birds’ life, it could be a recommendable measure for the practice. Because in this early stage the birds need high ambient temperature, high protein diets (often high potassium levels due to high proportion of soybean meal) and are specially predisposed (between 3-6 weeks of life). Nevertheless, there is a need to look on “side effects” of floor heating technique. For example, as it was observed in field studies that it seems to be a trend for higher airborne dust levels in barns when floor heating comes in use. From veterinary point of view, it should also be tested what the effects are, when the whole litter has a higher temperature. The combined occurrence of humidity
and higher temperature might affect the diversity of micro-organisms but also of parasites (such as coccidia) and their ability to harm animal health. Moreover, some people hypothesize that using floor heating with such temperature that was used in own experiments (35 °C) at litter surface could lead to some behavioural changes for the birds such as increase the activity of birds and become more nervous.

In conclusion, at well balanced diets, at proper stocking density and good health without infections in herds, there will be no need to implement floor heating. However, with floor heating you can overcome mistakes during diet formulation/misdoing of anticoccidia and guarantee a high litter quality and avoid FPD.

### 7.2.3. Diet composition

As demonstrated in details by YOUSSEF (2011), there are many ways by which the diet could interfere with FPD “a wide spread problem in poultry flocks”. There are changes in the diet that could increase the risk of FPD, but also there are some other dietary strategies that could help to reduce or minimize the prevalence and severity of FPD (adding zinc and/or biotin in a surplus). In the present investigations it was demonstrated that a surplus levels of potassium in the diet composition (15.3 g/kg diet), did not result in detrimental effects on foot pad health when floor heating was in use. Thus, by using floor heating we will not realize those mistakes of diet composition/formulation.

But testing the diet composition, should not let us neglect the potential role of coccidiosis or – from the feed production point of view – the correct use/adding of an effective coccidiostat. In the diet composition with high efficient coccidiostat, the role of infection could be neglected. However, due to misdosing and/or inefficient anticoccidia additive in the diet, the excreta and bedding material will be markedly influenced by the coccidial infection resulting in higher scores of FPD. Thus, the consequences of own experimental studies are that in case of increased FPD problems/prevalence a chemical analysis of the diet (misdosing of nutrients/minerals) is recommended but also counting the oocysts in the excreta is necessary to detect “sub-clinical coccidiosis”. In field studies it seems to be worth to check concomitantly the FPD scores but also the counts of coccidial oocysts. In the experiment 4, there was an interesting relation between the counts of oocysts in the excreta and the DM content of excreta that could be used in the field comparison (without neglecting the other
enteric infections such as clostridia which could have identical effects and consequences). But here, the artificial infection with coccidia was chosen to demonstrate what could happen in a consequence of misedosing of additives or confounding diets (with/without coccidiostat).

7.2.4. Role of coccidiosis
To simulate a condition that could happen in a field, two replicated trials were done. It was observed that oocyst numbers in the excreta were closely correlated with the changes in excreta quality. Experiment 4 provides a major great detail on the effects of the intensity of coccidia infection on mean excreta DM. The oocyst counting “Log 10/g excreta” was being able to be classified into 3 categories (numbers 0-2 = low; numbers 2-3.5 = medium and numbers 3.5-5 = high). Accordingly, it was observed that high oocyst numbers in excreta resulted in a significantly lower excreta DM content and excreta DM content increased markedly with lower oocyst numbers. Thus, coccidial infection acts additively on excreta/litter moisture contents. This could provide a quantitative idea about correlation between excreta DM and number of oocysts which could help as a diagnostic tool in the farm.

Additionally, combination of floor heating and dry litter resulted in markedly reduced oocysts shedding in the seeder birds “primary” as well as in secondary infected birds. Using floor heating for birds resulted in significantly lower FPD scores compared with groups housed without using floor heating. Despite forced watery excreta due to coccidial infection, the litter became drier when floor heating was in use. Therefore, floor heating is likely to be highly effective in reducing the development and severity of FPD. However, the highest oocysts in the chymus were found in the group housed on dry litter with using floor heating. This an interesting point, which needs to be investigated.

7.2.5. Type of litter
Only in one experiment, lignocellulose was used as a litter material. It was found that at 35 % moisture content, lignocellulose was accompanied with lower FPD scores compared with wood shavings. Nevertheless, from economical point of view, lignocellulose will never be used for the whole fattening period (20 weeks), due to its high costs (12.5 kg/m$^2$ = 5 €/m$^2$). But some people hypothesize that very good litter conditions in the rearing period would give advantages for the following fattening period. So, may be that using lignocellulose in the
rearing period and wood shavings in the fattening period could be a solution acceptable from economical point of view. However, ABD EL-WAHAB et al. (2011, unpublished data) observed that lignocellulose in the first 6 weeks of rearing turkeys caused significantly lower FPD scores compared with wood shavings. Nevertheless, with shifting from lignocellulose (6 weeks rearing period) to wood shavings until end of the fattening period (20 weeks) did not result in marked differences in comparison to those housed continuously on wood shavings. Furthermore in another study, ABD EL-WAHAB et al. (2011, unpublished data) found that lignocellulose is much better regarding health of foot pad than straw-granulate pellets (new bedding material) in the first five weeks of rearing turkeys. But due to high costs of lignocellulose and due to some technical problems during its processing which lead to increase airborne dusts in poultry houses seem to be the most limiting factors for using lignocellulose as litter material in the field. Therefore, lignocellulose could be not suitable as a litter material for rearing turkeys but a promising bedding material for rearing broilers due to fewer amounts of lignocellulose littered and shorter rearing period in comparison to turkeys.

