



Campylobacter:

Can we slow the clock on this chicken timebomb?

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Ross 308 faster chicken growing breed

It's not too late to change

Broilers (chickens reared for meat) have been selectively bred to grow extremely quickly, using as little feed as possible, with today's conventionally reared broilers reaching their market weight of 2.2kg in as little as 35 days. While this is great for business, there is a serious cost to the birds themselves. Multiple studies and reports have shown that faster growing, lower welfare birds suffer much more than slower growing breeds, but these unnaturally faster growing birds still dominate the food market.

It has been demonstrated that faster growing breeds often also have more compromised immune systems than slower growing, higher welfare breeds, like those approved by the RSPCA. We wanted to explore how this links in with **campylobacter**, a bacteria that causes serious food poisoning in humans, with an estimated nine million cases annually in the EU. Campylobacter was, and sometimes still is, thought to be commensal in chickens, meaning that it lives naturally in the gut of a chicken with no negative effects for the bacteria or the bird i.e. they live commensally. While this was once the case, through selective breeding to increase growth rates, in what appears to be a race to increase profits over welfare, these faster growing birds can no longer live commensally with campylobacter and actually suffer in multiple ways because of it. Not only do faster growing birds suffer more physically – through inflammation and hock and feet burns – but campylobacter in stressed birds can be more virulent and more invasive, potentially increasing the risk of human infection.

It is extremely disappointing that an animal which previously lived harmoniously with a naturally-occurring gut bacteria now seems to actively suffer because of it. Physical and mental suffering is now often a customary price of profits. While much hard work has been done over the past few years to reduce the amount of campylobacter that reaches the consumer, most of these steps are made after slaughter and are therefore of no welfare benefit to the birds themselves. To improve the birds lives changes should be made, including only slower growing, higher welfare breeds being reared.

Furthermore, while there is some great work going on to reduce the amount of antibiotics being used on farms in the UK, campylobacter continues to develop increasing antibiotic resistance at an alarming rate – an antibiotic timebomb. This does not just affect the birds; antibiotic resistance in human campylobacter has been strongly linked with antibiotic use in poultry, with some of those antibiotics still being used today, and some no longer able to be used in the birds because of the resistance. Although it isn't yet clear how this resistance in the human campylobacter infections might impact human medicine we believe it is an avoidable risk through making improvements in the breeding and care of broiler chickens.

This report is a review of the evidence already available that demonstrates how a higher welfare, slower growing bird would suffer less and how use of these breeds may also reduce the human campylobacter risk, as well as highlighting the potential antibiotic timebomb.

We strongly believe the Better Chicken Commitment provisions should be the minimum welfare standards for all broiler chickens. While some are adopting these changes, many have yet to do so and widespread problems continue across the food industry.

It's not too late to change.

...fluoroquinolone resistance in *C. jejuni* from retail chickens was 52.4 percent... tetracycline resistance in *C. jejuni* from retail chickens was even higher at 60.6 percent.



Key findings

- Approximately 70 percent of human campylobacter infections in the UK come from chickens, making it a public health concern.

- Campylobacter is no longer a commensal bacteria in chickens and can lead to increased welfare problems.

- Stress levels associated with lower welfare negatively affect the gut microbiota balance making the birds more susceptible to disease.

- Campylobacter in stressed birds is more virulent and more invasive, travelling out of the gut and into surrounding tissues such as the liver, leading to increased shedding in faeces.

- Infected faster growing breeds exhibit a more severe and prolonged inflammatory response than infected slower growing breeds, resulting in intestinal mucosa damage, diarrhoea, and further environmental welfare problems can then arise through the wetness of the litter caused by the diarrhoea.

- Faster growing birds infected with campylobacter are more likely to physically suffer with increased pododermatitis and lesions.

- Substantial human campylobacter antibiotic resistance is linked with poultry antibiotic usage, with one class of antibiotics now only able to be used in broilers as a last resort.

- Campylobacter is now showing alarming resistance to the class of antibiotics that are still being used in broiler chicken production.

One-day-old broiler chicks

Background

Campylobacter is globally the cause of one in four diarrhoeal diseases (WHO, 2020), and the most common cause of food poisoning in the UK (NHS, 2021) with approximately 70 percent of all human campylobacter infections coming from chicken (FSA, 2021). While there are consistently more than 50,000 confirmed cases (2009–2017) in England and Wales alone (Public Health England, 2017), there are more than 246,000 cases reported in the EU annually. However, it is believed that the actual number is much closer to nine million each year (EFSA, 2021). This is likely due to gastrointestinal diseases often just having the symptoms treated rather than formal identification of the underlying cause.

