Experimental studies on effects of diet composition and litter quality on development and severity of foot pad dermatitis in growing turkeys

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Ibrahim Mohamed Ibrahim Youssef
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Academic supervision: Univ.-Prof. Dr. Josef Kamphues

Institute of Animal Nutrition
University of Veterinary Medicine Hannover, Germany

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

Institute of Animal Nutrition
University of Veterinary Medicine Hannover, Germany

2. Referee: Priv.-Doz. Dr. Gerhard Glünder

Clinic for Poultry
University of Veterinary Medicine Hannover, Germany

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To my parents and my family
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List of abbreviations

FPD foot pad dermatitis
SBM soybean meal
OS oligosaccharides
DM dry matter
SCFA short chain fatty acids
MOS mannan oligosaccharides
VDLUFA Verband Deutscher landwirtschaftlicher Untersuchungs- und Forschungsanstalten
NSP non starch polysaccharides
ME metabolizable energy
h hour
d day
°C grad/degree Celsius
Fig. figure
ppm parts per million
NfE nitrogen free extract
FCR feed conversion ratio
TS Trockensubstanz
SES Sojaextraktionsschrot

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

1.1 Foot pad dermatitis: definition and prevalence in turkeys

Foot pad dermatitis (FPD), also known as plantar pododermatitis is a condition characterized by lesions on the ventral foot pad of poultry. It is also commonly termed ‘pododermatitis’ or ‘foot pad burns’. FPD is a widespread challenge in turkey production and is a potential economic and welfare issue in intensive production systems. The prevalence of FPD in turkeys is extremely high, where it can reach to 98% in turkey poult (Ekstrand and Algers, 1997). Berg (1998) estimated the prevalence of FPD in Swedish turkeys to be 20% for severe lesions (ulcers) and 78% for mild lesions (discolouration, erosion). Paulus (2002) reported a prevalence of 70% among female, and 78% among male turkeys in the USA, whilst the UK showed 67% prevalence in female, and 83% in male turkeys. Moreover, Hafez et al. (2004) observed that the prevalence ranged from 91 – 100% in meat type turkey lines. Only 2.80% of turkeys in Germany showed unaffected foot pads at the end of the fattening period when turkeys of five genetically different lines were tested (Große Liesner, 2007).

1.2 Character of foot pad dermatitis

1.2.1 Macroscopic lesions

Foot pad dermatitis is a type of contact dermatitis (Nairn and Watson, 1972; Greene et al., 1985) affecting the plantar surface of the feet. Turkeys of almost all ages suffer from FPD and the disease can develop at a very early age (Mayne et al., 2006b). At an early stage, discolouration of the skin is observed. Hyperkeratosis and necrosis of the epidermis can develop, and in severe cases, these changes are progressed into ulcers with inflammatory reactions of the subcutaneous tissue (Ekstrand et al., 1997). The ulcers are often covered by crusts formed by exudate, faecal material and litter. The lesions can develop in less than a week and then progress to ulcers (Greene et al., 1985). The skin of the affected foot pad becomes hard, scaly, swollen and necrotic (Mayne, 2005). The lesions mainly affect the metatarsal pads but may also involve the digital pads of the feet in severe cases. Plant material and bacteria were usually present on the surface within necrotic debris, but were seldom found in deeper layers of affected tissues (Martland, 1985; Greene et al., 1985) suggesting that FPD is - primarily- not a response to bacterial invasion (Mayne et al., 2006b). FPD develops at an early age in commercial turkey flocks, where skin discolouration appears at 1 week-old, fully developed lesions (in which the integrity of the epidermis has been lost).
occur by 3 weeks of age and from 6 weeks onwards lesions simply increase in size (Mayne et al., 2006b; 2007b). Furthermore, turkeys aged 6 to 21 weeks with foot pad lesions showed increasing levels of necrosis. It was observed that the external signs of FPD were evident within 48 h after continuous exposure of growing turkeys to wet litter (Mayne et al., 2007c), this supports the findings of Breuer (2005) that young turkey pouls might be more sensitive to FPD than older one. Also, Clark et al. (2002) observed the FPD as early as 3 days of age as a reddening of the foot pads. Moreover, Mayne et al. (2007b) found that FPD lesions increased rapidly from 2 to 6 weeks and remained similar thereafter.

Foot pad lesions developed in older turkeys seemed to be different from those seen in younger birds. Younger birds exhibit scaliness and/or thickening and cracking of the skin, whereas older birds also show an enlarged ball of flesh within the foot pad which sometimes contained scar tissue (Richardson and Wilgus, 1967).

The severity of the lesions can be estimated by a scoring system which is important in determining the prevalence of FPD. A number of these scoring systems are available; the major ones are those of Martland (1984; 1985) and Ekstrand et al. (1997). These scoring systems assess the degree of external lesion development according to the size of the lesions, and the extent to which the foot pad is necrotic. The scoring systems record only the necrotic damage of the foot pad, but do not include the early stages of FPD such as changes in colour or texture of the skin. Other scoring systems used by turkey producers and companies are all based on modifications to these previous systems. Recently, Mayne et al. (2007c) devised and recommended “an external scoring system” which involves all stages of foot pad dermatitis including that were not assessed by the previous systems. This scoring procedure was accepted by the majority of attendances at the meeting of the Working Group 10 (Turkey)-World’s Poultry Science Association (WPSA) in Berlin (2007). Moreover, this scoring system was modified to 5 categories, instead of 8 grades, to facilitate the scoring of foot pads at slaughter plants (Hocking et al., 2008), where the time of inspection of foot pads is limited due to the high speed of the slaughtering process.

### 1.2.2 Histopathological findings

The histological development of foot pad lesions in turkey pouls was studied by Platt et al. (2001). At 6 weeks they observed hyperkeratosis of the foot pads and separation of keratin layers (horn cells). Lesions were mostly superficial but sometimes ulceration extended into the dermis. Both superficial and ulcerative lesions were accompanied by infiltration of
granulocytes and lymphocytes but also by a proliferation of lymph follicles in the adjacent dermis. After 14 weeks, the incidence of superficial lesions decreased, while more severe ulceration increased. Also, the thickness of the keratin layer increased and the separation of horn cells was worst as well. The hyperkeratosis increased to such an extent that the interpapillary fissures were filled with horn cells. Mayne et al. (2006b) noted also that FPD lesions are associated with massive increases in heterophils and macrophages and the loss of surface keratin. They also reported that, as the turkeys aged, fewer superficial lesions were observed, while the number of ulcers that spread into the dermis increased, as found in a comparison of 6- and 14-week-old turkeys (Platt et al., 2001).

Mild lesions show an infiltration of heterophils into the stratum germinativum, and also defects in keratin formation (Martland, 1984). Heterophils were also found in the dermis, sub-epidermis, and epidermis along with basophilic debris (necrotic cells) in the stratum corneum (Greene et al., 1985). Small vacuoles (often containing heterophils) have also been found in the epidermis and inside blood vessels of the foot pad (Harms and Simpson, 1975; Martland, 1984 and 1985; Greene et al., 1985). In the centre of the lesion, the epidermis and keratin were often completely destroyed, exposing necrotic tissue and a mass of inflammatory cells, predominantly heterophils (Greene et al., 1985).

In 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 keratin layer such as thickening and the formation of ‘horned pegs’ (Martland, 1984; Greene et al., 1985; Whitehead, 1990). The epidermis was more eroded, and the dermis was filled with fluid. Dermal blood vessels were congested and dilated, and sometimes necrotic (Whitehead, 1990) as well as epidermal hyperplasia at the edges of the lesion occurred.

Under experimental conditions, fully developed lesions (in which the epidermis was ruptured) were observed after 5 d in the feet of turkeys that were housed on wet dirty litter (with excreta) and also in birds housed on wet clean (free from excreta) litter. The dermis contained a dense mass of inflammatory cells. Mild cellular reactions were detected in birds reared on dry clean litter and bare concrete floors (Mayne et al., 2004). 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). From 6 weeks of age onwards, lesions were increasingly numerous and became more
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Overtly necrotic. 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).

There was no scoring system available up to 2007 to evaluate the histopathological changes that occur within the foot pad. Therefore, Mayne et al. (2007c) recorded a system for histopathological assessment of foot pad lesions, which ranged from score 0 (normal skin) to 7 (ruptured epidermis and widespread inflammatory cells). Hocking et al. (2008) proposed only an external scoring for use at slaughter plants, but did not develop a scoring system for histological evaluation of foot pad lesions.

1.2.3 Healing of lesions

The lesions of foot pad dermatitis may heal. Martland (1985) found a rapid healing of the lesions (within 2 weeks) in broilers when the birds were transferred from wet to dry litter. Similar results were observed in turkeys by Mayne et al. (2007c) 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. This rarely takes place under commercial conditions, except when flocks are thinned as parts of the flock are removed for slaughter. Furthermore, Platt (2004) found that foot pad lesions can heal under scar formation. After healing, the foot pad does not show the normal skin pattern and has a slightly paler colour (Greene et al., 1985).

1.3 Risk of foot pad dermatitis

The foot pad lesions may cause pain, particularly in severe cases, which together with a deteriorated state of animal’s health constitutes a welfare issue. Also, the lesions can be a gateway for bacteria or other pathogens, which can cause partial or total condemnation of the carcass (Schulze Kersting, 1996; Ekstrand et al., 1997). Recently, chicken paws are of high economic value as their prices have rised due to an insatiable demand for high-quality paws in export markets. Thus, FPD causes downgrades and condemnations of these chicken paws (Shepherd and Fairchild, 2010). The inflammatory condition of the skin of foot pads is probably accompanied by suffering and pain (Julian and Gazdzinski, 1999; Buda et al., 2002; Mayne, 2005; Mayne et al., 2007c). As in the majority of the cases both feet are affected in
the same degree, severely affected birds are reluctant to walk and spend more time resting (Ekstrand and Algers, 1997). Consequently, the body weight of birds suffering from FPD was lower (Martland, 1984 and 1985; Mayne et al., 2007c) probably due to a lower feed and water intake. However, Große Liesner (2007) did not find a correlation between the severity of FPD and the body weight of turkeys at the end of the fattening period. As flocks with a high prevalence of FPD frequently also exhibit a high incidence of other types of contact dermatitis, such as breast blisters and hock burns (Greene et al., 1985; Martland, 1985).

1.4 Possible causes of foot pad dermatitis

The etiology of FPD is complex and it seems to be ‘‘multifactorial’’. However, many contributing risk factors have been suggested such as diet composition, bird weight and sex, litter moisture and litter type (Mayne, 2005). Litter quality is affected by many other factors such as ventilation, air temperature and humidity, season, amount and consistency of excreta (influenced by diet composition and digestibility), stocking density and drinker design (Mayne, 2005; Meluzzi et al., 2008a).

The potential causes of FPD can be categorised into three main classes: husbandry and environmental factors (litter moisture, litter type, weather, season, ventilation, drinker design, stocking density), nutritional reasons (protein, Na, K, Cl, Mg, soybean meal, biotin, Zn, methionine), and factors related to the animal itself (breed, age, sex, weight, pressure, health condition).

1.4.1 Husbandry and environmental factors

- Litter

Since turkeys are more or less in continuous contact with the litter during their life, the litter condition (quality) is of major concern in the pathogenesis of foot pad dermatitis. The bedding condition is influenced by the litter material and litter quality (Jodas and Hafez, 2000; Bilgili et al., 2009).

Litter moisture:

Litter moisture is the most likely factor affecting the development of FPD (Mayne, 2005). Broilers and turkeys reared on wet litter (71% moisture content) had an increased incidence and severity of FPD lesions, but the problem was alleviated by replacing the wet litter with dry and the lesions began to heal (Martland, 1984; 1985). Mayne (2005) suggested that continuous standing on wet litter may soften the foot pad, predisposing to the incidence of
FPD. Jensen et al. (1970) observed that housing of birds on wet litter also increases the chance of faecal adhesion to the feet, which has been claimed to induce FPD. Turkeys reared on wet litter, were found after 20 weeks, to have a larger number of lesions, when compared with those raised on dry litter. In turkeys that had FPD lesions, a larger mean percentage of the foot pads were ulcerated in birds raised on wet litter (60 – 71% moisture) when compared with those reared on dry litter (18 – 48% moisture; Martland, 1984). Harms and Simpson (1975) also found that broilers housed in pens with damp litter had more foot pad dermatitis than those housed in pens with dry litter.

The foot pad lesions are thought to be caused by a combination of wet litter, high ammonia content and other chemical factors in the litter or excreta (Nairn and Watson, 1972; Harms et al., 1977; Greene et al., 1985; Martland, 1985). Wet litter was found to contain a higher concentration of nitrogen and higher pH values than dry litter (Lerner, 1996; Alchalabi, 2002), resulting in 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, and thus the presence of excreta in the litter may be not necessary for the development of FPD (Mayne et al., 2007c). Similar results were reported by Youssef et al. (2008). 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,c). Moreover, Mayne et al. (2004) found severe foot pad lesions in birds housed on wet litter, but birds reared on dry clean litter and on bare concrete floor (no litter) exhibited mild lesions that may be due to the fact that the feet were maintained relatively dry or that there was less of the putative irritant released from the litter.

Litter moisture is considered to be the major factor leading to foot pad dermatitis (Jensen et al., 1970; Wang et al., 1998; Butterworth, 1999; Clark et al., 2002; Meluzzi et al., 2008a,b; Allain et al., 2009; Youssef et al., 2009). The moisture in the litter affects the general condition of the litter and therefore is also expected to increase proliferation of bacteria, but its direct effects on foot dermatitis are more likely related to change of litter surface condition. Lynn and Spechter (1987) observed that when the moisture content in the litter exceeds 46%, the litter surface becomes wet and unfriable. 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). Also, Abbott et al. (1969) found that wet or “crusty”
litter resulting in the incidence of foot pad dermatitis. They suggested that foot pad dermatitis can be maintained at a minimum if ‘‘proper litter management’’ is practiced.