7.2.6. General critical points

At the end there are some critical points in these experimental studies that generalized FPD in turkeys: The results in all these experimental studies were carried out at young turkeys over a period of 3-4 weeks, but not till the end of fattening period. Therefore, might be possible that the results of FPD scores are more expressive, when birds are heavier with increasing the body weight and pressure on foot pads. Also, the proportion of excreta in the litter at the first rearing period of turkeys is absolutely different from that at the end of fattening period. Therefore, the results of FPD scores in this study do not simulate the scores in a field conditions at end of fattening period.

In our system, we had not a typical ventilation system (no need for ventilation) but perhaps under field conditions ventilation is a most important factor. Thus, air movement might target all the tested factors that already done. For example, in a field condition with ventilation system the exposure to wet litter for only 4 h/d may not be the right value. Also, the incoming air in the farms plays a role in the FPD prevalence. For example, if the incoming air is moist it will worsen the condition of the litter and consequently increase the severity of FPD. But if
the incoming air is warm, it could lead to dryness of the litter surface and hence decrease the severity of FPD.

In this thesis, the type of bird was used is only (BUT-Big 6), but there are some indications that under identical field condition, the genetic lines could differ significantly regarding the severity of FPD scores.

At the end and for the future, some comments need to be investigated:

1. to differentiate the role of temperature independent the role of water.
2. the physical form of the diet was not tested in this study, therefore the new technical processing of the diet should be tested regarding health of foot pad.
3. to standardize a method to estimate the exact water content at litter surface. As it is only the upper surface of the litter is more important because of the foot pad in contact with than the whole depth of the litter.

7.3. Recommendations for management

1. Keep the surrounding places around the drinking and feeding places dry as possible, as even short exposure to wet litter will increase the severity of FPD markedly. Recently, in north of Germany there is a new system called “changing positions of water lines” that could be effective by allowing the surface of the litter to dry. But the disadvantage of the previous system, it is very expensive.

2. If there is a surplus of energy or heat, it should be used for heating the floor (floor heating system). However, some people prefer to warm the incoming air instead of heating the floor. But in a summer season there is a need to cool the incoming air. Thus, at these circumstances, there is a chance to apply floor heating system because it is so effective.

3. Using lignocellulose as a litter material for the first 5-6 weeks of rearing turkeys and shifting to wood shavings afterwards till end of fattening period did not result in decrease the severity of FPD. Also, lignocellulose may never be used for the whole fattening period (20 weeks), due to its high costs (12.5 kg/m$^2$ = 5 €/m$^2$).

4. Out of the experimental studies that had done in this thesis and whenever in a farm with high prevalence and severity of FPD scores, the veterinarian has to think on:

1. Effect of diet composition (surplus of electrolytes) → FPD score is 4.7 (in this study).
2. Coccidial infection with wet litter (35 %) without floor heating and housed on high wet litter (65 %) for 8 h/d. FPD score are 4.45 and 4.1, respectively (in this study).

7.4. Conclusions

The key point is that the prevalence and severity of FPD were clearly affected by the litter quality. The first marked increase of FPD lesions was observed after one week of exposure for 4 h/d at 35 % litter moisture with increasing severity of FPD for higher moisture contents. Both factors (moisture content/exposure time) significantly and additively influenced severity of FPD. However, even short exposure to wet litter around feeding or drinking places may result in a markedly increased prevalence and severity of FPD. According to the increased FPD scores at longer exposure time on wet litter, it has to be emphasised that higher litter moisture contents (>35 %) have to be avoided.

Improving the general standards of rearing, considering housing facilities, equipment, management and stockmanship should be considered as these factors are mainly related to the animals’ welfare. Litter quality has a great impact on the bird’s welfare. Using floor heating even with wet litter (35 % moisture), independent of the litter type, resulted in reduced severity of FPD compared with those birds housed in pens without using floor heating. In addition, using lignocellulose as a litter material resulted in lower FPD compared with wood shavings. Generally, when feeding birds with a well balanced diet, at proper stocking density and good health without infections in herds, there will be no need to implement floor heating. However, with using floor heating the mistakes mentioned above can be overcome and guarantee a high litter quality and avoid FPD. Additionally, lignocellulose will never be used for the whole fattening period (20 weeks), due to its high costs (12.5 kg/m$^2$ = 5 €/m$^2$). High levels of electrolytes increased the severity of FPD significantly. Despite of forced water intake the litter became drier when floor heating was in use. Doubling the electrolytes levels in the diet increased the FPD scores by 50 % compared with normal levels. Using floor heating reduced the FPD scores by 40 %. Therefore, using floor heating overcomes the mistakes during diet formulation regarding FPD. Moreover, exposure to wet litter of 35 % for only 4 h/d increased the FPD scores by 10 %.
Furthermore, the present results suggest that coccidial infections were done successfully and forced diarrhoea led to a poor litter quality resulting in significantly increased severity of FPD which can be overcome by using floor heating especially with a diet without anticoccidia. Generally, keeping litter dry could be achieved by using “floor heating” which could be a practical step to enhance animal health and welfare.

