Intestinal Integrity

A critical parameter in sustainable broiler production

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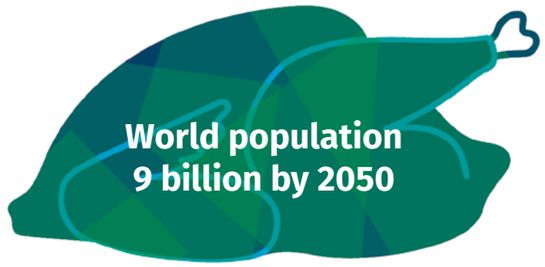
Introduction

Chickens represent the predominant species in poultry production, having grown by approximately 4.5-fold over the past 50 years (FAOSTAT, 2017).

Around 137 million tonnes of poultry meat were produced in 2020 worldwide (FAO, 2020), and poultry production is expected to rise even more drastically in the coming years to fulfil the global demand for the world population, which is projected to reach 9 billion people by 2050 (Foley et al., 2011; Perez, 2015).

Therefore, there must be greater emphasis on long-term sustainable poultry production, which is assisted through maintaining optimal intestinal flock health (Blake and Tomley, 2014; Vaarst et al., 2015).



Why does intestinal health matter?

A healthy gastrointestinal tract reduces the amount of resources required for production through optimising feed usage and limiting nutrient wastage – this increases consumer access to safe and affordable food, and promotes production sustainability (Dharne, 2008; Kasab-Bachi et al., 2017).

Intestinal diseases – such as coccidiosis, necrotic enteritis, proventriculitis and gizzard erosion – may result in a significant adverse effect on performance through impaired (increased) feed conversion ratio (FCR, defined as feed intake divided by weight gain) and increased morbidity and mortality (Van Immerseel et al., 2009; Timbermont et al., 2011; Blake and Tomley, 2014; Cervantes, 2015).

This means optimising Intestinal Integrity is fundamental, because the mechanisms of digestion and absorption are critical to the normal metabolism of the gastrointestinal tract and hence poultry performance and sustainability.

Measuring Intestinal Integrity (I^2)

Elanco Animal Health has developed a platform-based comprehensive index on Intestinal Integrity as a quantitative measurement tool for assessing intestinal health, based on performance data and flock level health, that uses a Health Tracking System (HTSi) (Kasab-Bachi et al., 2017; Swirski et al., 2020).

This Intestinal Integrity tool, also known as the I^2 index, consists of 23 health conditions known to affect intestinal health and thus expected to impact performance, welfare, and profitability – and in turn, sustainability.

Table 1 describes these conditions, which can be classed as directly related to pathogens (e.g., gross and microscopic *Eimeria maxima*, tapeworms) or non-pathogen specific conditions (e.g., excessive bile, gizzard erosions):

Pathogen-related intestinal conditions	Non-pathogen specific conditions
gross and microscopic *Eimeria maxima*	gizzard erosions
gross *E. acervulina*	mouth lesions
gross *E. tenella*	proventriculitis
gross *E. brunetti*	intestinal tone and feed passage
gross *E. mitis*	excessive intestinal fluid
gross *E. necatrix*	thin and thick intestines
necrotic enteritis	excessive bile and mucus
roundworms	cellular sloughing
tapeworms	hyperaemia and intestinal haemorrhage

Table 1. The 23 intestinal conditions of the I^2 index (Swirski et al., 2020)

How does the I^2 index work?

The I^2 index score for each flock is calculated using the average flock level lesion scores for each condition within the I^2 index and a weighting scheme (Kasab-Bachi et al., 2017).

The optimal score of the I^2 index is 100, indicating an absence of all the mentioned conditions (Table 1); however, the I^2 index can potentially be negative if all 23 diseases are present.

According to previous work done by Kasab-Bachi et al. (2017) and Swirski et al. (2020), the proposed I^2 index was found to be positively associated with average daily gain (ADG), the European Production Efficiency Factor (EPEF), and FCR.

This is in line with expectations since the I^2 index consists of intestinal conditions that reflect pathogenic invasion of the intestinal system, which intuitively would be expected to decrease feed efficiency and increase resource demand for affected birds to reach target weights (Cravens et al., 2013; M'Sadeq et al., 2015).

Kasab-Bachi et al. (2017) also reported that analysis of the HTSi database revealed significant associations between key performance indicators and many intestinal conditions (*E. acervulina*, *E. maxima*, necrotic enteritis, gizzard erosions, roundworms, excessive intestinal fluid, thin intestines, excessive intestinal mucus, and feed passage), as well as management factors (production flow and down time).