Moisture contents in litter exceeding 35% are detrimental to health of birds, often resulting in foot pad dermatitis (Harms et al., 1977; Martland, 1984 and 1985; Kamyab, 2001, Mayne et al., 2006a; Glebocka, 2008; Youssef et al., 2010; Abd El-Wahab et al., 2011). There is a strong association between ‘‘poor litter quality’’ and the prevalence of FPD (Martland, 1985; Ekstrand and Algers, 1997; Martrenchar et al., 2002; Spindler et al., 2005; Haslam et al., 2006; Mayne et al., 2007c). 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 foot pad dermatitis (Jodas and Hafez, 2000; Glebocka, 2008).

Obviously, any stress factor (physical, chemical, or infectious) that affect the integrity and optimal function of the gastro-intestinal tract can induce enteritis, diarrhea and malabsorption, all of which rapidly increase excessive nutrients and moisture excretion into the litter, leading to wet litter conditions (Bilgili, 2009). Furthermore, the diet composition can affect the litter quality through the amount and constituents of the excreta which are likely to affect the moisture content and pH of the litter (Haslam et al., 2007). High levels of certain dietary nutrients such as Na, K, Cl, Mg and protein (nitrogen) may increase the water intake and excretion (Eichner et al., 2007; Kamphues et al., 2009). Soybean meal may contribute to FPD by production of wet or ‘‘sticky’’ excreta (Jensen et al. 1970). The effects of these dietary factors as possible causes of FPD will be discussed later in more details. In poultry, wet excreta and/or litter are often caused by unbalanced feed composition and poor quality of diet and water (Jodas and Hafez, 2000; Kamphues, 2005). 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. Although most of the studies suggest that litter moisture is a critical agent in the development of FPD, other researchers have found no significant correlation between litter moisture and the prevalence or severity of FPD (Eichner et al., 2007; Nagaraj et al., 2007b).
Introduction

**Forced water intake**
- high barn temperature
- secondary to diet composition
- water quality (e.g. SO$_4^{2-}$-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 non-starch 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 temp.)

**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.)

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

**Type of litter:**

The most commonly used litter materials in poultry are wood shavings and cereal straw. Rice and peanut hulls are other materials that can be used regularly as bedding materials where it is economically feasible (Grimes et al., 2002). The type of litter appears to have a marked effect on the incidence of FPD. Turkeys reared on fine particleboard had a lower incidence of FPD, breast lesions and foot abnormalities, than coarse particleboard residue or hardwood shavings (Hester et al., 1997). Perhaps due to the jagged edges and coarser texture, coarse particleboard increased the incidence of foot pad dermatitis when compared to the other two litter sources. Consequently, the physical structure of the litter (soft/hard) may affect the incidence of FPD. 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).
In turkeys, Mayne et al. (2007c) reported that long barley straw was associated with high foot pad scores on wet and dry treatments. In addition, the foot pad scores for turkeys on reprocessed cardboard chips were similarly high like on barley straw, whereas scores were similar on paper litter and pine wood shavings whether dry or wet. This may be attributed to a poor water absorbing capacity in both straw and recycled cardboard chips. Also, Ekstrand and Algers (1997) found that turkeys reared on straw in commercial conditions showed a higher prevalence of FPD than those reared on wood shavings. This finding is consistent with that of Rudolf (2008). Similar results were also observed in broilers (Su et al., 2000; Sirri et al., 2007; Meluzzi et al., 2008b; Berk, 2009b; Baere de et al., 2009; Bilgili et al., 2009), where chopped straw was associated with the highest severity of FPD. The type of litter material may affect the amount of moisture which is trapped within the litter (Berk, 2008; 2009a). 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 (Kuczynski and Slobodzian-Ksenicz, 2002; Ritz et al., 2005; Bilgili et al., 2009). However, McIlroy et al. (1987) and Bruce et al. (1990) found no significant difference in the occurrence of hock and breast lesions in broilers reared on straw or wood shavings in commercial flocks. The litter deterioration was found to occur with equal frequency on both straw and wood shavings, resulting in the same incidence of lesions. Lignocellulose as alternative type of litter was reported to reduce the incidence and severity of FPD in female turkeys, when compared with wood shavings alone or combined with straw (Berk, 2007). The same results were observed in male turkeys when lignocellulose was compared with pelleted straw or wood shavings during the rearing time (up to 6 weeks) of animals (Berk, 2009a). This could be related to higher water binding ability of lignocellulose, and thus resulting in a dryer litter. Lignocellulose is produced from wood by chopping the wood into fine particles which are then pressed into pellet form using steam and high temperature. Moreover, sand was also found to be an acceptable litter alternative to pine shavings, consistently showing a lower prevalence of foot pad lesions in broilers compared to pine shavings (Bilgili et al., 1999a,b). Recently, Bilgili et al. (2009) studied the effect of different litter materials (pine shavings, pine bark, chipped pine, mortar sand, chopped straw, ground hardwood pallets, ground door filler (a wood fiber-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. Moreover, 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 pine shavings; however, there was more “caking” with the cotton waste products.

A really modern trend is the use of maize silage as a litter in starting pouls (Bosse and Meyer, 2007). In the breeding shreds, there is currently both moist and dried maize silage being used. The moist silage is introduced 2 -3 days before the pouls are brought into the shed and is dried at a temperature of 35 ºC. At a depth of 2 -3 cm, it should be mixed and possible spread and dried. 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, Wilms-Schulze Kump, 2007), which may lead to a lower incidence of FPD.

### Litter depth:

The prevalence of foot pad dermatitis 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., 1997). A possible explanation could be that the chickens are less prone to peck, scratch and turn the litter particles over if the layer of litter is thick and compact, and are thereby less effective in ventilating the litter and keeping it dry. Also, Martrenchar et al. (2002) observed that high-quality flocks were reared on thin layers of litter and adding a big quantity of litter appeared to be a risk factor for FPD, but it is difficult to state whether this was the cause or the degradation of the litter condition. In contrast to these results, Meluzzi et al. (2008b) 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). An increase in final litter depth at the end of the broiler cycle was associated with a lower hock burn score (Haslam et al., 2007). 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, as well as differences in the type of floor.
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- Drinker design

Drinker design can affect the moisture content of litter and thus eventually lead to FPD. Broiler flocks with small water cups showed significantly higher prevalences of FPD when compared with water nipples (Ekstrand et al., 1997). However, Ekstrand and Algers (1997) reported that turkey flocks reared with small cup drinkers had a lower prevalence of FPD than those with bell drinkers. This finding is supported by the results of Tucker and Walker (1999) who found that small cup drinkers’ designs reduced the wetness of litter and consequently decreased the incidence of hock burn. In broilers, Bray and Lynn (1986) observed that nipple drinkers with small drip cups were most efficient in reducing water spillage compared with bell drinkers or nipples alone, thereby decreasing the risk of wet litter and contact dermatitis (Berg, 2004).

- Stocking density

Stocking density was reported to affect litter quality. Litter condition deteriorates rapidly and litter moisture increases as stocking density increases (Tucker and Walker, 1992; Feddes et al., 2002; Bessei, 2006). Higher stocking density was found to increase the incidence of hock and breast lesions in broilers (Mcllroy et al., 1987; Svedberg, 1988; Dozier et al., 2005). This is probably related to poorer litter quality because more cases of poor litter quality were found in highly stocked pens ($\leq 0.48$ ft$^2$/bird) when compared with low density pens ($\geq 0.49$ ft$^2$/bird (0.15 m$^2$/bird)). The effect of stocking density on the incidence of FPD is still controversial. Some studies reported that higher stocking densities are associated with a greater incidence of FPD than lower stocking densities (McIlroy et al., 1987; Ekstrand et al., 1997; Sorensen et al., 2000; Dozier et al., 2005, 2006; Haslam et al., 2007; Meluzzi et al., 2008b). This is probably due to production of higher amounts of excreta which result in a high litter moisture. However, other studies suggested that stocking density has little or no effect on the prevalence of FPD (Algers and Svedberg, 1989; Martrechear et al., 2002; Haslam et al., 2006; Sirri et al., 2007; Meluzzi et al., 2008a; Rudolf, 2008). Some researches on broilers reported that stocking density alters foot pad scores, particularly with values over 40 kg live weight/m$^2$ (Dozier et al., 2005; Jones et al., 2005), whereas Buijs et al. (2009) found that FPD was only negatively affected when density reached 56 kg/m$^2$. 
- Climate

The weather affects litter quality. High relative humidity, either outdoors (McIlroy et al., 1987) and/or inside the house (Weaver and Meijerhof, 1991), is accompanied with poor litter quality. When the air temperature decreases below a set point (the dew-point temperature), condensation occurs on exposed surfaces, including litter (Tucker and Walker, 1999). The condensation is increased if the temperature is low, and relative humidity is high. Air temperature and humidity may be affected by stocking density, season and ventilation system (Alchalabi, 2002). Poor litter quality and poor ventilation may result in high levels of ammonia. There is a significant seasonal effect on the prevalence of FPD, hock and breast lesions, with the highest prevalence found during the winter months (McIlroy et al., 1987; Bruce et al., 1990; Ekstrand and Algers, 1997; Ekstrand and Carpenter, 1998a,b; Haslam et al., 2007; Meluzzi et al., 2008a). This may be due to the effects of high relative humidity and outdoor air quality on the litter condition. In winter, there is a decrease in ventilation rates as operations try to avoid reducing house temperature and save on heating costs, resulting in an increase of relative humidity inside the house. Moreover, poor litter quality (wet and/or sticky litter) was more recorded during the winter season (McIlroy et al., 1987). Not all research reported a higher incidence of FPD in the winter months. Wang et al. (1998) observed no cases of FPD in White Leghorn chickens when outside temperature was between 9 and 15°C, but a large number of birds with FPD was found when the temperature was warmer, between 20 and 26°C. The authors suggested that moisture and temperature are important contributing factors for the occurrence of FPD.

1.4.2 Dietary factors

Nutrition is considered to be a major factor in the development of FPD along with poor litter quality. Various dietary nutrients can affect the foot pad quality such as unbalanced levels of protein, minerals or vitamins (Mayne, 2005; Kamphues et al., 2009). Distinct feed ingredients such as soybean meal, the main protein source in turkey diets, may also contribute to FPD when fed at high levels (Jensen et al., 1970; Nagaraj et al., 2006). Different mechanisms/modes of action may be involved in the etiology of FPD when dietary influences are on debate.
- Feed nutrients

Protein:
The incidence and severity of FPD was significantly affected by the level and source of protein (Nagaraj et al., 2006 and 2007b). Birds reared on low-protein diet based on vegetable and animal protein source showed the lowest incidence of FPD compared with those fed either high/low protein diets composed of all-vegetable source or high-protein diet contained vegetable and animal protein source. 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., 2007b). Similar results were observed by Eichner et al. (2007) who 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. However, Nagaraj et al. (2007c) found that protein source affected significantly the incidence and severity of FPD (all vegetable > vegetable + animal), but no effect of the protein level was observed. The author’s explanation for this could be due to excessive wetting of litter, possibly due to increased water consumption in response to high environmental temperatures during the study, thus masking the effect of protein level on the foot pad lesions. 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 a high protein diets. Excess crude protein levels in the diet could increase the production 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. Wet litter conditions and high pH act as a catalyst in this process. A combination of wet litter and high ammonia content in the litter was suggested to cause FPD (Nairn and Watson, 1972; Martland, 1985). Higher levels of NH$_3$ released from the litter may induce severe irritation to the respiratory tract and skin of birds, which result in pododermatitis, hock burns, and breast blisters (Food Animal Initiative, 2004). However, Nagaraj et al. (2007a) reported that other factors in the litter apart from NH$_3$ may play a role in the incidence of pododermatitis. Mayne et al. (2006a and 2007c) also found no direct correlation between NH$_3$ concentrations released from the litter and the incidence of FPD in turkeys. Furthermore, Nagaraj et al. (2007c) showed that the incidence of FPD was
not influenced by a high dietary protein level, despite the increased excretion of nitrogen in
the litter and a higher generation of NH$_3$. Jensen et al. (1970) observed little or no dermatitis
when poults fed a casein-gelatin-corn diet but increased the incidence of FPD with increasing
levels of soybean meal in the diet. However, the condition was not produced by substituting
isolated soybean protein for casein and gelatine, indicating that the protein portion per se of
the soybean meal was not the responsible ingredient.

Fat:
Dietary fat quality can also affect on litter surface friability (Bray and Lynn, 1986; Tucker and
Walker, 1992). Bilgili et al. (2006) evaluated the effect of low and high density diets on foot
pad quality. The low-density diet contained less fat than a high-density diet, but both diets had
identical energy and protein contents. Broilers reared on the high-density diet had
significantly higher incidence of FPD compared with the low-density diet. Up to now, it is not
known whether unabsorbed fat (fatty acids) could play a role in the development of FPD.

Mineral imbalances:
The effects of minerals, especially the macrominerals, on the incidence of foot pad dermatitis
are thought to be related to increased water intake, resulting in higher moisture content in
excreta and/or litter or due to excretion of chemically irritant macrominerals in the droppings
which may irritate the feet. 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 litter moisture, whereas an increase in Cl did not
have the same effect (Murakami et al., 2000 and 2001). The increased level of K in the diet is
related to inclusion of higher levels of plant-protein rich ingredients especially soybean meal,
and thus its effect specified in the “feed ingredients” section below. Murakami et al. (2000)
observed that higher levels of Na (exceeding 0.15%) in the broiler diets lead to increased litter
wetness. Ingestion of high levels of sodium, potassium, magnesium or phosphorus – but not
for calcium - in the feed and/or water increases water consumption/excreta moisture and
consequently contributes in production of wet litter which predispose the birds to FPD (Smith
et al., 2000; Bilgili, 2009). 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
foot pad dermatitis. Steenfeldt et al. (2005) found no effect of different dietary levels of Ca
and P on the incidence and severity of FPD in broilers. FPD is claimed to be induced by a
combination of wet litter and other “chemical irritants” in the excreta or litter (Nairn and
Watson, 1972; Martland, 1984; Greene et al., 1985). Moreover, contact with excreta may also induce FPD (Jensen, 1985).