There are 17 species and six subspecies of campylobacter, although the two most commonly reported in human disease are *C. jejuni* and *C. coli*, with *C. jejuni* believed to make up over 90 percent of cases (CDC, 2019). The optimum temperature for campylobacter growth is 37°C to 42°C, making chickens and humans alike an ideal host, with chickens having an average body temperature of 41°C to 42°C. Retailers have worked well with the Food Standards Agency (FSA) in previous years to successfully reduce campylobacter contamination and still now continue to report their results. However, they only report the percentage of chickens contaminated with >1,000 cfu/g. To put this in perspective, the infectious dose of campylobacter can be incredibly low with <500 organisms able to cause disease in humans (Laughlin et al, 2019). This means that only 0.5g taken raw from just one of those chickens could cause disease in a human. The full 10g sample from one of those chickens would have >10,000 organisms, more than 20 times the number required to cause illness. The Food Standards Agency (FSA) (2019) showed that the average campylobacter levels, across all nine major retailers, smaller retailers, and butchers, were seven percent at >1,000 cfu/g, and 20 percent at 100–1,000 cfu/g. This means that 27 percent of all the samples taken by the FSA contained enough campylobacter to cause illness in humans. This shows that while the retailers continue to meet the FSA seven percent target, following previous data, the actual number of retail chickens carrying enough campylobacter to infect a human can be approximated at up to 270,000 birds for every one million reared, if not more.



Introduction

Due to the health implications campylobacter causes in humans, there is a lot of research into how to reduce colonisation at multiple control points across the poultry production cycle. While this is an extremely important area of research for food safety, the actual negative effects campylobacter has on the birds themselves is often overlooked due to the assumption that it is still a natural commensal relationship. This report aims to challenge this assumption and identify some of the ways that birds suffer due to the inoculation of campylobacter. Building on this premise, multiple areas will be explored to identify further links between campylobacter colonisation and bird welfare, including faster growing and slower growing breed comparisons, through to another extremely concerning area – antibiotic usage and resistance.

If campylobacter is a commensal bacteria in chicken, infection would result in no underlying negative health or welfare implications. However, campylobacter has been shown to negatively affect chickens in a variety of ways, with further links being drawn between breed, environment, and welfare. It's been shown that broilers, regardless of faster growing or slower growing, elicit an early innate inflammatory response when colonised with *C. jejuni*. This initial inflammatory response, while natural, remains elevated in fast-growing breeds, leading to prolonged

intestinal inflammation and even diarrhoea. Even with the initial welfare implications involved with the intestinal inflammation, further environmental welfare problems can then arise through the wetness of the litter caused by the diarrhoea. It has also been demonstrated that both faster growing and slower growing birds show evidence of intestinal mucosa damage from campylobacter (Humphrey et al, 2014), although at significantly different levels, further challenging the concept of campylobacter being a commensal bacteria.

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As well as welfare implications, *C. jejuni* has also been shown to affect weight performance in birds, a characteristic of 'quality'. Awad et al. (2014) observed a weight gain reduction in broilers that had been orally infected with *C. jejuni*. This observation was replicated in a second study, using the same strain, in which a significant body weight reduction manifested in the broilers 21 days post oral infection (Awad et al, 2015). As well as bird welfare, it appears bird 'quality' can also be affected by campylobacter, however, this report will focus on bird welfare.

Without taking away from the human health consequences associated with campylobacter, which will be linked in later, it is important to understand the health and welfare implications it is having on chickens, and how higher welfare, slower growing breeds are better equipped to deal with the bacterium. The first area of focus will be stress levels and its role in disease susceptibility. This includes how the microbiota is disrupted as well as how stress can increase the virulence of campylobacter. The second area will explore the differences between faster growing and slower growing breeds, including the immune responses and resulting welfare implications. The next area will examine some of the environmental factors that link in with campylobacter, including litter quality and the thinning process. Finally, the timebomb that is antibiotic resistance will be evaluated, specifically the resistance that campylobacter continues to develop against classes of antibiotics used both in the past and presently. Exploring these areas will demonstrate that campylobacter is not merely a commensal bacteria as previously thought. Campylobacter negatively impacts bird health and welfare, particularly in industry standard faster growing breeds, and is also detrimental to the future

efficacy of antibiotics for the birds, and potentially for humans. An industry shift in animal welfare to implement the Better Chicken Commitment as the minimum welfare standard would not only significantly improve the lives of millions of birds, it would undoubtedly help with the antibiotic timebomb that is happening now.