In the puzzle of factors that could result in FPD, here experimental studies were done to demonstrate the field relevant interactions. A lot of exact data were generated that might be used for epidemiological studies (for example, the value/range of critical moisture content in the litter). But the main result and experience of the four own different experiments is whenever you change one factor of the above called “puzzle” – willing or not – you change the parameters and findings in another part of the puzzle. But finally there are recommendations for future experiments, needed/required to come to field relevant improvements regarding FPD. It is the time to optimize all factors in the puzzle.
8 Summary

Amr Abd El-Wahab (2011)

**Experimental studies on effects of**

diet composition (electrolyte contents), litter quality (type, moisture) and
infection (coccidia) on the development and severity of foot pad dermatitis
in young turkeys housed with or without floor heating

Foot pad dermatitis (FPD) is a common disease and an important aspect of poultry welfare. In recent years the level of FPD has been used to characterise the health and welfare of poultry flocks. FPD is a type of contact dermatitis affecting the plantar region of the feet, with lesions surrounded by a reddening of the foot pads as a first symptom, then discoloration and hyperkeratosis often in combination with erosions and necrosis of the epidermis, with deep ulcers occurring in severe cases. Many factors have been implicated in the prevalence of FPD, however the most important cause is wet litter. Therefore, the following questions should be investigated and answered:

1. What is the minimum level of moisture in the litter and/or the time of exposure that together result in elevated risks for FPD development?
2. What is the effect of the litter material per se, i.e. at identical feeding/watering/housing conditions with and without floor heating?
3. What are the interactive effects of high electrolyte contents in the diet, when concomitantly the modern technique of floor heating is available or not?
4. What are the consequences regarding FPD, when coccidiosis develops (here due to an experimental infection/in the field caused by missing an effective coccidiostat)?

**Material and methods:**

Four consecutive experiments were conducted on 2 week-old female turkeys (BUT-Big 6) over a period of 3 or 4 weeks. In each experiment, the birds were divided into 4 groups with 20 birds each (except in the first experiment 18 birds each). The external and histopathological scoring for foot pads were done according to MAYNE et al. (2007).
Experiment 1: The control group was housed on dry wood shavings continuously, whereas each other group was divided into two equal subgroups and exposed daily for 4 or 8 h to different moisture litter contents (35%, 50% and 65% DM) in adjacent separated boxes. These different moisture contents were achieved by adding water as required. All turkeys were fed ad libitum a commercial pelleted diet. Foot pads were assessed weekly for external and at the end of experiment for histopathological scoring.

Experiment 2: The first 2 groups were kept on wood shavings (35% moisture) with and without floor heating, the other 2 groups on lignocellulose (35% moisture) with and without floor heating. Half of birds in each group were housed for 8 h/d in adjacent separate boxes where the litter was kept clean and dry (85% DM) throughout the experiment. The temperature at litter surface varied at 35 °C in boxes with floor heating vs. 25 °C in ones without floor heating. All turkeys were fed ad libitum a commercial pelleted diet.

Experiment 3: All birds were housed on wood shavings. Two groups were fed on normal dietary levels of electrolytes (1.7 g Na; 8.5 g K and 1.5 g Cl/kg), while the other two groups were fed on a diet with doubled levels (3.3 g Na; 15.7 g K and 3.2 g Cl/kg). For each dietary treatment, half of the birds were exposed to floor heating. Half of birds in each group (n = 10) was exposed daily for 4 h in adjacent separate boxes on wood shavings litter with a “critical” moisture content (35% water). In each experiment, foot pads were assessed weekly macroscopically and at d 35 for histopathological scores.

Experiment 4: Two replicated trials were done. All birds were fed ad libitum on identical pelleted diets without anticoccidia. The first 2 groups were kept on dry wood shavings with or without floor heating; the other 2 groups were housed on wet wood shavings litter with critical moisture content (35%) with or without floor heating. Only two birds in each group were experimentally infected with *E. adenoeides* (~50,000 oocysts/bird) nominated as seeder birds and/or primary infected birds. Foot pads were assessed weekly for external scoring and at d 42 of life for histopathological scoring. The number of oocysts in excreta was determined repeatedly.

**Results:**

1. In the first experiment, it was assumed that the “critical moisture content” for the development of FPD lesions is about 35% litter moisture content. Furthermore, doubling exposure time (4→8 h) led to only slightly increased severity of FPD for the
low litter moisture contents (35 and 50 % moisture) and a higher rise for the wettest litter treatment (65 % moisture) at the end of the trial.

2. In the second experiment, lignocellulose as litter material resulted in significantly lower histopathological FPD scores (1.4 ± 0.7) compared with wood shavings (1.7 ± 0.8). Moreover, it was observed that lignocellulose showed the highest amount of dust compared with wood shavings. Using floor heating resulted in significantly lower FPD scores (0.8 ± 0.2) compared with groups without floor heating (2.0 ± 0.8). At using floor heating no significant differences were found between wet wood shavings and wet lignocellulose.

3. In the third experiment, high dietary electrolytes increased the severity of FPD (3.65 ± 1), whereas floor heating decreased it significantly (2.36 ± 0.5) due to higher DM content in the litter. Combining low electrolytes levels with floor heating system reduced the severity of FPD by about 60 %, compared to high dietary electrolytes levels without floor heating. Using floor heating resulted in higher water:feed intake ratios (2.9 and 3.6) for birds fed normal or high dietary electrolytes vs. (2.3 and 2.8) for birds housed without using floor heating and fed normal or high dietary electrolytes.