These associations were expected given that these intestinal health conditions are considered indicators of a pathogenic invasion on the intestinal system resulting in the obstruction of normal growth performance and increased demand for resources necessary to reach the target market weight (Timbermont et al., 2011; Cravens et al., 2013; M'Sadeq et al., 2015).

As such, the current I^2 index is a valuable tool for managing bird performance.

The aim of this white paper

This white paper aims to identify peer-reviewed papers on the intestinal conditions of the I^2 index and their effect on key production parameters – ADG, feed intake (FI) and FCR.

It also aims to further enhance confidence of the I^2 index being directly linked to production efficiency, and increase industry recognition of the critical importance of gut health and Intestinal Integrity in meeting sustainability challenges within broiler production.

Coccidiosis

Coccidiosis is one of the most detrimental and prevalent parasitic diseases in poultry production – it costs approximately £8.3 billion per year to the poultry industry worldwide, caused by prophylaxis and production costs (Blake et al., 2021).

What causes coccidiosis?

Coccidiosis is caused by protozoan parasites of the genus *Eimeria*.

Whilst many *Eimeria* species have been described, there are seven *Eimeria* species recognised to be most important for poultry: *E. acervulina, E. maxima, E. tenella, E. brunetti, E. necatrix, E. mitis* and *E. praecox.*

Of these, *E. necatrix, E. brunetti* and *E. tenella* are the most pathogenic (Long et al., 1976). However, *E. maxima* is the most immunogenic and moderately pathogenic, but known to induce production losses (Conway et al., 1990; Zulpo et al., 2007; Sharman et al., 2010).

Different *Eimeria* species explained

E. maxima infects the middle section of the small intestine (jejunum) causing damage from the duodenum to the ileum (Lillehoj and Trout, 1993). Therefore, an extensive gut area that is associated with nutrient digestion and absorption is potentially affected – this is likely to be one of the main reasons why *E. maxima*-infected birds have typically the largest (worst) FCR compared to birds infected with other *Eimeria* types (Idris et al., 1997; Kipper et al., 2013).

E. tenella infects the caecum and adjacent intestinal regions, usually manifesting clinically with greater intensity after day five of infection (Long and Joyner, 1984; Johnston et al., 2001). Moreover, caecal coccidiosis causes severe clinical signs and consequently higher mortality (Jeurissen et al., 1996; Mateos et al., 2002).

E. acervulina has been reported to be slightly pathogenic at initial infection and its pathogenicity is dependent on the number of the ingested oocysts (Kawazoe et al., 2005). However, in infections which resulted in more than a 20% decrease in FI, *E. acervulina* tended to cause a more adverse effect on weight gain compared to other species of *Eimeria* (Kipper et al., 2013). This could be due to *E. acervulina* infecting the duodenum, which has a greater impact on nutrient digestion.

Eimeria under a microscope

The impact of coccidiosis

In general, coccidiosis is associated with impairment of gut morphology marked by decreased villus height and associated decreased villus height/crypt depth ratio (Tan et al., 2014), and protein and amino acids digestibility (Rochell et al., 2016).

This translates into a reduction in performance, as such intestinal damage negatively affects post-absorption nutrient availability and host metabolism (Chalvon-Demersay et al., 2021).

The intracellular lifecycle of *Eimeria* causes epithelial damage, which results in functional losses leading to interruption of digestive processes, reduced weight gain, and increased mortality (Fernandes et al., 2018).

Although such deterioration of performance is undesirable, it is associated with repartitioning of nutrients from weight gain to generate protective immunity and repair pathophysiology-induced damage. Often these performance losses are transient with some degree of mitigation by compensatory growth over the bird's life (Lee et al., 2011).

Coccidiosis is usually associated with a reduction in feed intake; this is known as pathogen-induced anorexia since counterintuitively it happens at times when host nutrient resources are increased, rather than decreased (Kyriazakis et al., 1998; Laurenson et al., 2011; Hite and Cressler, 2019).

Thus, this phenomenon can be considered a defence mechanism through which birds try to divert their limited nutrient intake towards the activated immune responses, and this reduced FI is likely to be the main cause of reduced performance (Kyriazakis, 2014; Oikeh et al., 2019).