**Nutritional deficiencies:**
Certain amino acids and vitamins as well as trace elements, such as methionine, cystine, biotin, riboflavin and zinc, are involved in formation and maintenance of the skin. Inadequate amounts of these nutrients in the diets increase the risk of FPD. Vitamins like riboflavin (Lepkovsky and Jukes, 1936a,b; McGinnis and Carver, 1947), pantothenic acid (Kratzer and Williams, 1948) and biotin (Patrick et al., 1943; Clark et al., 2002) have an impact on the prevalence of FPD. Also, deficiency of zinc resulted in a higher incidence of foot pad lesions (Whitehead, 1990; McDowell, 2003). In addition, amino acids such as methionine and lysine play a role in the development of FPD. Chavez and Kratzer (1972) found that supplementation of the diet with methionine reduced the incidence of FPD in turkey poults. A subsequent study showed that the condition was caused primarily by a deficiency of methionine and that cystine aggravated the condition (Chavez and Kratzer, 1974). The same findings were reported by Murillo and Jensen (1976) who observed a high incidence of FPD in poults fed diets unsupplemented with methionine. Adding methionine to these diets significantly lowered the incidence and severity of dermatitis.

**- Feed ingredients**
The inclusion of the feed ingredient soybean meal has been suggested to cause FPD. It is thought that indigestible carbohydrates from plant sources (primarily soybean meal) may be caustic and contribute to FPD (Jensen et al., 1970; Hess et al., 2004). 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). As the diet NSP concentrations increase, the viscosity of digesta increases, resulting in ‘’sticky’’ excreta which adheres more readily to the foot pads of the birds. Diets containing wheat with high levels of NSP tend to produce higher digesta viscosity than normal wheat diets. These diets can be improved with addition of NSP-degrading enzymes, showing significantly lower digesta viscosity than the wheat diet without enzyme (Choct et al., 1995).

Soybean meal (SBM) is the most common protein source for use in turkey diets. It contains low digestible carbohydrates by poultry, such as oligosaccharides or more complex ones (non-starch polysaccharides) as pectins, hemicellulose and cellulose (Eldridge et al., 1979; Honig and Rackis, 1979; Potter and Potchanakorn, 1985; Knudsen, 1997). The carbohydrate fraction
of SBM is considerably high, representing about more than 30% of its total dry matter. Approximately one third of them are oligosaccharides which implicated in the acceleration of feed transit in the intestine and also quickly ferment in the gastrointestinal tract (Coon et al., 1990). Other two-thirds are pectins and hemicellulose, which increase ingesta viscosity and reduce nutrient availability (Langhout et al., 2000). Some studies reported that soybean meal contains carbohydrates (oligosaccharides) which are difficult to digest leading to watery/sticky droppings and wet litter problems (Boling and Firman, 1997; Nagaraj et al., 2007b). This may increase the probability of excreta and litter adhering to the birds’ feet and causing FPD lesions. Nagaraj et al. (2006; 2007b,c) observed that broilers fed soybean meal diets showed a higher incidence of pododermatitis than birds fed SBM plus poultry by-product meal. This may be due to increasing the viscosity of gut contents and a higher total N excretion into the litter. Soybean meal contains also a high amount of potassium (> 20 g/kg DM), which is an electrolyte known to increase water intake (James and Wheeler, 1949). Similar effects of dietary potassium were also reported by Smith et al. (2000). Birds fed all-vegetable diets based exclusively on corn and soybean meal showed an increased water intake due to higher contents of K in these diets, resulting in an increased excreta or litter moisture and higher incidence and severity of FPD (Eichner et al., 2007). Vieira and Lima (2005) found higher water intake and increased moisture content of the excreta in broilers fed all vegetable diet compared to a diet containing up to 7% animal by-products with less K content. Diets formulated exclusively with corn and soybean meal resulted in litter with higher moisture content than a corn-soybean meal diet with 10% fishmeal and 0.22% less K (Murakami et al., 2000). Jensen et al. (1970) found that high dietary levels of soybean meal caused foot pad dermatitis in poults as a result of sticky excreta and subsequent irritation of the foot pad. They observed also 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 soybean meal. It was also reported that poults fed diets containing 40% or more of soybean meal exhibited a high incidence of FPD, while birds fed diets containing low amounts or no SBM had little or no FPD lesions (Jensen, 1969; Jensen et al., 1970). However, Abbott et al. (1969) reported that regardless of the level of SBM in the diet, FPD in turkey poults can be kept at a minimum level if proper litter management is maintained. They suggested the wet litter was the cause of FPD and not SBM per se.
- Feed manufacturers

Feed manufacturers are thought to affect the development of FPD due to production of diets with different compositions or ingredients. Bruce et al. (1990) found that between 1984 and 1985, flocks supplied by one feed manufacturer had a significantly lower level of hock burns and breast lesions than those supplied by two other feed manufacturers. However, the same authors observed that between 1986 and 1987, flocks did not differ significantly in hock burns and breast lesions with respect to feed manufacturer. These contradicting results may suggest that there is some variation between feed producers, depending on preferred ingredients or "treatment" of feedstuffs. McIlroy et al. (1987) and Ekstrand et al. (1998) reported significant differences in foot pad quality between feed manufacturers, with no obvious deficiencies or imbalances between the feed products. Feed quality variations between suppliers was thought to have an effect by adding moisture to the litter through droppings or by an effect on skin integrity from insufficient levels of vitamins such as biotin (Haslam et al., 2007).

1.4.3 Animal factors

- Genetic

Under experimental conditions, the turkey line can affect the prevalence and severity of foot pad dermatitis, especially in turkey lines with a heavy body weight (Hafez et al., 2004). Some studies suggested that the skin of the birds may not have adequate time to mature fast enough to carry the rapidly developing body weight (Breuer et al., 2006). 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) found also no correlation between the body weight and the severity of FPD (at slaughter) when 5 genetically different turkey strains were simultaneously compared. In broilers, genetic variation in the propensity to develop FPD and hock burns has been reported and that genetic selection may be used as a tool to prevent FPD and hock burns (Kestin et al., 1999; Kjaer et al., 2006a,b; Allain et al., 2009; Ask, 2010). Moreover, 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., 1997). Chavez and Kratzer (1972) reported that Large White poults had a higher incidence of FPD than Broad Breasted Bronze poults when reared in the same conditions on wire floors. However, this may
be attributed to the fact that Large White poults had a more rapid growth rate than Bronze poults, which result in heavier animals.

- **Age**
There are contradictory results with respect to the effect of age on the incidence of foot pad dermatitis. Several studies reported an increased prevalence of FPD with age (Stephenson et al., 1960; Bruce et al., 1990; Sørensen et al., 2002; Berk, 2009a), whereas others have not found any effect of age (Martland, 1985; Ekstrand et al., 1997). However, other researches showed an increase in FPD score to 6 weeks of age and little change thereafter (Mayne et al., 2006b; 2007b).

- **Gender**
The impact of gender is a subject of controversial debate. Some research found that females had a higher incidence of FPD than males (Kamyab, 2001; Kjaer et al., 2006b; Nagaraj et al., 2007a; Rudolf, 2008). This could be due to female skin contains more fat and less protein and collagen than males, indicating that female skin may be more likely to tear than male skin and consequently resulting in a higher incidence of FPD (Mayne, 2005). However, some authors reported a higher prevalence of foot pad lesions in males compared with females (Buffington et al., 1975; Harms and Simpson, 1975; Harms et al., 1977; Harms and Simpson, 1977; Greene et al., 1985; Mcllroy et al., 1987; Bruce et al., 1990; Cravener et al., 1992; Bilgili et al., 2006; Nagaraj et al., 2007b), while other authors found a better foot pad health in females, probably due to a lower body weight at slaughter (Clark et al., 2002). Nevertheless, other studies found no differences between males and females in the prevalence of FPD (Martland, 1984; Ekstrand and Algers, 1997; Berg, 1998; Nagaraj et al., 2007c).

- **Body weight and mechanical pressure**
FPD may be caused by rapid growth and high body weight. The increase of body weight may increase the physical pressure exerted on the foot pad. Males are heavier than females and consequently are more susceptible to develop foot pad lesions (Harms et al., 1977; Harms and Simpson, 1977; Mcllroy et al., 1987). The higher body weight in heavier birds results in more weight placed on the foot pads, leading to increased surface area contact with the litter. Since the body weight increases, the force per area (pressure) of foot pad increases, and thus the pressure on the foot pads in heavier birds may be increased (Mayne, 2005). Yet, no study has reported if pressure on the foot pads increases with bird weight. However, many studies found
no effect of body weight on the incidence or severity of foot pad dermatitis in turkeys or broilers (Buffington et al., 1975; Martland, 1984; Kjaer et al., 2006b; Große Liesner, 2007).

- Gastrointestinal infections
The health condition of animals can influence the incidence of foot pad dermatitis. Birds suffer from diarrhea, which caused by either intestinal parasites (such as coccidia), viral or bacterial infections, or by poor feed quality, this can probably result in wet litter condition (Neill et al., 1984; Custodis and Hafez, 2007) and consequently predisposing for the development of FPD. Among bacteria, E. coli and Clostridium perfringens are commonly associated with enteritis in chickens and turkeys (Gazdzinski and Julian, 1992; Akashi et al., 1993; Edens, 1997). The prevalence of Clostridium perfringens - associated necrotic enteritis in poultry has increased in countries that stopped using antibiotic growth promoters (Van Immerseel et al., 2004). A variety of other bacteria, such as megabacteria of cage birds, and ‘‘long-segmented filamentous organisms’’ (Goodwin et al., 1991) have been identified in stomach and/or intestine of birds suffering from intestinal diseases. The most prominent viruses related to wet litter and enteritis in turkeys are Rotavirus, Reovirus, Adenovirus, Entero-like and Corona-virus (Gough and Drury, 1998). Also, parasites like coccidia, Histomonas meleagridis and Ascaridia as well as fungi or mycotoxins are capable of initiating intestinal disease (McMullin, 1998). In cases of occurrence of enteric infections outbreaks, vaccination should be provided (if applicable) to prevent the incidence of such diseases.

1.5 Prevenion of foot pad dermatitis

1.5.1 Control of litter moisture
The incidence and severity of FPD can be reduced by minimising litter moisture (Mayne et al., 2007c). The litter moisture can be controlled by appropriate litter management through regular turning, removal of wet litter and addition of fresh dry bedding. Furthermore, proper control of drinker design, ventilation and humidity is also indicated (Ekstrand et al., 1997; Meluzzi et al., 2008a). The litter material should be highly water absorbent, non-dusty and clean (Berk, 2007). Optimising environment, nutrition and enteric health throughout the life cycle of animals can contribute in diminishing the development of FPD. High litter quality (loose structure of the top layer) could be achieved with a proper litter management which means frequently adding new material (‘‘top-dressing’’) and litter cultivations (Geraedts, 1983). Therefore, measures which improve litter quality may reduce the prevalence of FPD
and hock burn lesions. Litter quality may be improved using ventilation and heating so as to maintain house temperatures and relative humidity (Haslam et al., 2007). Moreover, using floor-heating systems can improve the litter condition and consequently reduce the incidence of FPD (Berg, 2004; Abd El-Wahab, 2011).

1.5.2 Application of litter amendments

Litter amendments are often used in poultry production to improve litter condition and to reduce volatile ammonia levels in houses (Blake and Hess, 2001; Watkins et al., 2003; Parsons, 2006). The most common type of litter amendments are litter acidifiers. These litter additives lower the litter pH as well as reduce ammonia volatilization by inhibiting the growth of bacteria which generate ammonia from splitting of uric acid. Some common litter amendments include sodium bisulfate, ferric sulphate, aluminium sulphate, propionic acid, monobasic Ca phosphate and clay. Sodium bisulfate effect on the prevalence of FPD in broilers was assessed by Nagaraj et al. (2007a). The NaHSO₄ was added to the litter at the rates of 0.22 or 0.44 kg/m² on the day of placement of chicks, whereas a third treatment had 0.22 kg/m² at both 0 and 21 d. It was found that the incidence of FPD appeared to improve numerically (although not significantly) with the use of NaHSO₄.

1.5.3 Dietary prophylaxis

The addition of some nutrients to the diet may help to reduce the incidence of FPD in poultry. The most commonly used additives in this aspect are biotin and zinc. The use of feed enzymes is also claimed to reduce the prevalence of FPD due to these enhance feed utilization, reduce nitrogen in the litter and NH₃ emission, and decrease excretion of water. Furthermore, the use of prebiotics is also assumed to have a relevant role in this condition.

- Biotin

Biotin plays an essential role in formation, maintenance and repair of skin through its being a cofactor for various enzymes in protein synthesis, fatty acid synthesis and carbohydrate metabolism (Whitehead, 1977). Deficiency of dietary biotin is usually manifested by hard scaly dermatitis lesions which appear firstly on the foot pads of turkeys and broilers (Patrick et al., 1942; Whitehead, 1977; Whitehead and Bannister, 1981; Oloyo, 1991). Commercial turkey diets usually contain between 200 and 300 µg biotin/kg (BUT, 2006) and it was thought that these level of biotin is inadequate to prevent the incidence of FPD in turkeys (Misir and Blair, 1988; Clark et al., 2002). Furthermore, symptoms of biotin
deficiency were noticed when turkeys were fed commercial diets without supplementation. Harms and Simpson (1975) suggested that a marginal biotin deficiency in practical diets may be responsible for the development of lesions in the foot pads of broilers. Harms et al. (1977) observed that broilers grown on wet litter had significantly increased incidence of FPD, and that the addition of biotin to the diet (200 µg biotin/kg) decreased the incidence and severity of this condition. Also, Harms and Simpson (1977) showed that the supplementation of biotin to the corn–soy diet (220 µg biotin/kg) resulted in decreased severity of FPD in poults maintained on dry litter but not in poults reared on wet litter. These findings indicate that both wet litter and a marginal biotin deficiency might be involved in the development of FPD in turkey poults. Similarly, biotin supplementations are able to reduce FPD to a certain extent if birds are reared on dry litter, but if on wet litter, lesions may still occur (Mayne, 2005).