4. The coccidial infection resulted in markedly decreased DM contents in excreta and litter (14.4 % and 53.8 %) in the group exposed to wet litter without using floor heating. However, using floor heating resulted in the highest mean DM content of litter (87.1 % ± 2.9) and also the highest body weight (2626 g ± 159) despite coccidial infection. Using floor heating resulted in significantly lower FPD scores (1.7 ± 0.7) compared with groups without floor heating (3.3 ± 1.1). Birds exposed continuously to wet litter (35 % water) showed significantly higher FPD scores (3.0 ± 1.3) compared with groups unexposed to wet litter (2.0 ± 0.9).

Conclusions: Both factors (moisture content/exposure time) significantly and additively influenced severity of FPD. First significant increase of FPD was observed after exposure for 4 h on litter with 35% moisture. Additionally, lignocellulose seems to be a desirable litter material regarding FPD. However, lignocellulose will never be used for the whole fattening period (20 weeks), due to its high costs (12.5 kg/m² = 5 €/m²). Furthermore, at well balanced diets, at proper stocking density and good health without infections in herds, there will be no
need to implement floor heating. However, with floor heating mistakes during diet formulation can be compensated and guarantee a high litter quality and avoid FPD. Also, the present results suggest that watery excreta caused by coccidial infection led to a poor litter quality and hence increased the severity of FPD which can be overcome by using floor heating.

Thus, with increasing prevalence and severity of FPD on farms, the veterinarian has to think not only of the diet composition (electrolytes) and management but also of intestinal infections, such as coccidiosis which should not be neglected. Excreta quality and bedding material were markedly influenced by the coccidial infection resulting in higher FPD scores. In the “puzzle” of factors that may provoke FPD there is also a variety of measures to avoid or to minimize the widespread problem in poultry production. At the end, this investigation presents diverse examples of interactions within the “puzzle” that have to be considered when reasons of FPD or measures against it are on debate.
Zusammenfassung

9 Zusammenfassung

Amr Abd El-Wahab (2011):

Experimentelle Untersuchungen zu Auswirkungen der Futterzusammensetzung (Elektrolytgehalt), der Einstreuqualität (Art, Feuchte) und einer Darminfektion (Kokzidien) auf die Entwicklung und den Schweregrad der Fußballenerkrankung junger Puten bei unterschiedlicher Haltung (ohne/mit Fußbodenheizung)

Die Fußballenerkrankung (foot pad dermatitis, FPD) ist eine in der Geflügelhaltung verbreitete Erkrankung, der gerade unter Tierschutzaspekten besonders Beachtung zukommt. Häufigkeit und Schweregrad dieser Erkrankung werden neuerdings sogar als Indikatoren für die Qualität von Haltung und Management insgesamt gesehen. Die Fußballenerkrankung ist als eine Kontaktdermatitis der Fußballen zu verstehen; entsprechende Veränderungen der Fußballen erstrecken sich über Rötung, Hyperkeratose bis hin zu Erosionen, Nekrosen oder gar tieferen Ulzera. Seit langem ist bekannt, dass es sich bei dieser Störung um eine multifaktorielles Geschehen handelt, in dem die Haltung, die Fütterung, das Management aber auch Infektionen eine maßgebliche Rolle spielen. Vor diesem Hintergrund wurden in den vorliegenden Studien folgende Fragen experimentell bearbeitet:

1. Bei welchem Feuchtegehalt in der Einstreu („kritische Feuchte“) kommt es zu einer Häufung und/oder besonderem Schweregrad der FPD?
2. Welche Bedeutung hat die Einstreuqualität (Art und Feuchte) für die FPD, wenn parallel eine Fußbodenheizung eingesetzt wird oder auch nicht?
3. Welche Bedeutung hat der Elektrolytgehalt im Futter für die Einstreuqualität und die FPD, und zwar bei einer Haltung ohne bzw. mit Fußbodenheizung?
4. Welche Auswirkungen hat eine experimentell induzierte Kokzidiose auf die Einstreuqualität und die FPD (Effekte eines Mischfutters ohne Kokzidiostatika/Resistenzen bei Kokzidien)?

Material und Methoden:

Die experimentellen Untersuchungen gliederten sich in vier aufeinander folgende Versuche mit jungen Puten (B.U.T., Big 6), die ab der dritten Lebenswoche in 4 Gruppen (à 20 Tiere;

Versuch 1: Hierbei ging es um die Bedeutung der Einstreufeuchte; während die Tiere der Kontrollgruppe kontinuierlich auf trockener Einstreu gehalten wurden, waren die Tiere der 3 Versuchsgruppen für 4 bzw. 8 h je Tag einer Einstreufeuchte von 65 %, 50 % bzw. 35 % ausgesetzt. Diese unterschiedlichen Feuchtegehalte in der Einstreu wurden durch Wasserzusatz erreicht und erhalten.

Versuch 2: In diesem Ansatz wurde ein Feuchtegehalt in der Einstreu von generell 35 % gewählt, und zwar bei Verwendung von Hobelspänen sowie von Lignozellulose, jeweils mit bzw. ohne Fußbodenheizung.