Welfare definition

Welfare is a term that can have slightly different meanings within different contexts. For example, a simplified definition would likely just cover generalised terms such as health and well-being. In fact, the *RSPCA Welfare Standards For Meat Chickens* (2017) covers more than 600 individual standards all the way from chick sourcing, through to slaughter. This is because the RSPCA believes that animals experience good welfare if they are physically fit, psychologically fulfilled and are happy and healthy. The following is the definition given by the World Organisation for Animal Health (2021):

"Animal welfare means the physical and mental state of an animal in relation to the conditions in which it lives and dies.

"An animal experiences good welfare if the animal is healthy, comfortable, well nourished, safe, is not suffering from unpleasant states such as pain, fear and distress, and is able to express behaviours that are important for its physical and mental state.

"Good animal welfare requires disease prevention and appropriate veterinary care, shelter, management and nutrition, a stimulating and safe environment, humane handling and humane slaughter or killing. While animal welfare refers to the state of the animal, the treatment that an animal receives is covered by other terms such as animal care, animal husbandry, and humane treatment."

For the purposes of this report, the term 'welfare' is used as a generalised term to encompass multiple welfare parameters. Low welfare can be assumed to mean any, or all of the following: lack of environmental enrichment; high stocking densities (>30kg/m²); faster growing breeds (~2.2kg in <36 days); poor environmental conditions (e.g. poor litter quality). Higher welfare infers the opposite.

Stress

Firstly, it is important to note that the inability, or lack of stimuli, to perform basic natural behaviours can lead to frustration and stress (Mason and Burn, 2011). It is also important to note that both faster growing and slower growing broilers share the same motivation to perform these natural behaviours, such as perching, walking and scratching (Bokkers and Coene, 2003). As lower welfare standards can include lack of environmental enrichment and the inability to perform natural behaviours due to bird size and morphology, and higher stocking densities, a number of avoidable stressors are all present in lower welfare systems as standard. This means that birds of higher welfare breeds reared under higher welfare standards experience fewer stressors during rearing.

Bull et al (2008) collected samples from 214 different farms throughout the UK and demonstrated that under commercial British broiler rearing conditions, there is a relationship between campylobacter colonisation levels and digital dermatitis/rejections at slaughter due to infectious disease, both of which have a negative impact on bird welfare. 'Commercial British broiler conditions' implies the birds reared were commonly-used faster growing breeds. The study found that if the dermatitis incidence in the flock was $\geq 2\%$, the flock was twice as likely to be campylobacter positive. Although the study shows correlation rather than cause and effect, these results were replicated by Williams et al (2013), showing an increase in hock and pododermatitis levels in faster growing birds infected with campylobacter, and again by Alpigiani et al (2017) in another similar study. This particular small scale study demonstrated that flocks that had more than 25 percent of birds with severe lesions (on 25–50 percent of the footpad) were predicted to be campylobacter positive by the end of the rearing period, with lesions acting as a predictive welfare measure. By improving bird welfare, this may also reduce campylobacter colonisation in broilers, with one possible link being stress.

It is known that raised stress levels, that can be caused by low welfare conditions, can cause elevated neurotransmitter levels, including noradrenaline (Cheng et al, 2002). In vitro studies have demonstrated that campylobacter spp. will have higher motility and growth rate when grown in the presence of noradrenaline (Cogan et al, 2006). This could lead to increased shedding (the expulsion of pathogenic bacteria), increasing

transmission through the flock when stress levels are high, caused by low welfare. This could also be triggered during other stressful situations such as thinning. This has been further investigated by Avoori et al (2014) who demonstrated that *C. jejuni* is more virulent and more invasive in the presence of noradrenaline. Within the noradrenaline-pretreated birds the campylobacter invaded more livers, and the number of bacteria in these livers was significantly higher. Noradrenaline not only increased the virulence and invasiveness of the campylobacter, but also stimulated the birds to shed more of it. With campylobacter being more virulent and more invasive in stressed birds, this implies that these birds pose a higher risk of human illness due to the campylobacter not remaining within the gut and penetrating surrounding tissues. During factory processing the innards of the bird are removed, however this is irrelevant if the campylobacter has already spread, especially as livers are often collected and sold separately.

Williams et al (2013) directly compared the effects of campylobacter on 37-day slaughter weight Ross (a commonly used breed) birds and 56-day slaughter weight Hubbard birds, kept under the same environmental conditions. At two, seven and 16 days post infection, there was no significant difference in spleen, liver or caeca colonisation between the two breeds, however the faster growing breed, the Ross, did have more infected spleens and livers overall. The small sample sizes could explain why these differences were not found to be significant, however this does still support the theory that campylobacter is more motile and invasive within faster growing birds.