Marusich et al. (1970) found that turkey poults fed semi-purified diet containing 100 µg biotin/kg showed 98% incidence of FPD which decreased to 0% when this diet was supplemented with 225 - 300 µg biotin/kg. Turkey poults fed a commercial ration (250 µg biotin/kg) without additional biotin for 3 weeks, had a 53% incidence of FPD which reduced to 5% when this diet was supplemented with 400 µg biotin/kg for further 3 weeks. On the contrary, Whitehead (1977) suggested that just 300 µg biotin/kg were adequate to prevent FPD in growing turkeys. Supplementation of the diet with 135 µg biotin/kg did not reduce the incidence of FPD in turkeys (Johnson, 1967). However, the poults showed a near total recovery with a supplementation of 250 µg/kg. Older male turkeys (14 week-old) exhibited no recovery from FPD when given 500 µg/kg orally for 5 weeks. The same findings were obtained for 22 week-old males injected with 1000 µg biotin for the first week, followed by 500 µg weekly. Although high levels of biotin provided, the foot pad lesions persisted. It is possible that FPD can be prevented in its early stages by increasing biotin supply in young poults, but it is difficult to be remedied when the lesions have become more severe.

Some studies demonstrated that biotin supplementation is still required to minimise FPD. Buda (2000a,b) reported that very high inclusion rates of dietary biotin (about 2000 µg/kg) reduced the severity of FPD in turkeys, when compared with the recommended dose of biotin (300 µg/kg). Moreover, Platt (2004) and Platt et al. (2001; 2004) suggested that biotin supplementation (440 or 880 µg/kg) to the diet were not able to prevent the development of foot pad lesions in male turkeys under field conditions, when compared to standard biotin
dose as used in commercial diets (220 µg/kg). Nonetheless, positive effects of high biotin levels on the healing of existing foot pad lesions were detected in the last seven weeks of fattening. Hafez et al. (2004) reported that feed supplementation with biotin (850 µg/kg) and simultaneous increase of energy content in the feed appear to reduce the incidence of FPD in turkeys. In contrast to these results, Jensen et al. (1970) reported that biotin did not reduce the incidence of FPD when injected to turkey pouls at the rate of 150 µg or when added to the diet (250 µg/kg) containing high level of soybean meal. Recently, Mayne et al. (2007b) reported that high dietary concentrations of biotin (1600 µg/kg) did not prevent the occurrence of FPD in growing turkeys. They suggested that current recommendations for biotin concentrations in diets for growing turkeys are adequate for normal growth and development.

- **Zinc**

Zinc is an essential trace element for normal growth and development and is integrated in a variety of metabolic processes, including synthesis of proteins and nucleic acids (McDowell, 2003). Zinc is necessary for optimum growth, feathering, bone development, healing of wound and immunity in poultry (Roberson and Schaible, 1958; Stahl et al., 1989). It was found that zinc deficiency (< 30 mg/kg) lead to development of FPD lesions (Whitehead, 1990). Also, deficiency of Zn decreases growth and feathering and increases leg abnormalities in broilers and turkeys (Young et al., 1958; Dewar and Downie, 1984).

Hess et al. (2001) found that foot pad lesions were reduced significantly in female broilers fed diets containing Zn-amino acid complexes (Zn-met, Zn-lys, Zn-met/Zn-lys: added at the rate of 40 ppm Zn to the diet already contained 75 ppm total Zn). However, male broilers grown under cool conditions (average temp. range 4 to 15 ºC) did not benefit from Zn complexes, indicating that the influence of Zn complexes appeared to vary with the environmental conditions. Recently, Bilgili (2009) reported that dietary zinc from organic sources decreased the incidence and severity of foot pad dermatitis under conditions of high stocking density.

- **Enzymes**

Feed enzymes were suggested to help in reducing the incidence of FPD by improving the digestibility of complex carbohydrates of SBM. These carbohydrates are poorly digested by endogenous enzyme, resulting in sticky and wet excreta and consequently predispose birds to FPD (Jensen et al., 1970). The common enzymes used are protease and carbohydrase, α-amylase, and multienzyme preparations containing xylanase, β-glucanase,
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arabinofuranosidase, glucosidase and galactosidase, and polygalacturonase. Nagaraj et al. (2007c) used a feed-grade enzyme in vegetable diets with or without animal protein to reduce FPD. The incidence of mild lesions was lower with the addition of the enzyme to the all-vegetable diet, with no differences observed when enzyme was added to the vegetable and animal protein diet. The improvement in foot pad quality was seen at later stages of the life and could be confounded with healing of the lesions.

- Prebiotic (Mannan oligosaccharide)

Mannan-oligosaccharide (MOS) is a fraction of the cell wall of yeast Saccharomyces cerevisiae, was used as a feed additive (prebiotic) for poultry (Hooge, 2003). MOS is thought to act by binding and removing the pathogens from the intestinal tract as well as stimulating the immune system and improving the integrity of the intestinal mucosa in studies with chickens and turkeys (Newman, 1994; Savage and Zakrzewska, 1997; Spring et al., 2000). The levels of inclusion MOS in the diet varied by trial and by feed phase in different studies, but in most of studies on poultry, MOS was mostly used in the diet at small doses (0.05 to 0.20 %), probably for economical reasons (Hooge, 2003).

Ferket (2002) observed that dietary supplementation of MOS product reduced total short chain fatty acids (SCFA) content of the jejunum digesta by about 40% in turkeys as well as decreased the jejunum digesta pH and ammonia concentration. MOS can also modify bacterial fermentation in the intestinal tract to increase nutrient availability to the poultry (Ferket et al., 2002). Some studies reported that MOS decreased ammonia as well as SCFA concentration (mainly acetate) in the caecal digesta when applied at medium (0.2 - 0.4 %) and higher (0.4 - 1.0 %) doses, although it did not change the caecal pH or the bacterial enzyme activity (Zdunczyk et al., 2005; Juskiewicz et al., 2006). However, Juskiewicz et al. (2003) found that the caecal ammonia concentration was significantly reduced, but the pH of caecal digesta was higher in turkeys fed a diet containing 0.1, 0.2, or 0.4 % MOS for 4 weeks compared with the controls.

Up to now, there are no available researches on the effect of MOS on the development of foot pad dermatitis. MOS is hypothesised to reduce the incidence of FPD due to its role in stimulating the immune system as well as its activity within the intestinal tract (lower pH and ammonia of the digesta; affects on the intestinal pathogens and modify the bacterial fermentation). Thus, MOS may improve the quality of excreta, resulting in excreta of higher DM content and consequently prevent the occurrence of wet litter conditions.
- Clinoptilolite (clay minerals)

Clinoptilolites are specially selected clay minerals, known for their typical structure which enables the clay mineral to bind ammonia (Suchy et al., 2006). A reduction of foot pad lesions may be expected because ammonia bound to clinoptilolite causes less irritation to the foot pads. The inclusion of clinoptilolite in broiler diets (0.5 – 1.5% on top of diet) was found to increase the DM content of the excreta, decrease water excretion, prevent wet litter formation and reduce the severity of FPD, without any negative effect on performance of birds (Hangoor and Balemans, 2007; Kampf and van der Aa, 2008; van der Aa, 2008).

1.6 Objectives of the study

The present study was conducted to find out the most relevant possible causes of foot pad dermatitis as well as to develop particular strategies which can be used to prevent it. Five experiments were conducted to evaluate the effects of specific dietary factors as well as litter type on the development and severity of FPD in growing turkeys under the influence of both dry and wet litter simultaneously. The wet litter was artificially achieved by adding water. In contrast to all previous experiments, the period of exposure to wet litter in this study was reduced to only 8 h/d, to simulate the litter condition in the field as only restricted areas of the litter in the barn are very wet especially around drinkers and feeders.

Intensive turkey meat production is characterised by high stocking density, high density of protein and nutrients, and high amounts of excreta in the litter (especially at the end of the fattening period). Some studies found that adding water to the existing dirty litter (containing excreta) resulted in FPD, but it is not clear from these findings whether the possible cause of FPD was due to the moisture per se (resulting from the water or within the excreta), protein rich diets itself, dietary mineral surplus or the litter material per se. These factors may act indirectly on the occurrence of FPD by irritation of the birds’ feet (due to moisture, main nitrogen metabolites (like uric acid and ammonia) or macromineral contents of the excreta).

Contrary to the field conditions, the present study was conducted under identical and standardised experimental conditions without any effect of artificial climatisation as there was no need for artificial ventilation for small groups of birds in a relatively large barn. All experiments of this study were performed in one barn which was divided into 4 boxes. The potential effects of dietary factors were studied under identical housing/litter conditions, whereas the influence of bedding factors was investigated under identical diet composition. In addition, the birds were housed at stocking density similar to that in the practice. Moreover,
the experiments were carried out on the turkeys in the period of life (from d 15 to d 36/42) that is supposed to be the most prone for the onset of FPD. The results of present study should finally provide recommendations to reduce the incidence and severity of FPD, but it has to be underlined that this study was conducted for a short term (like a challenge) and not for the whole fattening period.

With respect to the development and severity of FPD, the aim of the applied experiments was:

1. to identify whether pure water alone or enriched with main protein metabolites (uric acid, ammonia) in the litter is the causative agent of FPD. It has to be emphasised that this experiment was done in the absence of excreta in the litter,

2. to evaluate the effect of a surplus in dietary macrominerals which are often supplemented to the diet in higher doses than those normally required for turkeys,

3. to investigate the influence of high soybean meal (SBM) level in the diet as well as to detect which constituents in SBM are associated with the development of FPD whether soybean oligosaccharides and/or the potassium content,

4. to assess the effects of commonly used litter materials (wood shavings and straw) and of currently available alternative bedding materials (lignocellulose, dried maize silage). These litter types were tested under experimental standardized conditions and with feeding identical diets to the birds, and

5. last but not the least to find out distinct dietary measures which can help to reduce the prevalence and severity of FPD. This was tested by supplementing diets with high levels of specific nutrients (biotin, Zn) exceeding the minimum requirements or by using prebiotic such as mannan oligosaccharide.

In all trials, the development and severity of foot pad lesions were assessed by scoring the foot pads of turkeys, not only macroscopically but also histologically, at the start and end of the experiments as well as at weekly intervals.
Chapter 1

2 CHAPTER 1: Effects of litter quality (moisture, ammonia, uric acid) on development and severity of foot pad dermatitis in growing turkeys

I.M.I. YOUSSEF 1, A. BEINEKE 2, K. ROHN 3 AND J. KAMPHUES 1

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


Summary

High dietary protein is thought to increase the incidence of foot pad dermatitis (FPD) as a result of increased uric acid and secondary ammonia production in the excreta or litter. This study was conducted on female turkeys over a period of 3 weeks to test the effects of water alone and/or these end products of protein metabolism, independent of the presence of excreta, on the development and severity of FPD. The animals were allocated into 4 groups, with 20 birds in each, and housed in floor pens on dry clean wood shavings (changed daily) throughout the experiment. The control group was housed continuously in its pen, whereas the other groups were additionally exposed daily (for 8h) to experimentally treated wood shavings, in adjacent separate boxes, enriched with water alone/or with NH4Cl or with uric acid. NH4Cl and uric acid were added via water to the litter to achieve concentrations of ammonia and uric acid in the litter as found in excreta of turkeys (about 0.50g ammonia and 20g uric acid/kg). The wet litter was kept clean by removing the excreta twice daily and change of the litter twice a week. The foot pads of all birds were examined on d 0, 7, 14 and 21 and scored externally. Three birds were selected from each group on d 0, 7 and 14, while the remaining 11 birds/group were sacrificed on d 21, for histopathological assessment of foot pads. The severity of FPD was found to be markedly higher (about 3 times) on wet than on dry litter. There were no negative effects of ammonia and uric acid on foot pad lesions. The results indicate that high litter moisture is the most likely factor causing FPD in turkeys. A focus on nitrogenous irritants in the litter was not substantiated. Exposure of birds to wet litter (in the absence of excreta) for only 8 h/d was sufficient to develop foot pad lesions. The present results suggest that a focus on the protein content of the diet as a cause of FPD may be misplaced, but all dietary factors which increase excreta and/or litter moisture should be considered. The prevalence and severity of FPD can be reduced by maintaining the litter dry.

Keywords: foot pad dermatitis; uric acid; ammonia; litter moisture; turkeys
CHAPTER 2: Effects of macrominerals - surplus in the diet and high litter moisture on development and severity of foot pad dermatitis in growing turkeys

I.M.I. YOUSSEF\textsuperscript{1}, A. BEINEKE\textsuperscript{2}, K. ROHN\textsuperscript{3}, J. KAMPHUES\textsuperscript{1}

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

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Summary

This study was conducted to test the hypothesis that a surplus of specific dietary macrominerals which are not seldom used at levels higher than necessary may aggravate the development of foot pad dermatitis (FPD) directly (irritants in excreta) and/or indirectly (increase of litter moisture). An experiment was performed on 2-week-old female turkeys over a period of 3 weeks. The birds were divided into 4 groups, with 20 birds each, and housed on dry wood shavings. In group 1 and 2, a complete pelleted diet containing low levels of specific macrominerals (6.65 Ca, 4.43 P, 1.40 Mg, 1.12 Na, 3.16 Cl g/kg diet) was fed, while the animals in groups 3 and 4 were fed a diet with high levels of these elements (17.1 Ca, 7.73 P, 2.79 Mg, 2.32 Na, 4.58 Cl g/kg diet). Only the birds of groups 2 and 4 were additionally exposed, for only 8 h/d, to wet litter (27% DM content). The excreta were never removed from dry or wet litter, to simulate field conditions. The foot pads of all birds were examined on d 0, 7, 14 and 21 and assessed by external scoring (0 = normal skin; 7 = necrosis of foot pad). Three birds were selected from each group for histopathology of foot pads on d 0, 7 and 14. The remaining 11 birds per group were sacrificed on d 21 and their pads were assessed histopathologically. There was a markedly higher FPD severity on wet than on dry litter (score 5 vs. 1.5). The high macromineral intake did not influence foot pad scores on dry litter, but it had a negative effect on wet litter (5.50 vs. 4.50), especially at the end of the experiment. Nevertheless, this effect of macrominerals on FPD was slight in comparison to that of wet litter (1: 3), indicating that the high litter moisture is the predominant factor leading to FPD and not the high macromineral supply.