Versuch 3: Bei ausschließlicher Verwendung von Hobelspänen als Einstreumaterial wurden zwei unterschiedliche Mischfutter eingesetzt (normaler/überhöhter Elektrolytgehalt). Zusätzlich wurden die Hälfte der Tiere wiederum für 4 h der kritischen Feuchte von 35 % ausgesetzt und unter diesen Bedingungen der Fütterung und Haltung die Fußballengesundheit näher bewertet.

Versuch 4: Dieser Versuch umfasst insgesamt 2 Versuchsteile (der letzte stellt prinzipiell eine Wiederholung mit Wechsel der Behandlung in den Buchtenpositionen dar). In jeder Gruppe wurden nur 2 der 20 Tiere experimentell mit Eimeria adenoeides (~ 50.000 Oozysten/Tier) infiziert, die Ausbreitung der Infektion überwacht und am Ende die Bedeutung der Kokzidiose für die Fußballengesundheit näher evaluiert. Auch in diesem Versuch wurden Einstreu- und Fußbodenheizung als zusätzliche Einflussgrößen mit bewertet (neben der kontinuierlichen Überwachung der Oozystenausscheidung in den Exkrementen).

Ergebnisse:
1. Im Versuch 1 wurde die „kritische Feuchte“ mit ca. 35 % bestimmt, d.h. bei diesem Wassergehalt in der Einstreu stieg bei nur vierstündiger Exposition die Schwere der FPD im hier gewählten Versuchsdesign erstmals signifikant an.
2. Im Versuch 2 erwies sich bei einem Vergleich von Hobelspänen und Lignozellulose die letztere als überlegen (histopathologischer Score: 1,7 ± 0,8 vs. 1,4 ± 0,7), und zwar
insbesondere unter den Bedingungen eines Verzichts auf die Fußbodenheizung (mit Fußbodenheizung: 0,8 ± 0,2; ohne: 2,0 ± 0,8).

3. **Versuch 3** Die Verdopplung der Elektrolytgehalte im Futter hatte massive nachteilige Effekte auf die Einstreuqualität sowie die Fußballengesundheit (3,65 ± 1,00). Dieses blieb aus bei der Haltung mit Fußbodenheizung (2,36 ± 0,5), da wegen der dabei günstigeren Einstreuqualität der „Fehler“ in der Mischfutterkonzeption zumindest partiell kompensiert wurde.

4. **Versuch 4**: Die experimentelle Kokzidien-Infektion hatte über die feuchteren Exkreme (Durchfall aufgrund der Infektion) erhebliche nachteilige Effekte auf die Einstreuqualität (TS-Gehalt: 53,8 %; mit Fußbodenheizung: 87,1 % ± 2,9) und dadurch auch auf die Fußballengesundheit. Auch unter diesen Bedingungen hatte die experimentelle Feuchte der Einstreu, aber auch die Nutzung der Fußbodenheizung die o.g. Auswirkungen auf die Fußballen.

Schlussfolgerungen:
10 References

Correlation between litter pH and airflow pattern.
Poult. Int. 42-46

ALGERS, B. and J. SVEDBERG (1989):
Effects of atmospheric ammonia and litter status on broiler health.
Proceedings of the 3rd European Symposium on Poultry Welfare, Tours, France, 11th to 14th June, pp. 237-241

Foot pad dermatitis in broilers and turkeys; prevalence, risk factors and prevention.

BERG, C. and B. ALGERS (2004):
The effect of floor heating and feed protein level on the incidence of foot pad dermatitis in turkeys poult.
EAAP-55th Annual Meeting (poster) L4.101, Bled, Slovenia., p.359

BERK, J. and T. HINZ (2010):
Effect of litter type on health, performance and air quality in a forced ventilated turkey house.
Proceedings of the 8th International Symposium on Turkey Diseases. Institute of Poultry Diseases, Free University: Berlin, Germany, p.11

Sand as litter for rearing broiler chickens.
J. Appl. Poult. Res. 8:345–351
BILGILI, S.F. (2009):
Factors contributing to foot pad dermatitis in broilers.
WATT Poultry USA, 10:26-27

Influence of age and sex on foot pad quality and yield in broiler chickens reared on low and high density diets.
J. Appl. Poult. Res. 15:433–441

Influence of bedding material on footpad dermatitis in broiler chickens.
J. Applied Poult. Res. 18: 583

BOSSE, H. and H. MEYER (2007):
Different methods for turkey - rearing.
Proceedings of the 4th international symposium on turkey production, Institute of Poultry Diseases, Free University, Berlin, Germany, pp.123-127

Monitoring the biological performance in broilers with special regard to subclinical coccidiosis.
Arch. Geflügelkd. 44:183-187

Investigation of the pre- and postnatal development of the foot pad skin of turkey poult.

Assessing welfare and suffering.
Behav. Process. 25:117-123
CARD, L.E. and M.C. NESHEIM (1972):
Chapter 10. Diseases and Parasites.
Poultry Production. 11th ed. Lea and Febiger, Philadelphia, PA. pp. 244-273

Coccidiosis in the turkey. A Review Article.
Avian Pathol. 37(3):205-223

CHAVEZ, E. and F.H. KRATZER (1972):
Prevention of Foot Pad Dermatitis in pouls with Methionine.
Poult. Sci. 51:1545-1548

Feed non-starch polysaccharides: Chemical structures and nutritional significance.
Feed Milling International, June Issue, pp.13-26

Pododermatitis in turkeys.
Journal of Avian Dis. 46:1038-1044

CLARKSON, M.J. (1959):
The life history and pathogenicity of Eimeria meleagridis Tyzzer, 1927, in the turkey poult.
Parasitol. 49: 519-528

Battery hens name their price: consumer demand theory and the measurement of ethological "needs".
Anim. Behav. 31:1195-1205
Animal welfare defined in terms of feelings.
Acta Agriculturae Scandinavica, 27(Section A, Animal Science Supplementum), pp.29-35

Litter Moisture and Footpad Dermatitis as Affected by Diets Formulated on an All-Vegetable Basis or Having the Inclusion of Poultry By-Product.