Faster growing breed resting on litter

Stress can have other physiological health impacts on birds, such as the gut microbiota balance. This can have a significant effect on disease susceptibility and immune response. Meimandipour et al (2010) demonstrated that rough handling-induced stress increased blood corticosterone concentrations in birds which negatively impacts both metabolic activities and bacterial composition within the gut microbiota. This could be due to the reduction in beneficial gut bacteria such as lactobacilli and bifidobacteria, caused by the stress. Although competitive exclusion can help reduce pathogenic bacteria, such as campylobacter,

Supporting technical information

Lactobacilli and bifidobacteria produce more lactic and acetic acids and less succinic, formic acids and ethanol, through the metabolisation of oligosaccharides (Van Der Meulen et al, 2004). Nazef et al (2008) found specifically that lactobacillus reuteri, isolated from a poultry faeces sample, exhibited anti-campylobacter activity.

bacteria of the lactobacilli and bifidobacteria genus can directly inhibit both growth and development of pathogenic bacteria through their natural metabolic activities. Fewer of these bacteria mean campylobacter can more easily grow.

Although other factors, such as diet, can also trigger a change in the gut microbiota, it is clear that the negative changes induced by stress can leave the birds more susceptible to infection. Reducing the stress levels in birds is therefore likely to help them regulate any infection.

Due to noradrenaline levels, microbiota changes, a combination of both, or a different mechanism entirely, it is clear that elevated stress levels have a detrimental effect on bird health. Regardless of the mechanism causing the increase in disease susceptibility in lower welfare birds, the outcome remains the same – the birds suffer. Further steps towards stress reduction in the majority of broilers could be taken by simply adopting the Better Chicken Commitment requirements.



Illustration of campylobacter bacteria

Breed responses

Humphrey et al (2014) demonstrated that there is no difference in campylobacter susceptibility between faster growing and slower growing broilers under the same experimental conditions. They did however show that the different breeds exhibit different physiological responses to the infection. By using the same environmental conditions for all breeds, it could be demonstrated that these particular results were due to breed differences and not other environmental factors. The specific breeds were not mentioned, however Table 1 gives a good indication. As expected, the inflammatory response in the faster growing breed was both more prolonged and more severe.

Table 1:

Breed group	Breed slaughter age (days)
A1	35
Control	36
A2	39
B1	48
B2	56



JA757 slower growing chicken breed

Supporting technical information

All four of the test groups expressed three types of cytokines associated with the inflammatory response (CXCLi1, CXCLi2 and IL-1 β) two days post oral infection with *C. jejuni*. Five days post infection these mediators were reduced in all but breed A1 in which they remained significantly higher than the other breeds. By 12 days post infection CXCLi2 levels continued to remain significantly higher in the A1 birds, although IL-1 β expression had reduced and there was no significant difference in CXCLi1 levels between breeds. In contrast to this elevated response in the faster growing birds, breed B2 had significantly higher levels of interleukin-10 (IL-10) at 12 days post infection. IL-10 is an inflammation feedback negative regulator that plays an important role in the regulation of the inflammatory response, and therefore the regulation of *C. jejuni*. The low levels of IL-10 in the faster growing birds could be the cause of the prolonged elevated inflammatory response, and lack of regulation in the faster growing breed.



Both the A1 and B2 groups showed morphological evidence of intestinal mucosa damage through inflammatory infiltration as a result of the campylobacter infection. However, the damage was not only more common, but more intense in the A1 group with 7/10 birds affected compared to 3/10, respectively. One result of the prolonged inflammatory response in the A1 birds was that they were found to have diarrhoea when welfare checks were conducted. None of the other groups did other than the infected control group, although the immune responses of this group were not monitored. With the close slaughter age between the A1 group and the control group, this is not unexpected. This physiological response seems to be due to a poorly regulated immune response in the faster growing birds, with the diarrhoea then leading to further welfare issues. Aside from the slaughter age (and hence growth rate), other welfare differences were also evident across the groups even though all birds were kept under the same experimental conditions with regular litter changes. 9/10 of the infected A1 birds showed signs of pododermatitis at 12 days post infection, whereas group B2 showed no signs. This is likely due to a combination of the diarrhoea in the A1 group, creating wet and infected litter, and the behavioural differences between faster growing and slower growing breeds, as shown by Dixon (2020), with faster growing breeds spending more time sitting in the wet infected litter. This falls in line with the findings in the stress related studies (see page 8).