Depending on the species involved, site of infection and level of challenge, *Eimeria* infections can result in variable outcomes, ranging from limited enteritis resulting in fluid loss and malabsorption of nutrients (*E. acervulina* and *E. mitis*), to intestinal wall inflammation with pinpoint haemorrhage and sloughing of epithelia (*E. brunetti* and *E. maxima*), or to extensive haemorrhage resulting from complete villus destruction (*E. necatrix* and *E. tenella*).

In addition to reduced performance, coccidiosis can lead to a shift in the gut microbiome due to a combination of changes in the immune response and the increased presence of undigested nutrients in the lower intestine (Oakley et al., 2014, Leung et al., 2019).

Variations in the impact of coccidiosis on different birds

Kipper et al. (2013) reported that bird age has a major impact on FI in *Eimeria*-challenged birds.

In addition, bird sex and genetics are another variable that impacts performance during challenge, with females being more sensitive to *Eimeria* challenges than males (Zhu et al., 2000), and cross-breed animals being more resistant to coccidiosis than pure-line animals (Kipper et al., 2013).

Cost of coccidiosis to the poultry industry worldwide

£8.3 billion

Coccidiosis and broiler performance

Analysis of the HTSi database revealed significant associations between two coccidian species – *E. maxima* and *E. acervulina* – and FCR, body weight gain (BWG) and EPEF, as key performance indicators (Kasab-Bachi et al., 2017).

These findings are in agreement with various studies that have attempted to quantify the negative impact coccidial challenge has on the performance of broilers (Conway et al., 1990; Parker et al., 2007; Giannenas et al., 2014; Amerah and Ravindran, 2015):

- Conway et al. (1990) investigated the relationship of the lesion scores of *Eimeria* spp and BWG in infections of *E. acervulina, E. maxima*, and *E. tenella* in broilers. For *E. maxima* and *E. tenella*, a modest increase in weight loss with increasing severity of infection was reported.
- Parker et al. (2007) demonstrated that the average FI and BWG of broilers challenged with a mix of *Eimeria* spp. were significantly reduced (21% and 45% respectively; P≤0.001), and that the FCR was increased by 43% (P≤0.001), in comparison to the unchallenged birds.
- Giannenas et al. (2014) reported a significant reduction in BWG (14%; P<0.001) and increased FCR (16%; P<0.001) for *E. maxima, E. tenella* and *E. acervulina*-challenged birds compared to unchallenged birds.
- Amerah and Ravindran (2015) found that *E. acervulina, E. maxima*, or *E. tenella*-challenged birds had reduced BWG (9%; P<0.05), reduced FI (18%; P<0.05), and increased FCR (13%; P<0.05), compared to unchallenged birds.
- Jones et al. (2019) reported a significant reduction in BWG (18%, P<0.005) and an increased FCR (18%, P<0.005) due to a severe and uncontrolled coccidiosis outbreak based on a review with referenced studies from 2005 to 2014.
- Gaucher et al. (2015) reported a live weight reduction of 50 g for birds of around 2.4 kg and a 4% increase in FCR due to coccidiosis in a large-scale commercial study in Canada.
- Kim et al. (2017) found birds challenged with *E. maxima* and *E. acervulina* had a depressed BWG, lower FI and increased FCR (38%, 21% and 31% respectively; P<0.001).
- Leung et al. (2019) found birds challenged with *E. maxima* and *E. acervulina* had a lower BWG (8%; P<0.001) and increased FCR (14%, P<0.001).

A recent meta-analysis (Taylor et al., 2022) focused on *E. maxima, E. tenella* and *E. acervulina* challenges on growth performance revealed that the average daily FI following either *E. tenella* or a mixed species infection was reduced by 8% and 10% respectively, compared with the control group (P<0.005).

In addition, all infected groups had decreased average BWG (*E. maxima*: 16%, *E. tenella*: 20%, and *E. acervulina*: 33%; P<0.05).

Apart from *E. tenella*-infected groups, FCR was significantly increased following challenge with *E. maxima* (30%), *E. acervulina* (28%) and mixed *Eimeria* species (44%) (P<0.05). In addition, penalised performance was also observed due to *Eimeria* infection in other studies (Sakkas et al., 2018; Teng et al., 2020; Eckert et al., 2021).

Overall, most of these studies show that the negative impact of coccidial challenges on FCR is greater than that on FI, supporting the view that some loss in BWG comes from a reduced digestive capacity and a diversion of available nutrients away from performance, presumably into dealing with the consequences of elevated pathophysiology and immune responses (Blake et al., 2020).