EKSTRAND, C. and B. ALGERS (1997):
The effect of litter moisture on the development of foot-pad dermatitis in broilers.
Proceedings of the 11th International Congress of the World Veterinary Poultry Association, Budapest, p.370

Prevalence and control of foot-pad dermatitis in broilers in Sweden.
Br. Poult. Sci. 39: 318-324

ELLERBROCK, S. (2000):

The influence of grinding and pelleting of feed on the microbial composition and activity in the digestive tract of broiler chickens.
Br. Poult. Sci. 43:569-579
EUROPEAN COMMISSION REGULATIONS (1997)

Nutritional factors affecting excreta/litter moisture and quality.
World’s Poul. Sci. 60: 64-75.

Science, values and animal welfare: exploring the 'inextricable connection'.
Animal Welfare 4:103-117

GARD, D.I.; D.C. YOUNG and M.E. CALLENDER (1969):
Simulating field conditions to evaluate coccidiostats.
Poul. Sci. 48:1811

GLEBOCKA, K. (2010):
Gut health is a critical factor for litter quality.
Alltech European Biosciences Centre: Dunboyne, Co. Meath, Ireland.

A Practical Guide University of Bristol, UK, p.19

A contact dermatitis of broilers - clinical and pathological findings.
Avian Pathol. 14: 23-38
Use of a litter material made from cotton waste, gypsum, and old newsprint for rearing broiler chickens.
Poult. Sci. 85:563–568

GROSSE LIESNER, B.B. (2007):
Vergleichende Untersuchungen zur Mast- und Schlachtleistung sowie zum Auftreten (Häufigkeit/Intensität) primär nicht-infektiöser Gesundheitsstörungen bei Puten fünf verschiedener Linien.
Doctorate thesis, Tierärztliche Hochschule Hannover, Germany.

Leg disorders in various lines of commercial turkeys with especial attention to pododermatitis:
Proceedings of the 5th International Symposium on Turkey Diseases, Berlin, Germany, pp. 11-19

Poultry coccidiosis: Prevention and control approaches.
Arch. Geflügelkd. 72(1):2-7

HARMS, R.H. and C.F. SIMPSON (1975):
Biotin deficiency as a possible cause of swelling and ulceration of foot pads.
Poult. Sci. 54: 1711-1713

HARMS, R.H. and C.F. SIMPSON (1977):
Influence of wet litter and supplemental Biotin on foot pad dermatitis in turkey poulets.
Poult. Sci. 56:2009-2012
HARMS, R.H. and C.F. SIMPSON (1982):
Relationship of growth depression from salt deficiency and Biotin intake to foot pad dermatitis of turkey poults.
Poult. Sci. 61:2133-2135

The applicability of particleboard residue as a litter material for male turkeys.
Poult. Sci. 76:248-255

HOCKING, P.M. (1993):
Welfare of turkeys.

JAMES, JR. E.C. and R.S. WHEELER (1949):
Relation of dietary protein content to water intake, water elimination and amount of cloacal excreta produced by growing chickens.
Poult. Sci. 28:465–467

A foot pad dermatitis in turkey poults associated with soybean meal.
Poult. Sci. 49:76-82

Litter management and related diseases in turkeys.
World Poult. Sci. 16:30-34

Anticoccidial drugs: lesion scoring techniques in battery and floor-pen. Experiments with chickens.
Exp. Parasitol. 28:30-36
JOYNER, L.P. (1978):


Fütterungs- und Haltungseinflüsse auf die Fußballendermatitis bei Puten. Akademie für Tierärztliche Fortbildung (ATF) Fachgruppe „Tierschutz“, 08./09.09.2011, Hannover, Germany.

KAMYAB, A. (2001):
Enlarged sternal bursa and focal ulcerative dermatitis in male turkeys.
World’s Poult. Sci. Journal 57:5-12

KENNETH, W.B. (1991):
Managing coccidiosis especially in broiler’s pullet.
Misset-World Poultry, 7:9

KHEYSIN, Y.M. (1972):
Chapter V. Sporulation of oocysts and their survival in the external environment.