The differences in immune responses seen in different breeds is further supported by a briefing conducted on Dutch slower growing broilers by Compassion In World Farming (2020). The data analysis showed that slower growing birds are three times less likely to require antibiotic intervention from illness. Even more significant, previous Dutch figures from 2015 showed that antibiotic usage in conventional faster growing breeds was more than five times higher than antibiotic levels used on slower growing breeds (Alliance to Save Our Antibiotics, 2016). While there may be a number of factors affecting the level of antibiotic usage on any one farm, this information adds weight to the view that slower growing birds are better able to regulate immune responses, and are therefore less susceptible to disease.

This demonstrates multiple positive factors associated with using slower growing broilers with regards to campylobacter. Lower welfare, faster growing birds were shown to not only have a more severe inflammatory immune response, resulting in poorer health, but to exhibit other poorer welfare outcomes in the form of pododermatitis. A switch to a slower growing breed would not only reduce physical suffering, but as they are better able to manage infections this may also further reduce overall antibiotic usage and thus reduce the risk of antibiotic resistance developing.

Environmental

While previous research has shown that breed has a significant impact on campylobacter-associated problems in chickens, breed alone is not the only factor at play as multiple environmental factors can also have an impact. These include litter quality, the thinning process, and post-slaughter control points affecting campylobacter colonisation.

During the Dixon (2020) study comparing the welfare of faster growing and slower growing broiler breeds, litter (wood shavings) was replenished as and when needed, as it would be on a commercial farm to maintain it in a dry and friable state. It was noted that faster growing birds needed shavings replenishing three to five times during the trial, resulting in 24kg extra per pen. Slower growing birds only needed shavings replenishing two to three times, resulting in only 14kg extra per pen. This shows that even without campylobacter as a contributing factor, faster growing breeds induce lower quality litter. With the added diarrhoea caused by campylobacter in faster growing breeds (as shown in Humphrey et al, 2014), litter quality would reduce even further and faster, inevitably leading to higher levels of hock burn and pododermatitis. Within the same study (Dixon, 2020) slower growing birds had a significantly higher proportion of lower (better) breast cleanliness scores than the faster growing

breeds. This is due to the slower growing birds spending less time feeding, drinking and sitting than the other breeds and more time standing, in locomotion, foraging, preening, dustbathing and perching. It is likely that the significant increase in wood shavings used for the faster growing birds is due to these behavioural differences, with the faster growing birds spending significantly more time sitting in the shavings, mixed with their own faeces. This is further backed up by the poor cleanliness scores in the faster growing breeds. An interesting side note to this is that with slower growing birds using less litter as standard, and less likely to get diarrhoea from infection, a switch to slower growing birds would not only save money on litter in the long run but also help contribute to sustainability goals.

Birds that are colonised with campylobacter will typically excrete up to $\log_{10} 8$ cfu of *C. jejuni* per gram of faecal matter (Line, 2006). This means