Although *Eimeria* infection can cause moderate to high mortality in broilers (Noack et al., 2019), this is not always apparent from small-scale experiments (Taylor et al., 2022).

A main reason for this discrepancy is that most of these studies are conducted under local and/or national ethics regulations for animal welfare, with birds being culled before their deteriorated health could lead to mortality.

Necrotic enteritis

Following coccidiosis, necrotic enteritis (NE) is arguably the second most severe and common infectious disease resulting from intestinal mucosal damage.

It is considered the most prevalent economically important poultry-related disease, as its clinical form causes up to 30% mortality in infected flocks (Van Immerseel et al., 2009; Gharib-Naseri et al., 2019; Bae et al., 2021).

However, as with coccidiosis, it is the sub-clinical form of NE that predominantly occurs in the poultry industry, which is considered to be most devastating because it results in poor feed efficiency and profitability losses, and it often persists untreated because it can pertain in flocks without any detectable signs (Skinner et al., 2010; Bansal et al., 2021; Salem et al., 2021).

Sub-clinical necrotic enteritis can have significant cost implications to the global broiler industry, in terms of production losses and mitigation measures. Annual estimates range from ~2 billion USD (approx. £1.6bn) (Van Der Sluis, 2000) to ~6 billion USD (approx. £4.9bn) (Timbermont et al., 2011).

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What causes necrotic enteritis?

Necrotic enteritis is caused by *Clostridium perfringens* – an opportunistic spore forming organism that is found in the gut of healthy chickens (Kondo, 1988).

Clostridia spores germinate under favourable conditions and release toxins such as alpha-toxins and NetB toxins, which have significant roles in NE severity as they are cytotoxic to enterocytes (Keyburn et al., 2010).

The amino acid glycine, which can usually be found in abundance in poultry rations, plays a significant role in NE. It forms a conjugated compound with bile salts and interacts with the clostridial SleC gene, both driving *C. perfringens* germination patterns (Miyata et al., 1997; Sorg and Sonenshein, 2008).

Moreover, high levels of protein in diets, especially from fish meal, coccidiosis co-infection, and any other factors that cause disturbance in the integrity of intestinal mucosa are considered predisposing factors that trigger the pathogenic state of *C. perfringens* (Collier et al., 2008; Stanley et al., 2014).

How necrotic enteritis affects different birds

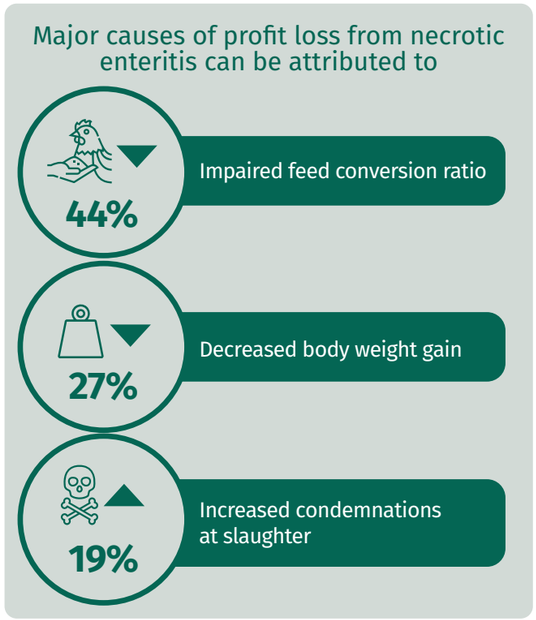
The incidence of NE is greatest between two and six weeks of age, and it leads to increased intestinal damage and mortality in broilers (Jackson et al., 2003; Cooper and Songer, 2009; Wade and Keyburn, 2015).

Clinical observations include diarrhoea, depression and melena (dark faecal droppings arising from internal bleedings), which together with sub-clinically decreased production performance result in significant profit loss (Shojadoost et al., 2012).

Whilst the clinical form of NE is associated with high mortality rates, and thus a major source of economic loss in the poultry industry (Cravens et al., 2013; M'Sadeq et al., 2015; Timbermont et al., 2011; Kasab-Bachi et al., 2017), the more difficult to detect sub-clinical form of NE has an even greater economic impact (Shojadoost et al., 2012). Such impaired performance arises from chronic damage to the small intestinal mucosa (Kaldhusdal et al., 2001, Van Immerseel et al., 2009).

The lesions of NE are very prominent in the small intestine, whereas the caeca are the main site of *C. perfringens* replication (Van Immerseel et al., 2004; Cooper et al., 2013).