Keywords: Foot pad dermatitis; macrominerals; turkeys; severity; litter moisture
CHAPTER 3: Effects of high dietary levels of soybean meal and its constituents (potassium, oligosaccharides) on foot pad dermatitis in growing turkeys housed on dry and wet litter

I.M.I. Youssef 1, A. Beineke 2, K. Rohn 3 and J. Kamphues 1

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


Abstract:

Soybean meal (SBM) is the main protein source in diets for turkeys. High dietary levels of SBM are thought to increase the incidence of foot pad dermatitis (FPD). Therefore, this study was conducted to test potential effects of high SBM and to elucidate which constituents in SBM might be associated with the development of FPD. Two week-old female turkeys were allotted to four groups of 29 birds each, and housed on dry wood shavings in floor pens over a period of three weeks. Four different diets were fed: control, high SBM, high potassium (K) or high oligosaccharide (OS) diet. Half of the animals in each group were additionally exposed to wet litter (27% DM content) daily for only 8 h in adjacent separate boxes. The foot pads of all birds were assessed on d 0, 7, 14 and 21 for external lesions. Three birds were selected from each group on d 0, then 6 birds (3 from each litter treatment) per group on d 7 and 14 for histopathology of foot pads. The remaining birds in each group were sacrificed on d 21 and their pads evaluated histologically. The external and histological evaluation scores were conducted according to Mayne et al. (2007).

High dietary levels of SBM, potassium or oligosaccharides did not influence the severity of FPD on dry litter, but slightly increased the severity on wet litter. However, there were no histopathological differences in FPD severity between these dietary treatments within each litter form compared to the control. Nevertheless, the FPD severity was in general much higher on wet (score 6) than on dry litter (score 2). Thus, the high litter moisture appears to be one of the most important factors involved in FPD in turkeys. In addition, all nutritional factors which increase water intake and excreta or litter moisture may contribute to increased development and severity of FPD in turkeys.

Keywords: soybean meal; foot pad dermatitis; potassium; oligosaccharides; turkey feeding
Chapter 4

CHAPTER 4: Experimental study on effects of litter material and its quality on foot pad dermatitis in growing turkeys

I.M.I. Youssef 1, A. Beineke 2, K. Rohn 3 and J. Kamphues 1*

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


Abstract:
Since turkeys are in continuous contact with the litter, the potential effects of bedding materials and their quality are of major concern in the etiology of foot pad dermatitis (FPD). Two week-old female turkeys were allotted to four groups, with 29 in each, and housed either on wood shavings, lignocellulose, chopped straw or dried maize silage over a period of four weeks without artificial climatisation. The birds in all groups were fed identical commercial diets. Half of the turkeys in each group were additionally exposed for 8 h/d to corresponding wet (27% DM; by adding water) litter in adjacent separate boxes. Foot pads of the birds were examined macroscopically and histologically at the start and end of the experiment as well as at weekly intervals. Lignocellulose showed the lowest severity of FPD on dry and wet litter treatments, whereas chopped straw was associated with high FPD scores on dry treatment. Foot pad scores were similar on wood shavings and dried maize silage whether dry or wet. The DM content of litter materials was determined and the highest moisture content among dry treatments was observed in straw (about 31%) which was paralleled with FPD severity. The severity of FPD was overall much higher (>2 times) on wet than on dry litter. Exposure of the birds to wet litter for 8 h/d was sufficient to develop FPD. Lignocellulose could reduce the FPD severity, probably due to higher water binding capacity and faster release of water, while straw may increase it due to lower water evaporation. The physical form of litter either soft (lignocellulose) or with sharp edges (chopped straw) may also affect the onset of FPD. The litter moisture appears to be the dominant factor resulting in the development of FPD and should be kept lower than about 30 % to minimise the prevalence and severity of FPD in turkeys.

Keywords: Foot pad dermatitis; litter type, litter quality; turkeys; litter moisture
CHAPTER 5: Influences of increased levels of biotin, zinc or mannan oligosaccharides in the diet on foot pad dermatitis in growing turkeys housed on dry and wet litter

I.M.I. Youssef 1, A. Beineke 2, K. Rohn 3 and J. Kamphues 1*

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


Summary

Foot pad dermatitis (FPD) is very common in turkeys and it is an animal health and welfare issue affecting not only performance and walking ability, but also the carcass quality. Thus, there is a great need to find out the preventive measures against this problem. The potential roles of extra dietary biotin, Zn and mannan oligosaccharides (MOS) in preventing the development of FPD were assessed in this study which was conducted on 2 week-old female turkeys over a period of 4 weeks. The birds were allotted to four groups, with 29 each, and housed on dry wood shavings in floor pens. The turkeys were fed a control (300 µg biotin and 50 mg Zn/kg), high biotin (2000 µg/kg), high Zn (150 mg/kg) or mannan oligosaccharides (MOS; 1%) diet. Half of the turkeys in each group were additionally exposed to wet litter (27% DM) for 8 h daily in adjacent separate boxes. Foot pads of the birds were examined on d 0, 7, 14, 21 and 28 and assessed macroscopically and histopathologically for foot pad lesions. High dietary levels of biotin or Zn significantly reduced the severity of FPD on dry litter (score 1 vs. 2 in control), but not on wet litter. However, MOS did not affect the severity of foot pad lesions either on dry or wet litter. In addition, the severity was overall substantially higher on wet (about 3 times) than on dry litter. The present results suggest that adding high levels of biotin or Zn to the diet could reduce the development and severity of FPD on dry litter but without having any preventive effects on wet litter. Finally, the high litter moisture appears to be the major factor resulting in FPD. Therefore, the litter should be maintained dry to minimise the prevalence and severity of FPD in turkeys.

Keywords: foot pad dermatitis; biotin; zinc; mannan oligosaccharides; turkeys; litter moisture
7 GENERAL DISCUSSION

The cause of foot pad dermatitis (FPD) seems to be multifactorial. Many risk factors have been suggested, such as litter moisture, litter type, husbandry and nutritional factors. FPD may be accompanied with pain (Martland, 1985; Mayne et al., 2007c) which can adversely affect animal welfare and performance. Five consecutive experiments were conducted in this study to find out the most relevant causes and preventive measures against foot pad dermatitis. Effects of moisture / nitrogen metabolites in the litter and of excessive dietary macrominerals on FPD were investigated as well as influences of high dietary soybean meal and of potassium and specific oligosaccharides it contains. Furthermore, the impact of litter type and quality was assessed. Finally, effects of dietary biotin, Zn and mannan oligosaccharides as preventive measures against FPD were also tested. The present study was not performed to describe FPD as it is found in the field especially at slaughter, the primary intention was to look on the potential role of specific dietary and management factors (that vary in the field) in the development or pathogenesis of FPD.

7.1 The experimental model

In all experiments (except trial 1), half of the birds in each group/treatment were housed continuously on dry litter (24 h), whereas other turkeys were additionally exposed to wet litter (27 % DM) for only 8 h/d and then returned back to the original dry litter (16 h/d). In experiment 1, the control animals were housed continuously on dry litter, but all the birds in other groups were exposed to wet litter (27 % DM) for 8 h/d. The period of exposure to wet litter was suggested to simulate the litter condition in the field as only restricted parts of the litter is very wet - especially in the vicinity of drinkers and feeders while other areas are more or less dry. The moisture content (calculated as: 100 - DM) of wet litter was maintained at about 73% by adding water as required. This moisture content was similar to that of wet litter used in previous experiments (Martland, 1984; Mayne et al., 2007c), but the birds in these previous studies were housed continuously on wet litter which is not consistent with the housing condition in the field. Nevertheless, the moisture content of wet litter used in this study was lower than that of “normal” excreta (about 82%) of fattening turkeys which was determined by Radko (2007). In addition, the moisture content of the litter in turkey (16 week-old) barn in the field (Research farm; University of Veterinary Medicine, Hannover) was found in own investigations (Youssef et al., 2009; unpublished) to be 73.5% around the
drinkers, 40.1% near to the feeders and 24.5% in the corners and middle of the barn. Moreover, the litter moisture in broiler (29 day-old) barn was noticed to be 70% in the vicinity of drinkers and 27.1% in the yard.

7.2 Critique of the method

- Birds and housing

Birds:
This study was conducted on one turkey line only (BUT – Big 6) which is the most used strain in the turkey fattening. This study was carried out on female turkeys only. Male turkeys are heavier in body weight than females and consequently may have a higher prevalence of FPD. In the literature, there are contradictory results on the incidence of FPD in males and females. Our investigations were focussed on the process of initial developmental steps of foot pad alteration, but not on the occurrence and intensity of FPD at slaughtering.

All experiments were performed on turkeys at the age of 2 week and continued up to the end of 5th or 6th week of the life. The birds were fed on commercial diets during the first 2 weeks of life before the beginning of the treatments, then switched to experimental diets or continued the feeding on commercial diets during the experimental period.

The present study was conducted on only young turkeys (d 15 to d 36/42). Thus, an abundance of litter and lower excreta amounts in the litter are expected at the early stages of life compared to those found at the end of the fattening period. Furthermore, the body weight of these young birds is lower than that of turkeys at the end of the fattening, resulting in a lower pressure on foot pads. However, some studies observed that FPD develops at an early age and its severity increased from 2 to 6 weeks and remained relatively constant thereafter (Mayne et al. 2006b, 2007b). Thus, the period of our investigations is the recommended one to evaluate the initial processes of the onset of FPD. Moreover, Mayne et al. (2007c) did their experiments for only one week on turkeys of similar age to those used in this study. It is also possible that older birds may be less susceptible to litter moisture and more or less liable to FPD from other causes (Mayne et al., 2007c).

Housing:
The experiments were conducted under standardised experimental conditions and not in the field. During the first 2 weeks of life, the birds were housed on clean dry litter by daily
replacing the top layers of litter including excreta with fresh dry litter. At the start of the experiments, the birds were reared on dry litter, but half of birds in each treatment were exposed to wet litter for only 8 h/d. The excreta were not removed from the litter either dry or wet during the treatments with exception of experiment 1 (water with or without \( \text{NH}_4 \text{Cl/uric acid} \) in the litter) as the litter was kept free from the excreta. The birds were housed on wood shavings in all experiments except experiment 4, in which the turkeys were housed on different bedding materials (wood shavings, lignocellulose, chopped straw, dried maize silage) which are also used in the field.

The moisture content of wet litter was very high which could mask the effects of other factors. In this study, there was no ventilation (artificial air movement) which differs to the condition in the field, resulted in absence of the conventional drying of the litter. Nevertheless, the absence of ventilation was helpful to compare between the treatments as any differences in the litter moisture should be related to the litter per se or dietary treatments.

- **FPD scores**

The assessment of the foot pad lesions was done personally using the external scoring system of Mayne et al. (2007c). However, these external FPD scores were confirmed by the histopathological examination and it was found that results of histological foot scores were highly consistent with those of external scores (\( r = 0.90 \)). The used scoring system characterised by including all developmental stages of FPD and it consists of 8 categories ranged from 0 to 7, but it is difficult to compare the obtained results with those evaluated by other scoring systems. Moreover, the score of Mayne et al. (2007c) is advised to be used in the experimental research to follow the developmental changes of FPD in the individual animals, but it is not easy to be applied at slaughtering. Thus, Hocking et al. (2008) proposed a standard scoring system (range from score 0 to 4) for use in turkey processing plants. Foot pad lesions may be accompanied with pain (Martland, 1984, 1985; Mayne et al., 2007c). Moreover, Mayne et al. (2007c) found that the turkeys (23 day-old) had an extreme inflammatory response and were reluctant to move after only 2 d from continuous exposure to wet litter. The external FPD scores of these birds varied around 6.70, indicating that there is a tendency of birds to show signs of inflammation from score 6 onwards. In this study, the external FPD scores on dry litter ranged from 0 to 4 while these on wet litter varied from 1 to 7. Regardless of the experimental treatments, the incidence of severe external scores (6.0, 6.5 and 7.0) at the end of the experiments was 13.7%, 3.59% and 1.43%, respectively of the total
number of birds housed on wet litter only (n = 139). However, no signs of discomfort or pain during moving activity were observed in this study. Moreover, the incidence of superficial lesions decreased in turkeys after 14 weeks, while more severe ulceration increased (Platt et al., 2001), indicating that the lesions extend in the depth in older birds and consequently may be become painful. The external scores on wet litter after one week from the start of the treatments were more evident and ranged from 1 to 5 and slightly increased until reached 2 to 7 at the end of the experiments. There was no need to extend the housing of birds on wet litter until the end of the fattening as score 5 was observed within one week from application of wet litter. This indicates that the proposed period of investigations is sufficient for inducing the most relevant alterations at the surface of foot pads as well as there is no need for adhering fecal material on the pad surface. Moreover, in experiments of Mayne et al. (2007c), the turkeys were housed continuously on wet litter for only one week.