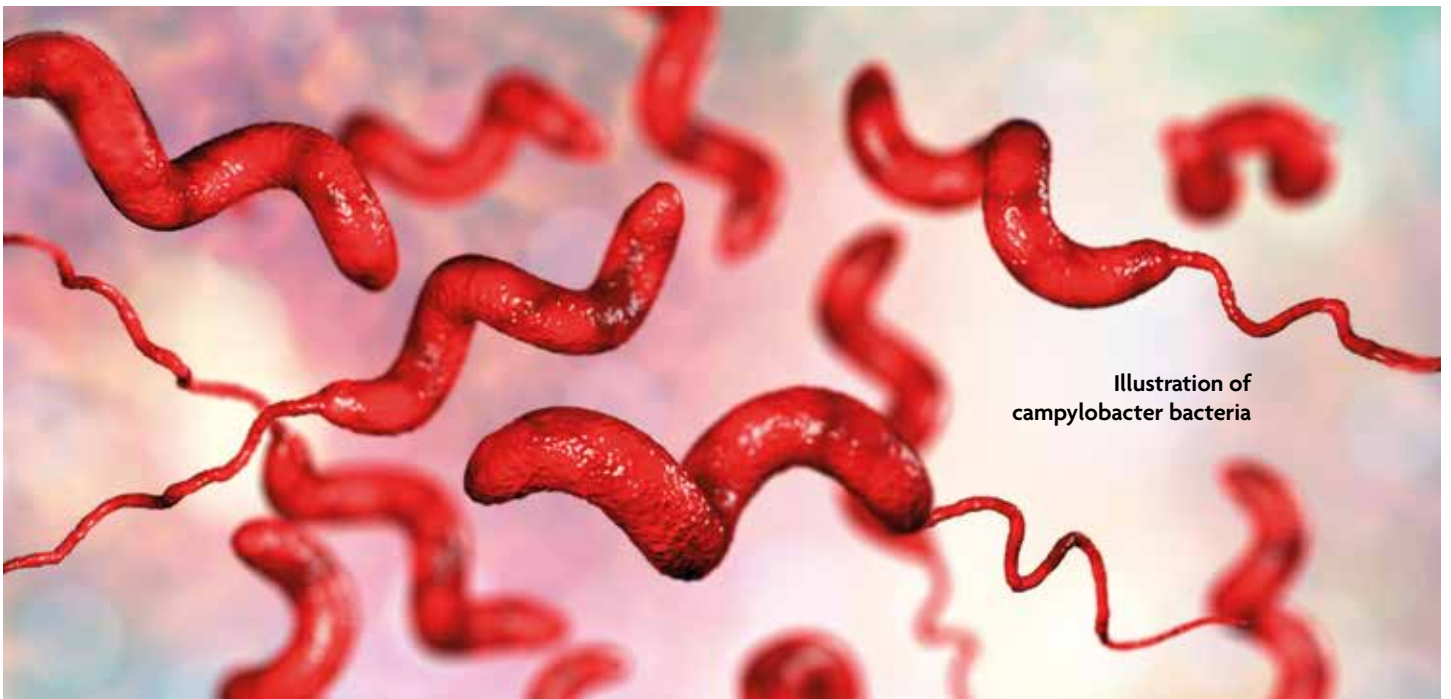


Illustration of campylobacter bacteria

that contaminated faeces will contain a high enough inoculum dose to infect other birds that consume them. As faster-growing birds spend more time sitting, they will have prolonged physical contact with the infected litter, just one possible explanation for increased hock burn and pododermatitis in infected flocks. The lack of ability to express natural behaviour in the birds is also likely to increase stress levels, making them even more susceptible to the infected litter they are forced to sit in.

Flock thinning is the planned early removal of a proportion of birds from a house for slaughter, usually to maintain a required stocking density. Thinning is not only a stressful process for the birds, it can significantly compromise biosecurity. For this reason thinning is discouraged by the Better Chicken Commitment, however one thin per flock is permitted. In contrast to this, RSPCA Standards do not permit thinning at all. Ramabu et al (2004) identified multiple sources of campylobacter introduction to flocks, which would be likely to occur during the thinning process. 209 samples were taken from a variety of fomites; drivers' and catchers' boots, truck beds, forklifts and tractors, truck wheels, crates and pallets. These samples were taken after cleaning, but before departing to the farm for the thin. Of the samples taken 53 percent were positive for *C. jejuni* with tractor wheels being the only sampling point with no positive results. Samples were taken at different times throughout the day, but there were no significant differences in the results. These results demonstrate how the thinning process can be a significant risk factor for campylobacter infection within a flock at farm level, with all fomites other than tractor wheels being a potential source of transmission. This data is further backed up by Bull et al (2008) who demonstrated a secondary finding that 65 percent of the GB reared flocks from their study were able to remain campylobacter free until the first thin. It is unrealistic to assume campylobacter can be completely eradicated at farm level, but it could be managed better. Thinning is stressful and a potential biosecurity risk, and while higher welfare birds are better able to manage infection than current faster growing breeds, the best long-term solution would be to avoid thinning in all flocks both for biosecurity and bird welfare reasons.

...elevated stress levels have a detrimental effect on bird health... fast-growing birds suffer more physically – through inflammation and hock and feet burns...

Although campylobacter control and prevention measures are present throughout the whole production process, the colonisation of the chickens pre-slaughter is the most significant factor known to affect the campylobacter counts on the carcass post production (Public Health England, Jorgensen et al, 2015). When birds are not colonised at slaughter, campylobacter detection is either very low or not detected on the chicken carcass at all (Allen, 2007. As cited in Public Health England, Jorgensen et al, 2015). A European Food Safety Authority (EFSA) (2010) study found that broilers colonised with campylobacter before slaughter were around 30 times more likely to produce a campylobacter positive carcass when compared to chickens not colonised before slaughter. This suggests that other potential contamination sources, such as factory machinery for example, are not likely to be a primary source of contamination/infection, though as outlined above, one of the most common causes of infection is thought to be introduced on the farm during the thinning process. With this in mind, reducing inoculation and spread at the farm level will help to reduce the levels of campylobacter reaching the consumer and should therefore be a priority for future changes in farming practices.