The severity of this disease depends on various predisposing factors including high levels of dietary crude protein, high fibre and other non-starch polysaccharides, management stress, immunosuppression, and imbalance of commensal intestinal microbiota (Moore, 2016).



The combination of necrotic enteritis and coccidiosis

The enhanced mucus production during *Eimeria* spp infection can act as a substrate for the growth and proliferation of *C. perfringens* (Collier et al., 2008; Prescott et al., 2016).

It has been suggested that coccidiosis and NE both change tight junction protein structure and function, destroy the intestinal barrier, and cause severe intestinal inflammation (Awad et al., 2017), eventually resulting in intestinal damage and decreased production performance (Zhang et al., 2022).

For instance, Zanu et al. (2020) found that BWG and FI was reduced (17% and 11% respectively; P<0.05) and FCR was increased (9%; P<0.05) in birds challenged with a combination of *E. acervulina, E. maxima, E. brunetti* and *C. perfringens*.

Moreover, Akerele et al. (2022) reported a significant reduction in BWG (31%; P=0.008) and an increase in FCR (33%; P=0.008) in birds challenged with *E. maxima* and *C. perfringens*.

The higher FCR in flocks infected with NE might be attributed to the chronic intestinal mucosal damage that results in poor digestion and increased FCR (Skinner et al., 2010; Timbermont et al., 2011; Van Limbergen et al., 2020).

Thus, data available on sub-clinical NE with sub-clinical coccidiosis suggest that impact on BWG is greater than impact on FI only; this suggests that affected flocks perform at a more detrimental FCR, arising from impaired feed digestion and diversion of available nutrients away from growth.

Gizzard erosion

Gizzard erosion and ulceration (GEU) syndrome was observed for the first time in the early 1930s by Holst and Halbrook, who described it as dark erosion spots in the gizzard lining of 21-day-old chickens.

Gizzard erosion and ulceration (GEU) syndrome is characterised by macroscopic defects in the solid layer covering the gizzard, known as the koilin layer, and also in the gizzard mucosa (Gjevre et al., 2013).

What causes gizzard erosion?

GEU may be congenital and therefore caused by factors that exist before hatching and associated with breeder age (Good et al., 1968), breeder diet, and capillary fragility arising from a transient increase in blood pressure during hatching (Tepper and Bird, 1942).

In addition, deprivation of feed and/or water was reported to induce gizzard erosion in broilers (Bierer et al., 1966). Furthermore, ingestion of toxic substances, such as biogenic amines, has been associated with gizzard erosions (Harry and Tucker, 1976).

Infections and microbial colonisation have also been reported to be associated with gizzard erosion (Ono et al., 2003; Manarolla et al., 2009).

Involvement of fowl adenovirus as a main infectious cause of gizzard erosion was first described by Tanimura et al. (1993).

Thereafter, fowl adenovirus was isolated from affected gizzards in Japan (Ono et al., 2001) with natural outbreak between 9 and 36 days of age reported in many countries including Japan (Abe et al., 2001), Belgium (Garmyn et al., 2018), Germany (Grafl et al., 2012; Schade et al., 2013) and Poland (Domanska-Blicharz et al., 2011).

The impact of gizzard erosion

Mirzazadeh et al. (2019; 2021) reported a significant reduction in body weight of the gizzard erosion affected flock compared to a healthy flock by ~25%, with a significantly higher total mortality rate (+1.7%) than the average total mortality rate of the unaffected flock at slaughter age.

Similarly, an increase in mortality rates was observed in previous outbreaks of gizzard erosions (Abe et al., 2001; Grafl et al., 2012; Schade et al., 2013). In addition, reduced FI and BWG (17%; P<0.05) were documented in Germany and Belgium (Grafl et al., 2012; Schade et al., 2013; Garmyn et al., 2018, Grafl et al., 2022).

Finally, *C. perfringens* in the small intestine was reported to causally correlate to GEU occurrence; several studies observed that its counts increased significantly with severity of mucosal gizzard erosions (Novoa-Garrido et al., 2006; Dinev, 2010; Kaldhusdal et al., 2012; Gjevre et al., 2013).

Thus, GEU syndrome as a multi-factorial basis, and where it is noted, is associated with reduced performance, though the impact on performance seems to be less pronounced than for the previously described (sub-clinical) coccidiosis and NE.

To our knowledge, detailed reports from adenoviral gizzard erosions within the UK are rare and available only from other European countries including Germany, Hungary, Poland, and Italy, as previously described.