7.3 New findings in the study

Experiment 1:
Pure water alone in the litter, without the presence of excreta, induced foot pad lesions, indicating that the presence of excreta is not necessary for the development of FPD. Exposure of the birds to wet litter (litter experimentally moistened by distilled water) for only 8 h/d was sufficient to induce foot pad lesions. Moreover, the repeated exposure of the birds to dry clean litter (16 h/d) did not retard the development of the lesions induced by wet clean litter and did not result in healing of the lesions. Furthermore, high levels of uric acid and ammonia in the wet litter did not aggravate the effect of pure water on FPD. This suggests that high protein levels in the diet are not the primary cause of FPD.

Experiment 2:
Surplus of macrominerals in the diet did not harm the foot pad as long the litter was dry, but it aggravated the effect of wet litter in developing FPD. This could be due to excessive excretion of these elements in the dropping along with high litter moisture, resulting in irritation of the foot pads. However, there was no marked irritation of the skin of foot pads due to high levels of macrominerals as it was discussed repeatedly in the literature. Moreover, the concentrations of these minerals are also tolerated by the mucosa in the hindgut, thus it is not surprising to find these findings. Nevertheless, the macrominerals surplus showed a tendency to increase water intake and thereby the moisture content of the excreta. This effect
was not distinctly developed, presumably due to a lower K content in the high macromineral diet than normally present in commercial diet (6.0 vs. 10 g/kg) and the level of Na (2 g/kg) was not sufficient to stimulate the water intake markedly. Moreover, the early stages of fattening could be associated with a higher amount of litter and lower excreta content of the litter compared to the final stages of fattening period. Thus, the marked effects of macrominerals on the development of FPD should be more obvious at the end of the turkey fattening. It is possible that water softens the epithelium of foot pads which results in the skin being more prone to damage by other factors in the excreta or litter (Mayne et al., 2007c). Moreover, the wet litter showed more alkaline pH values (7.0 – 8.0) compared to dry litter ($\leq$ 7.0). High proportions of excreta in the litter appear to be risky due to it bringing water in the litter, whereas the other compounds of excreta seem to be not necessary for developing FPD.

Experiment 3:
High levels of SBM were found to increase the development and severity of FPD on wet litter, which support the results of earlier research (Jensen et al., 1970), but there are no available researches up to now on the mechanisms by which the SBM induces this effect. However, the present study explained this impact of SBM as it contains high levels of both K and oligosaccharides which act additively in production of wet and sticky excreta. The birds fed the high SBM diet were observed to have a markedly higher water intake than the others (SBM > K > OS > control) as well as their excreta appear visually wet or sticky. Adhering of these excreta to the feet of birds increased the severity of FPD. The present results suggest that the incidence of FPD is not a consequence of soybean protein, but it should be associated to further substances which are normally found in SBM such as potassium and oligosaccharides. The potassium contents in soybean meal, corn gluten, peas, peas protein and meat meal are about 24.4, 3.89, 11.4, 3.50 and 6.50 g/kg DM, respectively, thus the predisposing role of SBM in the development of FPD is quite obvious.

Experiment 4:
Wood shavings and cereal straw are the common litter materials used for poultry. In addition, alternative bedding materials have recently become available from plant sources such as lignocellulose and dried maize silage but their suitability for preventing FPD in turkeys has not been assessed. Contrary to conditions applied in the field, this experiment was done under experimental standardized conditions without application of artificial ventilation along with feeding the birds on identical diets, resulting in the effect of bedding materials on FPD was
due to the litter per se or to the experimental adding of water. The DM content of wet litter treatments was always similar (about 27%), but that of dry treatments was different. The ability of the litter to bind and/or release water is of special interest in the etiologic standpoint of FPD. Lignocellulose had the capacity to absorb well and release quickly the water due to its fine structure (large surface of particles). Chopped straw showed a similar water binding capacity to lignocellulose, but it had a lower water evaporation as well as a tendency to caking formation resulting in a higher moisture retention which moistens the feet of birds. The physical structure of the litter either soft (lignocellulose) or hard (straw) could also play a role in the development of FPD. Moreover, the dried maize silage could not persist in the experiment when it became wet due to growth of moulds. Nevertheless, wood shavings litter is suitable for birds as it had a lower FPD severity than chopped straw. The FPD severity paralleled the litter moisture and began to increase when the litter contained more than about 30% moisture, independent of the type of litter.

**Experiment 5:**
The literature showed controversial results concerning the effects of biotin and zinc on FPD. Some studies reported that biotin can reduce the incidence and severity of FPD (Buda 2000a,b; Platt et al., 2004, Hafez et al., 2004,) but other researches found no effect of biotin (Jensen et al., 1970; Mayne et al., 2007b). The same situation for Zn was observed by Hess et al. (2001) who found a beneficial effect of Zn complexes on FPD in female broilers, but not in males raised under cold conditions. Thus, the results of our study can explain these contradictory findings. In this study, the potential effects of biotin and Zn depend mostly on the DM content of litter. On dry litter (without adding water), higher supply of biotin (2000 µg/kg) or Zn (150 mg/kg) significantly reduced the severity of FPD, despite the requirements were satisfied in all groups (300 µg biotin, 50 mg Zn/kg). This indicates that either the requirements of both biotin and Zn were inadequate to prevent FPD or these nutrients had supra-nutritional effects (pharmaceutical like effect) that can remedy these lesions. It is known that excess of dietary biotin or Zn had "drug like" effects on the health of horses’ hoofs and pigs’ claws. On wet litter, there were no positive effects of biotin or Zn on FPD. These results could explain the findings of previous studies (Jensen et al., 1970; Hess et al., 2001; Mayne et al., 2007b). In addition, the lesions on wet litter could be accompanied with secondary infections which retard the effects of biotin and Zn. The tested dietary concentration of Zn (150 mg/kg) is the maximum allowed level by European Union
legislation (Regulation (EC) No 1831/2003), but biotin can be used without upper limits. In regard to MOS, it tends to reduce the severity of FPD but its effect was not significant.

7.4 Development and severity of foot pad dermatitis

- Effect of dietary factors

High dietary protein level has been assumed to increase the incidence and severity of FPD in broilers (Nagaraj et al., 2006 and 2007b), which may be due to increased nitrogen excretion and NH₃ formation in the litter. In this study, the pure water alone in the litter was the major causative agent of FPD, but the main protein metabolites (uric acid, NH₃) in the wet litter had no significantly negative effects. This indicates that a focus on the protein content of the diet as a causative agent of FPD may be misplaced as also reported in previous experiments (Mayne et al., 2007c; Nagaraj et al., 2007c). Nevertheless, some researches suggested that the higher concentration of ammonia within the litter is a causative agent of FPD (Nairn and Watson, 1972; Schmidt and Lüders, 1976; Martland, 1984; Lerner, 1996; Alchalabi, 2002). However, other studies reported that volatile ammonia in the litter was not the cause of FPD (Mayne et al., 2006a and 2007c; Nagaraj et al., 2007a). Additionally, Nagaraj et al. (2007c) found that a high dietary protein level did not affect the prevalence of FPD in broilers, despite the increased excretion of nitrogen in the litter and higher release of NH₃.

Nutritional factors that increase water intake and excretion may contribute to FPD. Mineral elements excreted via urine (Na, K, Cl and Mg) forced the water consumption more than those excreted through faeces (as calcium), resulting in wet litter conditions with subsequent a higher prevalence of FPD (Smith et al., 2000; Eichner et al., 2007; Bilgili, 2009). Especially high potassium and sodium levels in the diets may result in excessive water intake and consequently increasing the moisture content in the litter (James and Wheeler, 1949; Tucker and Walker, 1992), whereas an increase in chloride did not have the same effect (Murakami et al., 2000 and 2001). Moreover, a less severe FPD in turkeys was observed when NaCl content in the diet was decreased (Harms and Simpson, 1982). Certain feedstuffs, especially soybean meal, contain high amounts of potassium which increases the moisture content in the excreta due to a higher water intake (Jensen et al., 1970; Vieira and Lima, 2005; Eichner et al., 2007). In the present study, at a low dietary level of potassium (about 6 g/kg) and a sodium content of about 2 g/kg, the high macromineral diet did not influence water intake and excreta or litter moisture. Furthermore, it was observed that a higher water intake occurred in birds fed diets
contained 8-9 g K and 2 g Na/kg compared to diets included about 7 g K and 2 g Na per kg (Vieira and Lima, 2005; Eichner et al., 2007). It seems that excreta moisture is highly correlated to dietary K content than to Na as noticed by Kamphues et al. (2007; unpublished data) who observed an increase in the moisture content of excreta in broilers fed diets contained 7.83 g K and 1.42 g Na/kg compared to 5.90/1.58 or 3.67/1.62 g/kg for K/Na contents (Figure 1).

![Graph](image)

Fig. 1: Effect of different potassium and sodium contents in experimental diets on DM content of the excreta in broilers (Kamphues et al., 2007; unpublished).

Murakami et al. (2000 and 2001) reported that a level of 2.50 g Na/kg in the diet could result in a significant increase of water intake and litter moisture. In the present study, a potassium content of about 12 g/kg increased the water intake and excreta or litter moisture. SBM contains, in addition to K, indigestible carbohydrates (such as oligosaccharides) which have been implicated in causing “sticky droppings” and wet litter problems (Leske et al., 1991; Bedford, 1995; Boiling and Firman, 1997). These sticky excreta may result in a higher incidence of FPD (Jensen, 1969; Jensen et al., 1970; Clark et al., 2002).

The present results showed that high amounts of tested macrominerals, SBM, K or oligosaccharides in the diets slightly increased the severity of FPD on wet litter only, but had no negative effects as long as the litter was dry. However, the effects of these dietary factors were very slight in comparison to the effect of wet litter per se. It is possible that water softens the epidermis which results in the skin being more susceptible to contact dermatitis (Mayne et al., 2007c). In addition, prolonged contact of feet with excreta and high litter moisture may contribute to a higher prevalence of FPD as reported in previous research.
(Abbott et al., 1969; Jensen et al., 1970; Harms et al., 1977). Also, FPD is thought to be caused by a combination of wet litter and chemical substances in the litter or unidentified irritants in excreta (Nairn and Watson, 1972; Greene et al., 1985; Martland, 1985; Ekstrand et al., 1998). The findings of our study support the results of earlier research (Steenfeldt et al., 2005), in which no effect of different levels of calcium and phosphorus on the incidence and severity of FPD in broilers was observed. Moreover, the obtained results suggest that the impact of high SBM proportions on FPD could be related to its content of both K (produce excessive excreta moisture) and oligosaccharides (with potential to produce viscous/sticky excreta).

- Effect of the litter

Poor litter quality is clearly associated with the incidence of foot pad lesions (Geraedts, 1983; Martland, 1984; Martrenchar et al., 2002; Mayne et al., 2007c). In this study, the high litter moisture (for only 8 h/d) had the ability to potentiate the prevalence and severity of FPD. Obviously, the severity of foot pad lesions in all experiments on wet litter was markedly higher (> 2 times) compared to dry litter. This indicates that litter moisture is the major factor resulting in the development of FPD. Similar results were observed in previous experiments (Martland, 1984 and 1985; Mayne et al., 2007c), but after continuous exposure of birds to wet litter. In this study, the exposure to wet litter for only 8 h/d was sufficient to provoke FPD. This implies that all factors which affect the litter moisture either directly or indirectly are of interest. Moreover, the prevalence of FPD paralleled high litter moisture as also reported by Bilgili et al. (2009). The severity of foot pad dermatitis began to increase when the litter contained more than about 30% moisture. Many studies also reported that the litter moisture exceeding 30 - 35% often resulting in a higher prevalence of FPD (Martland, 1984 and 1985; Jodas and Hafez, 2000; Mayne et al., 2006a; Glebocka, 2008). Wet litter was associated with a higher pH and a higher ammonia formation compared to dry litter as also detected by previous researchers (Lerner, 1996; Alchalabi, 2002; Nagaraj et al., 2007a). In contrast, Martland (1985) observed a lower pH in the wet litter which had a higher incidence of FPD than in the dry litter.

The type of litter had a marked effect on the prevalence of FPD (Bilgili et al., 2009). Of all tested bedding materials, lignocellulose showed the lowest severity of FPD either on dry or wet litter. This could be due to high absorbing capacity as well as a quick release of water.
These findings are consistent with the results of Berk (2007; 2009a). In dry litter treatments (without adding water), chopped straw was associated with higher FPD scores which probably due to lower water evaporation and caking formation (Bilgili et al., 2009), resulting in a higher moisture content in this litter. Also, many studies reported that chopped straw was associated with the highest FPD severity scores in broilers (Berk, 2009b; Baere de et al., 2009; Bilgili et al., 2009) and in turkeys (Ekstrand and Algers, 1997). Based on these results, the ability of litter to bind and/or quickly release water seems to be a very important factor in the etiology of FPD. The physical structure of the litter either soft (lignocellulose) or with sharp edges (chopped straw) may also contribute in lowering or increasing the severity of FPD. The FPD scores on wood shavings and dried maize silage were similar on dry treatments. On wet litter treatments, there was no difference in foot pad scores between wood shavings, chopped straw or wet maize silage (histologically only). The external FPD scores on artificially wetted maize silage were decreased which is probably due to obligatory change of this litter per week (as a result of mould growth) or due to low pH and lactic acid content (formed during ensiling) which might have bactericide effects (Bosse and Meyer, 2007; Wilms-Schulze Kump, 2007).