Antibiotic resistance

Finally, and perhaps most importantly, as well as the links between antibiotic usage and breeds previously mentioned, there are some extremely concerning links between antibiotic usage and campylobacter itself, with past experiences having shown us how the over usage of antibiotics in animal husbandry is highly likely to impact humans. In 2014 there was a record high of 49 percent fluoroquinolone resistance in campylobacter species isolated from retail poultry meat (Jorgensen et al, 2016). This was followed by a record high in 2015 of human *C. jejuni* resistance at 48 percent (Public Health England as cited in Alliance to Save Our Antibiotics, 2016). The increased fluoroquinolone resistance in human campylobacter is strongly linked to these antibiotics being used in poultry production and the resistance that followed (Alliance to Save Our Antibiotics, 2016). While it is unclear what impact this has had directly on human medicine it is worrying and is a potential risk, for example when considering the ability of bacteria to transfer resistance genes to other bacterial species. Learnings were taken away from this with this particular class of antibiotics now rarely used in poultry, but the risk of resistance developing in campylobacter to the antibiotic classes that are being used in broilers remains. There is no argument that the irresponsible usage of any antibiotic will result in resistance developing (WHO, 2020), and this resistance, when seen in human infections, will further reduce the efficacy of these antibiotics for humans.

Tetracyclines and penicillins are now the two most common classes of antibiotics currently used in poultry (UK-VARSS, 2020). Between 2013 and 2017, the amount of campylobacter species isolated from humans increased resistance to tetracycline antibiotics, from 33 percent resistance to 39 percent. Interestingly, isolates taken from retail chickens 2014–15 also showed significant resistance to tetracyclines, with 63 percent of *C. jejuni* showing resistance (Jorgensen et al, 2016). This could be following the same path as fluoroquinolones. The most concerning part is that between 2014 and 2019, tetracycline use had dropped by 87 percent, but resistance in campylobacter species seems to have still increased during that time. This strongly suggests that while there is a direct link between antibiotic usage and resistance developing (Who, 2020), this problem may not be easily reversible.

A very recent FSA surveillance research project on human campylobacter infections in the UK has highlighted a number of poultry-related concerns, including the confirmation of a sustained contribution of poultry to human campylobacter infections in England (FSA, 2021). As well as source attribution, the project that ran from 2015 to 2019 also analysed antibiotic resistance. The project identified fluoroquinolone resistance in *C. jejuni* from retail chickens was 52.4 percent, which tops the Jorgensen et al (2016), at the time record high, findings of 49 percent. What is more concerning is that tetracycline resistance in *C. jejuni* from retail chickens was even higher at 60.6 percent. As previously established, tetracyclines are still commonly used in poultry production, so when evaluating this recent finding along with previous findings, have tetracyclines already passed the point of no return?

It must be acknowledged that significant work has been done to reduce antibiotic usage in the chicken industry, with an overall reduction of 64 percent between 2014 and 2019 (UK-VARSS, 2020). However, there was a small spike towards the end of that timeframe with a 7.6mg/kg increase between 2017 and 2019. While targets for the next four years are to maintain the current usage of antibiotics, rather than reducing them further (RUMA, 2020), it is clear that switching to higher welfare slower growing birds, that seem to require fewer antibiotics, can only serve to improve the approach to antibiotic usage and help ensure these targets are not exceeded. This is agreed with by the WHO (2020) that states the agricultural sector can control and prevent antimicrobial resistance by preventing infections through improved hygiene and animal welfare. While new EU antibiotic regulations came into force in January 2022 prohibiting the routine use of antibiotics in farming, these will unfortunately not apply to Great Britain. Reductions in antibiotic usage within the poultry sector will reduce the rate at which resistance is developing in bacteria such as campylobacter, which can then infect humans and may have an impact, directly or indirectly, on the effectiveness of important antibiotics. It is of vital importance that all help maintain the efficacy of the antibiotics that are still effective. While the initial antibiotic reduction was a great success, it is now that the challenge really begins. If antibiotic usage in broilers can safely be further reduced, without compromising their health and welfare, and potentially through improving health and welfare, there is surely a responsibility to do so.