In addition, there are no available vaccines for gizzard erosions on the UK market (Chapman and Hersey, 2022).

Adenovirus is resistant to many common approved disinfectants used on poultry farms in the UK; therefore this may lead to carryover from crop to crop and aid in horizontal transmission of the disease (Chapman and Hersey, 2022).

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Proventriculitis

Proventriculitis is the inflammation of the proventriculus, which is a narrow glandular region between the crop and the gizzard, and it usually impacts feed digestion and consequently growth performance.

What causes proventriculitis?

It can be caused by dietary exposure to biogenic amines, mycotoxins, and lack of dietary fibre (Dorner et al., 1983; Goodwin et al., 1996; Barnes et al., 2001).

Furthermore, it can also be caused by a large number of infectious agents such as bacteria, fungi and viruses (Huff et al., 2001; Pantin-Jackwood et al., 2003; Tomaszewski et al., 2003).

One of the most known causes of proventriculitis is from a virus called chicken proventricular necrosis virus (CPNV).

This virus is responsible for transmissible viral proventriculitis (TVP) and it impairs glandular epithelial cells of the proventriculus that are responsible for pepsin and hydrochloric acid secretion (Wali, 2021). As such, proventriculitis impacts on digestion in general, and protein digestion in particular.

TVP was first described in the Netherlands more than 40 years ago (Kouwenhoven et al., 1978), and it has since been reported in several countries in North America (Noiva et al., 2015), Europe (Grau-Roma et al., 2010; Marguerie et al., 2011) and Asia (Kim et al., 2015).

TVP has been reported in broilers from 13-59 days old (Grau-Roma et al., 2020), broiler breeders from 9 to 20 weeks old and laying hens (Marusak et al., 2012; Kim et al., 2015; Noiva et al., 2015).

Mortality

3-5%

In the UK, the mortality rate in flocks with TVP is reported to be 3-5% (Grau-Roma et al., 2017).

How proventriculitis affects birds

This disease is characterised by enlargement and pallor of the proventriculus, and proventricular wall thickening (Guy et al., 2011; Allawe et al., 2017).

The clinical symptoms of the disease are non-specific and characterised by stunted growth, poor FCR (Dormitorio et al., 2007; Śmiałek et al., 2017) and impaired digestion (Marguerie et al., 2011; Allawe et al., 2017).

TVP has great economic implications since it can increase costs by decreasing production indicators in affected flocks and increasing the rate of stunned broilers (Guy et al., 2005; Kim et al., 2015; Noiva et al., 2015).

However, the available information about both TVP and CPNV is very limited (Grau-Roma et al., 2020), and most of the TVP reports were based on few cases or on experimental infections (Kim et al., 2015; Noiva et al., 2015).

A first peer-reviewed report describing the occurrence of this disease in the UK was published only 6 years ago (Grau-Roma et al., 2017).

Polish insight into TVP

A recent published study in Poland (Śmiałek et al., 2021) reported a significant reduction in BWG (30%; $P<0.05$) in TVP-affected groups compared to the control group – this resulted from pepsinogen and hydrochloric acid-producing cell destruction.

The study also found that in more severe cases of TVP, 8% of these cells are destroyed because of necrosis caused by the infection.

Furthermore, though this study did not observe an increase in mortality in the course of TVP, the number of culled birds likely from meeting ethical endpoints within the study protocol increased significantly.

Other intestinal conditions

There are a number of other conditions included in the I^2 index that are clearly observed as identifiable pathology, even though their main causes are harder to track. Some of these include cellular sloughing, excessive intestinal mucus content, and feed passage.

The presence of excessive mucus in the intestine may be associated with certain disease agents – viruses, coccidia, bacteria – and feed toxins which stimulate excessive mucus secretion.

C. perfringens – the leading cause of NE, as detailed before – can use mucus as a growth medium and energy source, further exacerbating bacterial enteritis.

Feed passage is an indicator of sub-optimal digestion as it refers to the presence of undigested feed in the large intestine or chicken droplets (greater than 25% content).

Whilst such undigested feed presence can be expected to have an impact on feed efficiency, and as such affects the I^2 index, its cause is not clear.

In addition, significant associations were found between these intestinal conditions caused by various parasitic, bacterial, or viral organisms (i.e, excessive intestinal fluid, thin intestines, excessive intestinal mucus, and feed passage), gastrointestinal nematodes, and the key performance parameters (Kasab-Bachi et al., 2017).