7.5 External and histological foot pad scores

Exposure of turkeys to wet litter for only 8 h per day was sufficient to induce foot pad lesions. Mayne et al. (2007c) found that fully developed lesions were induced within 2 to 4 days after continuous housing of the birds on wet litter. The foot pad scores were always much higher on wet than on dry litter in all trials, regardless the effects of litter type/constituents and of dietary factors (Table 1). In addition, the FPD scores were increased on wet dirty litter compared to wet clean litter, indicating that contact with excreta can aggravate the effect of wet litter. Nevertheless, Mayne et al. (2007c) found that wet litter alone causes similarly severe lesions as wet dirty litter. Furthermore, the scores were also higher on dry dirty litter than on dry clean one, suggesting that the excreta may play a role in the onset of FPD especially its moisture content. The correlation between external and histopathological scores was high (about 0.90). However, Mayne et al. (2006b and 2007b) reported that the relationship between external and histological FPD scores was relatively weak ($r = 0.56$). Moreover, the external foot pad scores increased in the severity over time in all experiments, whilst the histological scores increased at the beginning of the trials and remained relatively constant thereafter. These results supposed that the necrotic area increases in the size over
Discussion

time, but did not deepen to increase the intensity of foot pad lesions. Accordingly, the necrotic lesions may increase in the size in young animals and in the depth (which may become painful) in older animals. Mayne et al. (2006b and 2007b) found that the external foot scores in turkeys increased in severity from 2 to 6 weeks and were similar from 6 to 14 weeks of age, suggesting that the severity of FPD increased rapidly at a relatively early age and remained relatively constant afterwards. Mild lesions have been observed on dry litter (redness, erosions or small black necrotic areas), whereas wet litter was associated with severe lesions in the form of necrosis or ulcer as also found in previous studies (Martland; 1984 and 1985; Mayne et al., 2007c). Histopathologically, FPD is associated with massive increases in heterophils and macrophages and the loss of surface keratin (Mayne et al., 2006b). These cellular changes of FPD induced by wet litter were shown to be an inflammatory response and not an allergic reaction (Mayne et al., 2007a).

Table 1. Averages of external/histopathological FPD scores on dry and wet litter (8 h/d) at the end of different experiments, independent of litter type/constituents and dietary contents

<table>
<thead>
<tr>
<th>FPD score</th>
<th>Litter</th>
<th>Exp. 1 (water, NH₄Cl, uric acid in litter)</th>
<th>Exp. 2 (macrominerals)</th>
<th>Exp. 3 (SBM/K/OS)</th>
<th>Exp. 4 (litter material)</th>
<th>Exp. 5 (biotin/Zn/MOS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>external</td>
<td>excreta</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>dry</td>
<td>0.82 abA</td>
<td>1.53 bcdA</td>
<td>1.80 baA</td>
<td>1.30 cbA</td>
<td>1.36 cdA</td>
</tr>
<tr>
<td></td>
<td>wet</td>
<td>3.36 abB</td>
<td>4.95 bB</td>
<td>5.43 bdB</td>
<td>4.28 cbB</td>
<td>5.59 dBb</td>
</tr>
<tr>
<td>histologic</td>
<td>dry</td>
<td>1.41 abA</td>
<td>2.37 abAB</td>
<td>1.88 abAB</td>
<td>1.91 abAB</td>
<td>2.16 bA</td>
</tr>
<tr>
<td></td>
<td>wet</td>
<td>3.75 abB</td>
<td>5.39 bB</td>
<td>5.89 cB</td>
<td>5.25 bB</td>
<td>5.81 cbB</td>
</tr>
</tbody>
</table>

Means with different small letters indicate significant differences between the experiments, whilst those with capital letters indicate differences between dry and wet litter within external or histological scores (P < 0.05).

7.6 Prevention of foot pad lesions

Generally, the development and severity of foot pad lesions were lower on dry litter. Similar results were observed in some research (Martland, 1984 and 1985; Mayne et al., 2007c). High levels of dietary biotin or Zn could help to reduce the incidence of FPD. However, the effects of these nutrients appear to depend largely on DM content of the litter. As observed in this study, the inclusion of high levels of biotin or Zn reduced the severity of FPD on dry litter, but not on wet litter. It was reported that supplementation of biotin decreased the severity of FPD in turkey poults raised on dry litter, but not in poults maintained on wet litter (Harms and
Simpson, 1977; Mayne, 2005). Many studies found a positive effect of biotin supplementation on the prevalence of FPD (Buda, 2000a and b; Clark et al., 2002; Hafez et al., 2004; Platt et al., 2004). In contrast, other authors observed that high dietary biotin levels did not prevent the occurrence of FPD (Jensen et al., 1970; Mayne et al., 2007b). Concerning the effect of Zn, some studies reported that dietary Zn reduced the incidence and severity of foot pad lesions (Hess et al., 2001; Bilgili, 2009). However, Hess et al. (2001) found also no effect of Zn on the severity of FPD in birds reared in cool weather (4 – 15 ºC), indicating that the impact of Zn appeared to vary with the environmental conditions (which may affect the litter moisture). High concentrations of biotin or Zn failed to reduce the severity of FPD on wet litter. It is possible that the effects of these additives on healing of the lesions were retarded by the vigorous effect of high litter moisture. Furthermore, the foot pad lesions on wet litter could be complicated by secondary bacterial contamination which inhibits the healing process induced by biotin or Zn. The lesions on wet litter were more severe (necrosis or ulcer) and accompanied with inflammatory reaction as indicated histologically by infiltration of inflammatory cells. This inflammatory response is probably related to bacterial infection. However, the foot pad lesions on dry litter were mild and could be not associated with bacterial invasion, and thus the lesions can respond to biotin or Zn supplementation.

7.7 Effect of foot pad dermatitis on animal performance

In trials 1 to 4, no negative effects of wet litter (which had a higher FPD severity) on the weight of turkeys were observed. Yet, at the end of the trial 5, the body weight was lower on wet litter than on dry litter when the effects of dietary treatments were neglected. As the feed intake of birds exposed to wet litter was not affected by the severity of FPD, the decrease in their weight could be due to the turkeys lose more energy by conductance through the wet litter and by evaporative cooling (Mayne et al., 2007c). Grosse Liesner (2007) found also no adverse effects of FPD on body weight of turkeys at slaughter. However, some studies observed a lower body weight in birds suffering from FPD, probably due to a painful response which decreases feed and water intake as suggested by experimental results (Martland, 1984 and 1985; Mayne et al., 2007c).

7.8 Conclusions

Exposure of birds to wet litter (with or without excreta) for only 8 h/d was sufficient to induce a higher FPD severity. Thus, the litter should be kept dry to minimize the prevalence of foot
pad lesions. Also, special attention to the litter around the drinkers should be given to maintain it dry as possible. Any surplus of macrominerals (especially Mg, Na, K) in the diet should be avoided as it could stimulate water intake and excretion and consequently predisposing the birds to FPD. The high dietary macrominerals might also irritate the foot pads secondary to high litter moisture. High dietary levels of SBM are associated with higher FPD scores. The protein portion of SBM has no role in the occurrence of FPD, but the higher K and oligosaccharide contents are the potential factors in SBM. These results are of special interest in feed industry to reduce proportions of SBM and substituting it with other protein-rich ingredients which contain lower amounts of K and indigestible carbohydrates, such as peas, poultry by-product or corn gluten meal. Lignocellulose could be used as an alternative litter material to reduce FPD. This is due to its physical and structural properties (high water binding capacity and rapid release of water as well as soft structure). However, chopped straw resulted in a higher FPD severity due to higher water retention in this litter resulting from lower water evaporation and caking formation. In practice, pelleted straw litter was found to reduce the incidence of FPD compared to chopped or long straw. A special caution is needed in using dried maize silage as a bedding material due to growth of moulds when it becomes wet, which could result in fungal infections of birds (especially via inhalation). Furthermore, the FPD severity began to increase when the moisture content of the litter exceeds about 30%. Adding zinc (cheap trace element) to reach a total of 150 mg Zn/kg diet could reduce the development and severity of FPD. A biotin supplement (2000 µg/kg) had comparable effects to Zn, but its use depends on the price. When the litter was wet, biotin and Zn failed to prevent the prevalence of FPD. In this condition, there is no doubt that the concentration of biotin or Zn in the diet is inadequate to prevent FPD.

Finally, there are still many open questions, whether wet litter persist in increasing the severity of FPD when the period of exposure was reduced lower than 8 h/d and what is the critical litter moisture which increases the incidence of FPD. In addition, whether the older birds are susceptible to litter moisture and the higher body weight increases the pressure on foot pads. Moreover, further trials are needed to evaluate the influence of gastrointestinal infections as well as to assess the effect of secondary bacterial invasions in wet litter on FPD. Additional research is needed to investigate the impact of feed enzymes on SBM to enhance feed utilization and also to test substitution of SBM with other protein sources (like protein
meal from swine slaughtering) which are lower in potassium and oligosaccharides contents, and to evaluate the effects of combining biotin and Zn in reducing foot pad lesions.

A higher prevalence of FPD is a challenge that earns the attention of different people. Thus, many efforts are being made to minimise the incidence of this problem: optimising diet composition, use of best litter materials, adding fresh dry litter, and intensive control of technical equipments (feeder and drinker lines / ventilation / heating) to maintain the litter as dry as possible. Whenever the litter becomes wet – independent of the primary reason – the risk of FPD is increased as demonstrated repeatedly in the present study.
8 SUMMARY

Ibrahim Mohamed Ibrahim Youssef:
Experimental studies on effects of diet composition and litter quality on development and severity of foot pad dermatitis in growing turkeys

Foot pad dermatitis (FPD) is very common in turkey flocks and it is an animal welfare issue. This study was conducted to find out the most relevant causes and preventive measures of FPD. Effects of water alone and/or protein metabolites (uric acid, NH₃) in the litter and of excessive dietary macrominerals on FPD were investigated as well as influences of high dietary soybean meal (SBM) in comparison to high potassium (K) and oligosaccharides (OS) contents. Also, the impact of litter type and quality (especially moisture content) was assessed. Effects of specific dietary supplements [biotin, Zn, mannan oligosaccharides (MOS)] as FPD- preventive measures were also tested. Each investigated factor was evaluated, in growing turkeys between 2nd and 6th week of life, under the influences of dry and wet litter (~ 73% moisture; achieved by adding water) concurrently.

Material and methods:
Five consecutive experiments were conducted on 2 week-old female turkeys (BUT-Big 6) for a period of 3 or 4 weeks. In each experiment, the birds were divided into 4 groups with 20 - 29 animals each.

Experiment 1: All birds were housed in floor pens on dry clean wood shavings litter which was replaced daily with fresh dry one to maintain the litter clean and dry. The control birds were kept continuously on this litter throughout the experiment (3 weeks), whereas the other animals were additionally exposed (for 8 h/d) to experimentally treated wet wood shavings in adjacent separate boxes. This wet litter contained water alone or enriched with NH₄Cl or uric acid. Ammonium chloride and uric acid were added via water to the litter to achieve the concentrations of ammonia and uric acid in the litter as found in fresh excreta of turkeys (about 0.50g ammonia and 20g uric acid/kg). The wet litter was kept clean by removal of the excreta twice daily and change of the litter twice a week.

Experiment 2: Birds were allotted to 4 groups; two groups were fed a control diet containing low levels of specific macrominerals (6.65 Ca, 4.43 P, 1.40 Mg, 1.12 Na, 3.16 Cl g/kg diet), whereas the other two groups were fed an experimental diet containing high levels of these elements (17.1 Ca, 7.73 P, 2.79 Mg, 2.32 Na, 4.58 Cl g/kg diet). The birds were housed in
floor pens on dry wood shavings, but half of the turkeys in either control or experimental dietary treatment were additionally exposed to wet litter for 8 h daily throughout the experiment (3 weeks).

**Experiment 3:** Birds were fed a control, high SBM, high potassium (K) or high oligosaccharides (OS) diet for 3 weeks. The high SBM diet contained high amounts of SBM (about 44%). The K or OS diet was designed to have the same contents of potassium and oligosaccharides as the SBM diet, respectively. Potassium bicarbonate was added to the K diet to achieve a potassium level nearly identical to the SBM diet (about 12g K/kg). A commercial soybean oligosaccharide product was used to increase the stachyose and raffinose content of the OS diet to be the same as in the high SBM diet (15g stachyose + raffinose /kg). During the experiment, the birds were reared on dry wood shavings, but half of the animals in each group were additionally exposed to wet litter for 8 h daily in adjacent separate boxes.

**Experiment 4:** Birds were housed on different litter materials: wood shaving, lignocellulose (SoftCell®), chopped straw (Strohfix®) or dried maize silage. Half of the birds in each litter treatment were additionally exposed to corresponding wet litter for 8 h/d throughout the experiment (4 weeks).

**Experiment 5:** Birds were housed on dry wood shavings. The control group was fed a diet containing the required amounts of biotin (300 µg/kg) and Zn (50 mg/kg). In group 2, a diet with high biotin (about 2000 µg/kg) was fed, while in group 3 a high Zn diet (150 mg/kg) was offered. The animals in group 4 were fed a diet containing mannan oligosaccharides (Bio-Mos®) at the rate of 1% of the diet. Half of the turkeys per group were additionally exposed to wet litter for 8 h/d during the experiment (4 weeks).

In all trials, the wet litter was maintained at about 73% moisture (± 27% DM) content by adding water as required (every 2 or 3 days). The foot pads of all birds in each experiment were examined at the start and end of the experiment as well as at weekly intervals, and assessed macroscopically and histopathologically according to scores (0 to 7) of Mayne et al. (2007c). The qualities of litter (DM, pH, NH₃, ammonia and uric acid content) and of excreta (DM, pH and macromineral content) were evaluated based on the experimental treatments. Levels of macrominerals, biotin and Zn were measured in blood of turkeys. Performance of animals (weight gain, feed and water intake, feed conversion rate) was also recorded.
Results:
In all trials, no mortality was found between the birds. Furthermore, the animals showed a good performance, normal feed and water intake, and did not suffer from any infectious diseases during the treatments.

1. In all experiments, the severity of FPD on wet litter was markedly higher (> 2 times) than on dry one. Exposure of birds to the wet litter (~ 73% moisture) for only 8 h was sufficient to cause FPD.

2. Pure water alone in the clean litter (free from excreta) was associated with increased development and severity of FPD. Presence of ammonia or uric acid in the wet litter did not significantly aggravate the effect of water (score 3.45 vs. 3.18). This indicates that the high litter moisture solely can cause FPD, but presence of the excreta increased in general the foot pad scores.