Breast blisters.
Proceedings of the 12th Minnesota, Poult. Workshop, Minnesota, USA, pp.47-49
LEVINE, N.D. (1982):
Taxonomy and life cycles of coccidian

The effect of drinker design on broiler performance, water usage, litter moisture and atmospheric ammonia.
FAC report No. 488, Gleadthorpe EHF, Meden Vale, Mansfield, Notts, UK

MAROUARDT, W.C., C.M. SENGER and L. SEGHETTI (1960):
The effect of physical and chemical agents on the oocyst of Eimeria zurnii (Protozoa, Coccidia).
J. Protozoo. 7(2):186-189

MARTLAND, M.F. (1984):
Wet litter as a cause of plantar pododermatitis, leading to foot ulceration and lameness in fattening turkeys.
Avian Pathol 13(2):241-252

MARTLAND, M.F. (1985):
Ulcerative dermatitis in broiler chickens: the effects of wet litter.
Avian Pathol. 14(3):353-364

Hygiene and welfare implications of alternative husbandry systems for laying hens.
Proceedings from the 3rd European Symposium on Poultry Welfare. J.M.Faure and D. Mills, eds. Tours, France. pp.201-212
A review of the aetiology and possible causative factors of foot pad dermatitis in growing turkeys and broilers.
World Poult. Sci. 61:256-267

What causes foot pad dermatitis in growing turkeys?

Foot pad dermatitis develops at an early age in commercial turkeys.
Br. Poult. Sci. 47:36-42

Foot pad dermatitis in growing turkeys is associated with cytokine and cellular changes indicative of an inflammatory immune response.
Avian Pathol. 36(6):453-459

High litter moisture alone is sufficient to cause foot pad dermatitis in growing turkeys. British Poult. Sci. 48:538-545

A contact dermatitis of broilers epidemiological findings.
Avian Pathol. 16:93-105

Survey of chicken rearing conditions in Italy: effects of litter quality and stocking density on productivity, foot pad dermatitis and carcass injuries.
Br. Poult. Sci. 49(3):257-264
Sodium source and level in broiler diets with and without high levels of animal protein.
J. Appl. Poult. Res. 9:53-61

Effect of highprotein and all-vegetable diets on the incidence and severity of pododermatitis
in broiler chickens.
J. Appl. Poult. Res. 16:304–312

NISHA, C. (2003):
A Review: Skin Blood Flow in Adult Human Thermoregulation: How It Works, When It
Does Not, and Why.
Mayo Clin Proc. 78:603-612

OPTIZ, H.M. (1996):
Disinfection of poultry houses requires attention to details.
Poult. Diget., 8:26-31

RUFF, M.D. (1993):
External and internal factors affecting the severity of avian coccidiosis.
Proceedings of the 6th International Coccidiosis Conference, pp.73-79

Assessing animal welfare: where does science end and philosophy begin?
Animal Welfare 1:257-267

SANOTRA, G.S. and C. BERG (2003):
Investigation of lameness in the commercial production of broiler chickens in Sweden.
Report, Department of Animal Environment and Health, Swedish University of Agricultural
Sciences, Skara, Sweden.
References

Broiler Welfare: problems and prospects.
Arch. Geflügelkd. (Sonderheft 1):48-52

SCHMIDT, V. and H. LÜDERS (1976):
Ulcerations of the sole and toe pads of fattened turkey cocks.
Berlin München Tierarztlicher Wochenschrift 89(3):47-50

Effect of excess dietary sodium, potassium, calcium and phosphorus on excreta moisture of laying hens.

Factors affecting the incidence and severity of a breast blister condition in broilers.
Poult. Sci. 39:1520-1524

SVEDBERG, J. (1988):
The connection between environment and foot conditions in laying hens.
Proceedings of the 6th International Congress on Animal Hygiene, Sweden, pp.125-130

TREES, A.J. (1990):

Hock burn in broilers.
References

Hock Burn in Broilers.
Nottingham University Press, Nottingham. pp.107-122

Sporulation of *Eimeria maxima* oocysts in litter with different moisture contents.
Poult. Sci. 80:1412-1415

WHITEHEAD, C.C. and D.W. BANNISTER (1981):
Aspects of metabolism related to the occurrence of skin lesions in biotin deficient chicks.
Br. Poult. Sci. 22:467-472

WHITEHEAD, C.C. (1990):
Roche, Basle, Switzerland, pp.6-58

WISE, D.R. (1978):
Nutrition-disease interactions of leg weakness in poultry. Recent advance in animal nutrition.
Oxford: Butterworth Heinemann Ltd, pp.41-57

WYLIE, L. (1999):
Factors Affecting Poor Breast Feathering in Modern Turkeys.
Doctorate thesis, Roslin Institute, Edinburgh University, pp.182-183

YOUSSIF, I.M.I. (2011):
Experimental studies on effects of diet composition and litter quality development and severity of foot pad dermatitis in growing turkeys.
Doctorate thesis, Tierärztliche Hochschule Hannover, Germany.
Appendix

11 Appendix

Fig. 2: External score 0: skin of the foot pad and digital pads appears normal

Fig. 3: External score 1: slight swelling and/or redness of the skin of the foot pad

Fig. 4: External score 2: the pad feels harder and denser than a non-affected foot

Fig. 5: External score 3: small black necrotic areas on the foot pad
Fig. 6: External score 4: the area of necrosis < one-eighth of the total area of the foot pad

Fig. 7: External score 5: the amount of necrosis extends to a quarter of the foot pad

Fig. 8: External score 6: half the foot pad covered by necrotic cells

Fig. 9: External score 7: over the half of the foot pad covered in necrotic scales
Fig. 10: histopathologic score 0: normal dermis and epidermal layers, (ker.: keratin, epi.: epidermis; der.: dermis), H&E (2.5x)

Fig. 11: histopathologic score 1: A= hyperkeratosis (excess keratin), H&E (2.5x)
Fig. 12: histopathologic score 2: A = hyperkeratosis (excess keratin); B = epidermal hyperplasia; C = epidermal acanthosis, H&E (2.5x)