I^2 index and leg health (footpad dermatitis, hock burn, lameness)

A reduced Intestinal Integrity, as evidenced by a lower I^2 index, would be associated with increased volume and wetness of excreta, which has an impact on litter condition and leg health.

Footpad dermatitis and hock burn are conditions that are characterised by necrotic erosions on the plantar surface of the footpads and hock joint in growing broilers and turkeys (Haslam et al., 2007).

These conditions result in serious welfare issues, but also economic losses, arising from downgrades and condemnations of the affected portions of the leg (Shepherd and Fairchild, 2010; Elson, 2015; Dawkins et al., 2017).

The necrotic and ulcerative lesions of the footpads and hock joint could be a gateway for pathogenic organisms, which when resulting in (sub-clinical) infection, further reduce performance, worsen FCR and lower final body weight (Abd El-Wahab et al., 2020).

Therefore, the assessment of those conditions is considered an important indicator of bird welfare in several countries such as the United States, Denmark, the Netherlands, Sweden, and Germany (Berg, 2004; Kjaer et al., 2006; de Jong et al., 2012; Piller et al., 2020).

footpad dermatitis

Footpad dermatitis

Hock burn

Footpad dermatitis was the most common condition across 53 UK flocks examined in 2017, with an average prevalence of 51.6%, whilst 20.5% of the flocks had some degree of hock burn (Dawkins et al., 2017).

Breed selection

World-wide, including in the UK, selection for fast-growth breeds has contributed greatly to improved feed efficiency, but it has also been reported to contribute to welfare issues, including leg disorders (Danbury et al., 2000).

In 2017 the Royal Society for the Prevention of Cruelty to Animals (RSPCA) commissioned a trial to assess the effect of fast- and slow-growing broiler breeds on production, leg health and walking ability when they achieved the average UK slaughter weight of 2.2 kg, as well as at 2.5 kg live weight to assess when these birds reached heavier weights.

The results revealed that the fast-growing breeds on average had significantly poorer leg health, higher mortality, and greater culling rates than the slow-growing breeds.

These figures accord with a UK survey of 4.8 million birds in 2008, in which 27.3% had gait scores of three or above, which implies that the welfare of more than a quarter of broilers produced in the UK might be compromised to some degree (Knowles et al., 2008).

Moreover, there are environmental and managemental factors significantly associated with leg health disorders – these include season, age of the bird, stocking density, and shorter dark periods during the day.

A reduced Intestinal Integrity, as evidenced by a lower I^2 index, would be associated with increased volume and wetness of excreta

Wet litter

I^2 and climatic conditions

Climatic conditions, and thus by extension seasons, have an important effect on poultry behaviour, with more free-range broilers observed outside the shed on warm summer days (Dawkins et al., 2003).

Climatic conditions have also been reported to be a major influence on leg health, mortality, and production efficiency (Jones et al., 2007).

Jones et al. (2007) reported that broilers exposed to cold weather showed leg stiffness and footpad dermatitis. Moreover, ascites were found to be profoundly worse during cold weather and instances of poor ventilation (Mullan et al., 2021).

On the other hand, it has been found that production efficiency was reduced in summer with increased mortality, poor growth, and feed wastage (Sanchez-Casanova et al., 2021).

This might be attributed to high levels of panting in hot weather (heat stress), which would contribute to reduced feed intake and growth performance.

In addition, heat stress results in heart enlargement, right ventricular hypertrophy, congestion and hyperaemia in lungs, and yellow and pale liver (Sanchez-Casanova et al., 2021).

Heat stress also elevates serum corticosterone levels, reduces BWG, and alters blood acid-base balance (Sandercock et al., 2001; Quinteiro et al., 2010).

Annual economic losses in poultry production are significant when the environmental conditions are outside birds' thermos-neutral zone; it has been estimated that heat stress increases economic losses by almost 30% (St-Pierre et al., 2003).

Consequently, controlling the environmental parameters (temperature, relative humidity, and air velocity) within broiler houses is very important to alleviate heat stress problems from negatively affecting growth performance (Aradas et al., 2005).

Variation in the ability to control climatic conditions may very well contribute to variation in I^2 over time.