3. The surplus of macrominerals (Ca, P, Mg, Na, Cl) in the diet had no effect on the severity of foot pad scores on dry litter (score 1.55 vs. 1.50 for control), but slightly increased the severity on wet litter (score 5.45 vs. 4.45).

4. High dietary levels of SBM, potassium or oligosaccharides did not increase the severity of FPD on dry litter, but slightly increased the severity on wet litter. The impacts of these dietary factors were due to increased water intake. Furthermore, the effect of high SBM level on FPD was related to its higher content of both K (producing wet excreta) and OS (resulting in viscous/sticky excreta).

5. Of all tested bedding materials, lignocellulose reduced the FPD severity, independent of litter moisture content, whilst dry straw treatment was associated with higher FPD scores. Moreover, the FPD severity paralleled high litter moisture, which began to increase when the litter contained more than about 30% moisture.

6. Supplementation of the diet with biotin (2000 µg/kg) or zinc (150 mg/kg) reduced the severity of FPD on dry litter (score 1.0 vs. 2.0 for control), but without preventive effects on wet litter (5.54 vs. 5.79). Also, MOS tend to reduce the foot pad scores on dry litter only (1.50 vs. 2.0).

Conclusions
The results indicate that a focus on the high dietary protein as a cause of FPD may be misplaced except in so far as it increases litter moisture. However, high amounts of macrominerals in the diet had just slight effects on foot pads when the animals were exposed
Summary

to wet litter. Also, high dietary levels of SBM, K and OS slightly increased the FPD severity, but only on wet litter. Presumably, water softens the epidermis which results in the skin being more prone to contact dermatitis. Furthermore, lignocellulose as an alternative litter material could reduce the incidence of FPD, but chopped straw may increase it. High dietary levels of biotin or Zn might be able to lower the severity of FPD, but only on dry litter and not on wet litter. Moreover, the severity of FPD was always much higher on wet than on dry litter, indicating that the high litter moisture is the dominant factor resulting in the development of FPD. Exposure of animals to wet litter for 8 h/d was sufficient to cause FPD. Additionally, all dietary factors which increase water intake and excreta or litter moisture may contribute to FPD. Therefore, control of litter moisture (through optimum diet composition and keeping the litter dry) is likely to be highly effective in diminishing the prevalence and severity of FPD in commercial turkey flocks. The present results suggest that the litter moisture should be maintained lower than 30 – 35 % to reduce the development and severity of foot pad lesions.
ZUSAMMENFASSUNG

Ibrahim Mohamed Ibrahim Youssef:

Experimentelle Untersuchungen zum Einfluss der Futterzusammensetzung und Einstreuqualität auf die Entwicklung und den Schweregrad der Fußballenentzündung bei jungen Mastputen


Material und Methoden:


Versuch 1: Die Puten der Kontrollgruppe wurden während der dreiwöchigen Untersuchung in Bodenhaltung auf trockenen, sauberen Hobelspänen gehalten, d. h. die Einstreu wurde täglich erneuert. Die Tiere der Versuchsgruppen wurden hingegen bei ansonsten gleichem Versuchsaufbau täglich für acht Stunden einer nassen Einstreu (∈ 73% Feuchte) ausgesetzt, der entweder nur Wasser bzw. zusätzlich Harnsäure oder Ammoniumchlorid zugesetzt war (Konzentrationen wie in frischen Exkrementen: 0,50g Ammoniak bzw. 20g Harnsäure/kg). Die Exkremente wurden zweimal täglich aus der nassen Einstreu entfernt, zweimal wöchentlich erfolgte ein kompletter Einstreuwechsel.

Versuch 2: Die Puten der Kontrollgruppe erhielten ein Futter, das nur dem Bedarf entsprechend Mengenelemente enthielt (6,65 Ca; 4,43 P; 1,40 Mg; 1,12 Na; 3,16 Cl [g/ kg
Zusammenfassung

Futter); die Tiere der Versuchsgruppen bekamen hingegen ein Futter mit etwa der doppelten Konzentration dieser Elemente (17,1 Ca; 7,73 P; 2,79 Mg; 2,32 Na; 4,58 Cl [g/kg Futter]). Alle Küken wurden in Bodenhaltung auf trockenen Hobelspänen gehalten; jeweils die Hälfte der Individuen war während des dreiwöchigen Versuchs außerdem täglich acht Stunden einer nassen Einstreu (~ 73 % Feuchte) ausgesetzt.

**Versuch 3:** Die Tiere erhielten über drei Wochen entweder das Kontrollfutter oder ein Mischfutter mit hohem Anteil an Sojaextraktionsschrot (SES) oder hohen Gehalten an Kalium (K) bzw. Oligosacchariden (OS). Das Mischfutter mit hohem Sojaschrotanteil enthielt ca. 44 Prozent SES, das der übrigen Gruppen entsprechende Gehalte an Kalium (Zusatz als K-Bikarbonat; 12 g K/kg) oder Oligosacchariden (15 g Stachyose + Raffinose/kg). Während dieser Versuchszeit wurden alle Tiere auf trockener Einstreu aus Hobelspänen gehalten, wobei wiederum jeweils die Hälfte einer jeden Gruppe täglich acht Stunden einer nassen Einstreu (Feuchte = ~ 73 %) ausgesetzt war.

**Versuch 4:** Hier kamen verschiedene praxisübliche Materialien zum Einsatz, nämlich Hobelspäne, Lignozellulose (SoftCell®), Häckselstroh (Strohfix®) oder getrocknete Maissilage. Bei praxisüblicher Mischfutterzusammensetzung wurde die Hälfte der Puten einer jeden Gruppe wiederum während der gesamten Versuchszeit (4 Wochen) täglich acht Stunden einer nassen Einstreu ausgesetzt.

**Versuch 5:** Die Tiere wurden auf trockenen Hobelspänen gehalten. Das Kontrollfutter enthielt bedarfsdeckende Gehalte an Biotin (300 µg/kg) und Zink (50 mg/kg), während das Alleinfutter mit hohem Biotin- (2000 µg/kg) bzw. hohem Zn-Gehalt (150 mg Zn/kg) deutlich darüber hinausging. Die Anreicherung des Alleinfutters mit MOS erfolgte mit einem Mannan-Oligosaccharid-Produkt (Bio-Mos®) in einer Dosierung von 10 g/kg. Die Hälfte der Puten einer jeden Gruppe war auch hier während der gesamten Versuchszeit (4 Wochen) täglich experimentell für 8 h einer nassen Einstreu ausgesetzt.

Zusammenfassung


Ergebnisse:

1. In allen Versuchen erwies sich die hohe Feuchtigkeit der Einstreu (73%) als dominierender Einflussfaktor. Eine achtstündige Exposition auf feuchter Einstreu war ausreichend für die Entwicklung entsprechender Fußballenveränderungen. Selbst von Exkrementen freie, durch Zusatz von destilliertem Wasser erreichte Feuchtegehalte von ~ 73% führen zur entsprechenden FPD Werten.

2. Der Zusatz von Ammoniak oder Harnsäure zur feuchten Einstreu blieb ohne Einfluss auf den Grad (Score) der FPD (3,45 vs. 3,18). Hohe Konzentrationen von Protein im Futter dürften per se somit nicht für die Entwicklung dieser Erkrankung maßgeblich sein.

3. Der Überschuss an Mengenelementen (Ca, P, Mg, Na, Cl) im Futter hatte auf trockener Einstreu keinen wesentlichen Einfluss auf den Grad der FPD (1,55 vs. 1,50), auf nasser Einstreu war hingegen bei erhöhten Mengenelement-Gehalten ein signifikant nachteiliger Effekt erkennbar (5,45 vs. 4,45).


5. Im Vergleich zu Hobelspänen erwies sich die Lignocellulose als günstig, d. h. die Ausprägung der FPD war hier reduziert (auf trockener und auf nasser Einstreu!). Bei dem hier verwendeten Stroh-Häcksel waren höhere FPD-Grade als in der Gruppe mit Hobelspänen zu beobachten. Eine Bewertung der getrockneten Maissilage muss aus versuchstechnisch-methodischen Gründen unterbleiben (bei schnell einsetzender Verschimmelung wiederholt kompletter Einstreuwechsel).

6. Die Zulagen von Biotin oder Zink (oberhalb des Bedarfs) reduzierten die Ausprägung der FPD auf trockener Einstreu deutlich (Score: 1,0 vs. 2,0), blieben aber ohne signifikante Effekte bei den Tieren, die täglich für 8 Stunden auf nasser Einstreu
Zusammenfassung

gehalten wurden (5,54 vs. 5,79); auch bei MOS-Zulage war auf trockener Einstreu ein tendentiell günstiger Effekt festzustellen (1,50 vs. 2,0).

Schlussfolgerungen:
In allen Versuchen erwies sich die achstündige Exposition auf feuchter Einstreu als stärker wirksam als alle anderen geprüften Einflussfaktoren. Diese Ergebnisse zeigen, dass erhöhte Proteingehalte im Futter per se keinen wesentlichen Einfluss auf die Entwicklung der FPD bei Mastputen haben. Erhöhte Mengenelementgehalte im Futter hatten zwar gewisse Auswirkungen auf die Fußballengesundheit – allerdings nur auf nasser Einstreu – dieser Effekt war aber viel geringer als der Einfluss der Feuchte. Erhöhte Gehalte von Sojaschrot, Kalium und OS im Mischfutter waren lediglich mit einem leichten Anstieg in der Ausprägung der FPD verbunden, allerdings nur bei nasser Einstreu. Es ist anzunehmen, dass die Epidermis durch die Feuchtigkeit in der Einstreu „aufgeweicht“ wird, wodurch leichter oberflächliche Alterationen entstehen, die sich dann sekundär entzünden.

10 REFERENCES


References


References


MAYNE, R.K., R.W. ELSE and P.M. HOCKING (2004): Histopathological changes that occur in the turkey foot pad as a result of wet and dry litter substrates. Spring meeting of the WPSA UK branch – Posters.


References


### 11 APPENDIX

Table 1: External foot pad scoring system (Mayne et al., 2007c)

<table>
<thead>
<tr>
<th>Score</th>
<th>Description of foot pad</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No external signs of FPD. Skin of the foot pad 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>A foot pad with over half of the foot pad covered in necrotic scales.</td>
</tr>
</tbody>
</table>

- The previously mentioned scores are shown in the following figures (photographs; 1 - 8) which describe the gross lesions of FPD in foot pads of young turkeys (d 15 – d 36/42 old).
Fig. 1: External score 0: normal foot pad and digital pads, (A = foot pad; B = digital pads).

Fig. 2: External score 1: slight swelling and/or redness of the skin of the foot pad.

Fig. 3: External score 2: the foot pad feels harder and denser than unaffected pad.

Fig. 4: External score 3: small black necrotic areas on the foot pad.
Appendix

Fig. 5: External score 4: the area of necrosis is < one-eighth of the foot pad.

Fig. 6: External score 5: the necrotic area extends to a quarter of the foot pad.

Fig. 7: External score 6: half of the foot pad covered by necrotic cells.

Fig. 8: External score 7: over half of the foot pad covered in necrotic scales.
Table 2: Scoring system for histopathological observations of foot pads (Mayne et al., 2007c).

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
<td>No change, sample normal.</td>
</tr>
<tr>
<td>1</td>
<td>Mild</td>
<td>Hyperkeratosis; ‘horned pegs’ of keratin on surface; epithelial hyperplasia; dead keratin on foot pad surface.</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>Epidermal acanthosis; increased dermal blood vessel density.</td>
</tr>
<tr>
<td>3</td>
<td>Mild</td>
<td>Vacuoles in dermis/epidermis; necrotic debris in keratin/epidermis.</td>
</tr>
<tr>
<td>4</td>
<td>Medium</td>
<td>Presence of heterophils, macrophages and lymphocytes in dermis.</td>
</tr>
<tr>
<td>5</td>
<td>Medium-Severe</td>
<td>Increased density of heterophils, macrophages and lymphocytes; congested/necrotic blood vessels; necrotic debris of cells in dermis/epidermis.</td>
</tr>
<tr>
<td>6</td>
<td>Severe</td>
<td>Split epidermis - 1 lesion.</td>
</tr>
<tr>
<td>7</td>
<td>Severe</td>
<td>Split epidermis - 1+ lesion or 1 very large lesion, more than one-third of total sample.</td>
</tr>
</tbody>
</table>

- These histological scores of foot pad lesions in young turkeys (d 15 – d 36/42 old) are shown in the following figures (9 - 16).
Fig. 9: Histopathological score 0 (Scale bar = 500 μm): normal dermis and epidermis layers, (Ker.: keratin; epi.: epidermis; Der.: dermis).

Fig. 10: Histopathological score 1 (Scale bar = 500 μm): A = hyperkeratosis (excess keratin); B = epidermal hyperplasia.
Fig. 11: Histopathological score 2 (Scale bar = 500 µm): A = excess keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis.

Fig. 12: Histopathological score 3 (Scale bar = 500 µm): A = excess keratin and necrotic debris in keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration.
Fig. 13: Histopathological score 4 (Scale bar = 500 μm): A = loose and excess keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = presence of inflammatory cells in dermis.

Fig. 14: Histopathological score 5 (Scale bar = 500 μm): A = loose and excess keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = increased density of inflammatory cells in dermis.
Fig. 15: Histopathological score 6 (Scale bar = 500 µm): A = compressed and eroded surface keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = increased density of inflammatory cells in dermis; G = ruptured epidermis (1 lesion).

Fig. 16: Histopathological score 7 (Scale bar = 1 mm): A = compressed and eroded surface keratin; B = epidermal hyperplasia; C = epidermal acanthosis; D = increased blood vessel density in dermis; E = hydropic degeneration; F = increased density of inflammatory cells in dermis; G = ruptured epidermis (more than one lesion).
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