Fig. 13: histopathologic score 3: A = hyperkeratosis (excess keratin and necrotic debris); B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration, H&E (2.5x)
Fig. 14: histopathologic score 4: A = hyperkeratosis (loose and excess keratin and necrotic debris); B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = presence of inflammatory cells in dermis, H&E (2.5x)

Fig. 15: histopathologic score 5: A = hyperkeratosis (loose and excess keratin and necrotic debris); B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = presence of inflammatory cells in dermis, H&E (2.5x)
Fig. 16: histopathologic score 6: A = hyperkeratosis (loose and excess keratin and necrotic debris); B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = presence of inflammatory cells in dermis; G = ruptured epidermis “ulcer” only one lesion, H&E (2.5x)

Fig. 17: histopathologic score 7: A = hyperkeratosis (loose and excess keratin and necrotic debris); B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = presence of inflammatory cells in dermis; G = ruptured epidermis “ulcer” more than one lesion, H&E (2.5x)
Fig. 18: external score 1: A = scarce petechial haemorrhages on the mucosal surface and slight thickening of the intestinal mucosa.

Fig. 19: external score 2: A = small number of haemorrhages up to pinhead size on the mucosal surface, oedema and thickening of the intestinal mucosa.
Fig. 20: external score 3: A = many haemorrhages up to pinhead size on the mucosal surface, oedema and thickening of the intestinal mucosa

Fig. 21: external score 4: A = many haemorrhages up to pinhead size on the mucosal surface, oedema and pronounced thickening of the intestinal mucosa, strong degenerative changes in the mucosal epithelium; B = caeca are full of necrotic cheese-like debris containing many oocysts and blood traces.
Fig. 22: Overview of the histopathological alterations in the caecum infected with coccidia (H&E); A = submucosal haemorrhage (arrows) in the mucosa associated with lymphoid tissue (2.5x). B = shows infiltration with heterophilic granulocytes (light red, 40x); C = bleeding into the lumen (arrow, 2.5x); D = note that the epithelium even in the immediate surrounding remains intact (arrow, 10x)
ACKNOWLEDGEMENTS

First of all, a great thank to the great creator for the great givens (THANKS ALLAH).

I thank deeply my main supervisor Prof. Dr. Josef Kamphues the director of Institute of Animal Nutrition, University of Veterinary Medicine Hannover, Germany for giving me the opportunity to be one of the members in his work team, and to study my thesis under his supervision and for correcting my manuscripts and inspiring me with his qualities as a bright scientist with a good perspective. Also, I thank him for his excellent mentorship, motivation and confidence as well as his gentleness and welcomingness in discussing my scientific results and present it at international conferences.

I would like sincerely to express my respect and thanks to Dr. Petra Wolf from Institute of Animal Nutrition, University of Veterinary Medicine Hannover for her helpful comments and suggestions which assisted me to improve my work.

I am thankful to Dr. Christian Visscher from Animal Nutrition Institute, University of Veterinary Medicine Hannover for his kind supervision and great suggestions throughout my experiments and during writing my manuscripts and his continuous help for doing the statistics as well as his gentleness and welcomingness in discussing my scientific results.

Many thanks to Prof. Dr. Andreas Beineke from Institute of Pathology, University of Veterinary Medicine Hannover for his valuable discussion and comments during my work with microscope and for providing me all the facilities that I need in pathology institute.

I am thankful to Dr. Martin Beyerbach from Institute of Biometry and Information Processing Institute, University of Veterinary Medicine Hannover for his helpful comments and suggestions in the statistics during my work and for his patient for my questions.

I am thankful to Dr. Sonja Wolken from Parasitology Institute, University of Veterinary Medicine Hannover for teaching me the skills for examination of coccidial oocysts in the laboratory and for her helpful comments and suggestions during my work which assisted me
to improve my thesis as well as for her gentleness and welcomeness in discussing my scientific results.

I am indebted to Mr. Peter Rust Institute of Animal Nutrition, University of Veterinary Medicine Hannover as he has taught me many laboratory skills and guided me every day by his continuous advices, discussing ideas which assisted me to improve my work and to keeping me up during the hard times.

I am grateful for the assistance of my colleagues especially Mrs. Dr. Sander and technical assistants in Institute of Animal Nutrition, University of Veterinary Medicine Hannover for their support, maintaining a pleasant working atmosphere.

I offer my regards to Mrs. Ledwoch in the office of international academic affairs in the University of Veterinary Medicine Hannover for her quick responding to overcome our problems, continuous support.

I am pleased to show my gratitude to Prof. Dr. Abd El-Hady Orma and Prof. Dr. Tarek Ibrahim, Department of Nutrition and Nutritional Deficiency Diseases, Faculty of Veterinary Medicine, Mansoura University, Egypt. They have supported my interest in research during both of my master and doctoral studies. I thank them for their continuous support, encouragement and valuable guidance.

All this work wouldn’t have been possible without the encouragement and support of my family. I would like to thank my parents for teaching me good values, and having faith in me all through these years. Thanks to my brothers for their love. I am sure that the success of this work would make them delighted.

Finally with thank sincerity; I thank my wife and my son. I am very proud of them. I would like to thank them for completing my life, their loving support, for keeping me up during the hard times, for their patience and understanding.