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Nutrition and wet litter problems in poultry☆

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ABSTRACT

The use and management of suitable bedding material is a crucial part of bird welfare in intensive production systems. Litter cushions the bird from the hard floor, provides insulation and has the capacity to both absorb and facilitate evaporation of faecal, urinary and spilled water.

In poultry production, an increase in water excretion rate is commonly referred to as flushing, because urine and faeces are excreted simultaneously through the cloaca, making it difficult to distinguish elevated urine output or polyuria from increased faecal water loss or diarrhoea. In today's intensive production systems even relatively minor nutritionally induced osmoregulatory perturbation can cause flushing. Whilst this increase in water excretion is often initially the result of a physiological diuresis or diarrhoea, nutritionally induced pathological change may aggravate the polyuria by compromising water recovery, or increase the severity of the diarrhoea by causing enteritis.

Flushing is commonly the result of a normal physiological response to nutritionally induced water, electrolyte, pH or osmotic imbalance, but sustained imbalance frequently leads to pathological change. Similarly, diets with a high proportion of non starch polysaccharides, animal protein, saturated free fatty acids, anti-nutritional factors or toxins can cause pathological change indirectly, by inducing a detrimental shift in the composition of the intestinal microbiota (dysbacteriosis), or directly, by causing toxic damage.

Inflammation of the renal or gastrointestinal lining compromises water and nutrient transfer, which increases the amount of water, mucus and non-digested nutrients in the excreta. Apart from loading the litter with water, the mucus and non-digested lipid in particular, reduce the litter's water buffering capacity and exacerbate the consequences of flushing.

1. Introduction

A variety of litter or bedding material is used in poultry production to enhance bird health and welfare. Pine shavings, straw and oat hulls are the most commonly used products but dwindling supplies have led to the introduction of alternatives like hard wood shavings, pelleted products, paper and paper products. Whatever the source, litter serves to cushion and insulate the birds from the floor and to keep them dry. The degree to which the litter achieves these objectives is influenced by the type of litter material, house and litter management practices and bird water balance.

Large volumes of water are continuously added to the litter through spillage, condensation and excretion. Since the primary purpose of poultry house environmental control is to ensure bird comfort, house design and operation is carefully calculated and controlled, to balance water addition and water removal. Once the litter moisture exceeds 250 g/kg its

Abbreviations: NSP, non starch polysaccharide; AME, apparent metabolizable energy; GE, gross energy.

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cushioning, insulating and water holding capacity is compromised. House ventilation, temperature control and litter turning are used to enhance water loss from the litter once the litter starts to become wet.

2. Wet litter – the consequence of disturbed water balance

As with any terrestrial vertebrate, birds have the capacity to carefully control their state of hydration by constantly balancing moisture loss with intake. Since water movement across semi-permeable membranes is solute dependent, this homeostatic endeavour involves balancing both water and electrolyte intake with their excretion, a process termed osmoregulation.

Water is lost from the body as liquid in the urine and faeces and as vapour from the skin and respiratory tract. When water loss causes body water to fall below normal, homeostatic feedback controls are instantly initiated to prevent dehydration. Water intake is requirement-driven and the urge to drink originates in the satiety centre of the brain (Goldstein and Skadhauge, 2000). Body water loss causes the solute concentration of the intracellular fluid to increase (detected by osmoreceptors) and blood volume and hence blood pressure to decline (detected by mechanoreceptors) (Kaufman et al., 1980). These stimuli are integrated and converted by the satiety centre into a neurological signal which together with the secretion and conversion of renin to angiotensin-II stimulates thirst. All three mechanisms of thirst stimulation are activated when birds are dehydrated, and the urge to drink increases in intensity until water intake returns body water to normal (Kaufman et al., 1980; Kanosue et al., 1990; Goldstein and Skadhauge, 2000).

Drinking accounts for approximately 80% of water gain and is the primary means of controlling intake since feed is generally low in moisture (100 g/kg) and metabolic water production is limited by diet formulation (~33.44 g/MJ of dietary energy) (Leeson and Summers, 2005). The urge to drink only ceases once secreted renin dissipates, and cellular and extracellular rehydration is achieved.

Being the primary mechanism for balancing body water, intake parallels water loss. In a healthy bird housed at thermoneutral temperature, approximately half of the water loss is in the liquid form (urine and faecal water) and the balance is lost through evaporation (Goldstein and Skadhauge, 2000). Evaporative moisture loss during normal respiration increases markedly when panting is initiated in response to heat stress and in such situations this insensible moisture loss can reach approximately 80% of total moisture loss although this limit is unlikely to be reached under commercial production conditions. Interestingly, 50–80% of this evaporative loss occurs through the skin, which is somewhat surprising since birds do not have sweat glands (Marder and Ben-Asher, 1983; Goldstein and Skadhauge, 2000). The control of this *per cutaneous* evaporation is not completely understood but it is at least in part regulated by adrenergic induced vasodilatation in response to elevated ambient temperatures (Marder and Ben-Asher, 1983; Marder and Raber, 1989). To compensate for heat-stress induced increase in evaporative loss, water intake increases, which also aids in thermoregulation since the consumed water acts as a heat-sink.

Poultry litter becomes wet when the rate of water addition (urine/faeces/spillage) exceeds the rate of removal (evaporation) (Collett, 2007). Many years of intense selection for growth rate has indirectly increased feed and water intake and consequently water excretion rate at any given age. This means that in the average broiler house carrying 20,000 birds at currently recommended maximum stocking densities of 34 kg/m^2 approximately 2.5 tonnes of excretory water is deposited into the litter daily. At these deposition rates even minor changes in urine volume or faecal moisture content can rapidly raise litter moisture above the optimum 250 g/kg. House design and house ventilation rates/capacity have not surprisingly had to improve to keep pace with genetics.

2.1. Polyuria

Urine is the end product of renal excretory function and the volume and concentration of urine remains fairly constant in birds kept at thermo-neutral temperatures with free access to balanced ration and potable water. Blood flowing through the kidney is filtered across the glomerular epithelial lining and the filtrate is subsequently modified by a combination of secretion and re-absorption in the renal tubules before being excreted as urine. This process is under hormonal control and since 95% of renal filtrate (approximately 11 times total body water per day) is usually reabsorbed during its passage down the renal tubules, urine volume can fluctuate markedly. Polyuria, an increase in urine volume, can occur in healthy birds as a result of over hydration, solute overload and osmotic diuresis or when renal disease compromises function.

Body water and electrolyte balance is regulated by positive-feedback hormonal control. Arginine vasotocin (antidiuretic) and aldosterone (antinatiuretic) secretion reduces urine volume and sodium excretion in response to a drop in exctracelluar fluid volume (blood pressure) or electrolyte concentration. In contrast, atrial natriuretic peptide (diuretic and natriuretic) increases urine volume and sodium excretion in response to over-hydration or an increase in electrolyte concentration. The effect of renin secretion and its subsequent conversion to angiotensin II is variable depending on the electrolyte concentration of the extracellular fluid. This hormone increases urine volume (diuretic) and sodium loss (natriuretic) with salt and volume loading but decreases urine volume (antidiuretic) and sodium loss (antinatriuretic) with salt and volume depletion (Goldstein and Skadhauge, 2000). In some situations parathyroid hormone can increase urine volume by causing an osmotic diuresis through calcium mobilization and the excretion of phosphorus (Morild et al., 1985; Clark and Mok, 1986; Gray et al., 1988; Gray, 1993; Goldstein and Skadhauge, 2000).