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The I^2 index and environmental footprint of broiler production

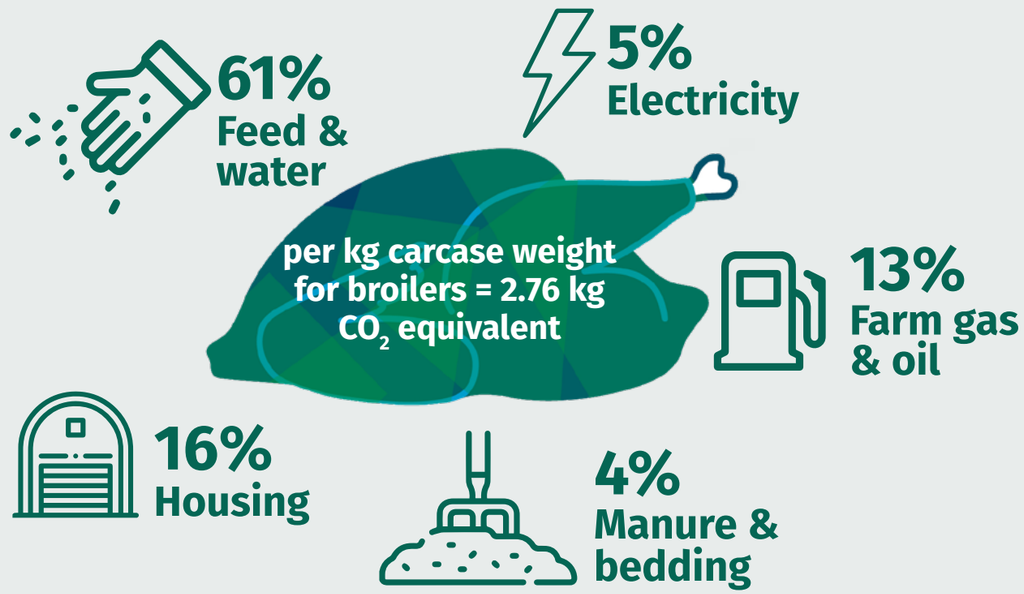
The previous overview clearly demonstrates that the conditions underlying within the I^2 index are linked, to variable degrees, to reduced feed efficiency, with demonstrable detrimental impacts on BWG, FI and FCR. Thus, a lower I^2 index is typically related to a greater (more detrimental) FCR.

The consequence of this, and especially the latter, is an increased resource input for the expected output, which results in increased feed required for the same amount of output as well as additional bedding needed, more manure being produced, and a lengthening of the fattening period, which attracts additional resource input for environmental control (electricity, fuels etc).

Each of these resource inputs, as well as manure management, have an environmental footprint and they contribute to the carbon footprint of poultry production.

Breeding for improved feed efficiency has arguably greatly contributed to the reduced environmental footprint of broiler production (Sell-Kubiak et al.,

Feed production contributes the most to the carbon footprint of poultry production; figures from a recent simulation study report that the global warming potential per kg carcase weight for broilers was 2.76 kg CO_2 equivalent, of which 61% was for feed and water, 5% for electricity, 13% for farm gas and oil, 16% for housing, and 4% for manure and bedding (Leinonen et al., 2016).



2017) – they quote that over the last 30 years or so, day 35 body weight increased from 1.4 to 2.44 kg (+71%), whilst feed intake increased from 3.22 to 3.66 kg (+14%) to reach this point.

Thus, the modern bird performs at a much more favourable FCR than its lighter counterpart (1.53 vs 2.37, an improvement of -35%), and at a lower global warming potential. All else being equal, it also performs at lower eutrophication and acidification potential.

Maintaining this advantage of genetic improvement is clearly sensitive to gut health.

A reduction in I^2 score for a current flock of broilers arising from more detrimental FCR effectively cancels out years, if not decades, of genetic selection.

In addition, it has been predicted that selection for improved feed efficiency is likely reaching its limit in broilers (Tallentire et al., 2018), largely arising from inherent biological inefficiencies of energy digestion, absorption and metabolism.

This suggests that, going forward, maintaining optimal production efficiency – and thus minimising global warming potential, eutrophication and acidification potentials – will increasingly become a function of other drivers than genetics, including safeguarding gut health with tools such as the I^2 index being instrumental to monitor and manage the latter accordingly.

A reduction in I^2 score for a current flock of broilers arising from more detrimental FCR effectively cancels out years, if not decades, of genetic selection.

Conclusion

To conclude, the HTSi database and the I2 index can be considered valuable tools for monitoring and managing poultry intestinal health, performance as well as welfare, profitability and sustainability.

Consequently, the application of such tools by poultry producers can support and evaluate other concurrent industrial efforts to improve sustainability and minimise carbon footprint resulting from optimising intestinal health metrics for this rapidly growing component of world food production.

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