Chicken breast quality control assessment



Conclusion

It is clear that the idea of campylobacter being commensal has become outdated. Perhaps it was in fact previously the case and many years of selective breeding and intensification in the industry have damaged this relationship. Either way, we can see that campylobacter now affects both faster growing and slower growing broilers in multiple ways, with faster growing birds significantly more susceptible to the negative consequences of infection. Stress has been shown to compromise the broiler immune system in multiple ways. This includes increased noradrenaline levels and a shift in the gut microbiota composition. As stress is more prevalent in low welfare birds, they inevitably are more susceptible to disease, including campylobacter infection. The combination of stress and campylobacter results in more virulent, tissue infiltrating bacteria that could increase the risk of human infection.

Faster growing broilers have also been shown to be unable to regulate the inflammatory response associated with campylobacter inoculation, the likely cause behind the more severe intestinal damage and diarrhoea seen. This is backed up by one report suggesting that slower growing breeds could be up to five times less likely to require antibiotic intervention from illness.

Under standard conditions faster growing birds require significantly more litter, meaning infected faster growing birds with diarrhoea would require even more. Faster growing birds also spend significantly more time sitting on wet litter, reflected in breast cleanliness scores, and are likely more stressed due to the inability to perform natural behaviours. Birds are then subjected to the thinning process, which poses serious risk to both welfare and biosecurity, and provides an opportunity for the introduction of campylobacter to the already stressed flock.

It has been repeatedly shown that in campylobacter infected flocks, more birds develop hock burn and pododermatitis compared to birds that are not infected regardless of breed growth rate. It has also been shown that when comparing faster and slower growing birds this incidence is significantly higher in faster growing breeds. This is likely due to a combination of multiple factors such as stress, an unregulated inflammatory response, and being forced to sit in the wet infected litter, as well as potentially being unable to divert adequate protein to repair tissue damage and sustain an immune response due to fast muscle growth rate. While biosecurity and other control interventions are in place to minimise campylobacter throughout the production cycle, it is unrealistic to believe that campylobacter on farms will be completely

eradicated any time soon. Although these control measures are constantly evolving to increase effectiveness, the most impactful action would be to move to slower growing breeds that are better able to manage campylobacter infection, require less antibiotics, and will suffer significantly less because of it.

Complete transition of the chicken industry from the current minimum legislation to the Better Chicken Commitment minimum requirements may not only lead to a reduction in campylobacter prevalence across the whole industry, this in turn may ultimately be reflected in a reduction of foodborne related illness across the human population. The relative EU risk of human campylobacteriosis through broiler meat could be reduced by up to 58 percent with only a 3-log₁₀ reduction in caecal campylobacter concentrations in broilers (EFSA Panel on Biological Hazards, 2020). Hence, the switch to higher welfare chicken would not only benefit the birds themselves with a better quality of life, but would also benefit the industry and consumers alike.

Due to the vast scope of this subject, there are a multitude of further research avenues to further explore. However, at this stage, our findings strongly suggest that campylobacter is no longer commensal and faster growing, lower welfare broilers suffer these consequences. The evidence also shows that antibiotic usage in the poultry sector requires further urgent attention and needs to be addressed now since development of resistance may not be easily reversible and may have implications for human medicine. Adopting the Better Chicken Commitment will not only reduce suffering, and also help sustain future antibiotic efficacy, it will undoubtedly slow the clock on this chicken timebomb.

JA757 slower growing chicken breed



Glossary

Bifidobacteria	A major genera of bacteria that inhabits the gastrointestinal tracts of mammals
CFU/g	Colony forming units per gram
Competitive exclusion	When multiple species compete for the same resources causing one species to be eliminated
Corticosterone	A hormone involved in stress responses
Cytokines	Molecules that help regulate inflammation
Fluoroquinolones	A class of antibiotics
Fomites	Objects or materials likely to carry infection
Gastrointestinal disease	A disease that affects the GI tract (mouth to anus), for example, food poisoning
Hock burn	Burns on the lower leg (hock), usually caused by prolonged contact with moisture and ammonia
Inoculum dose	A dosage of bacteria able to cause disease
Lactobacilli	A major genera of bacteria that inhabits the gastrointestinal tracts of mammals
Microbiota	The microorganisms that make up a particular site, e.g. the gut
Neurotransmitter	A signalling molecule released by a nerve fibre, triggering another nerve, a muscle, or other structure
Noradrenaline	A type of neurotransmitter
Oligosaccharides	Carbohydrates made up from three to six simple sugars
Penicillins	A class of antibiotics
Pododermatitis/Digital dermatitis	Burns on the foot, usually caused by prolonged contact with moisture and ammonia
Tetracyclines	A class of antibiotics
Thinning	Removal of a proportion of birds from a flock at a lower weight part way through the production cycle, leaving the others to grow on to a higher weight



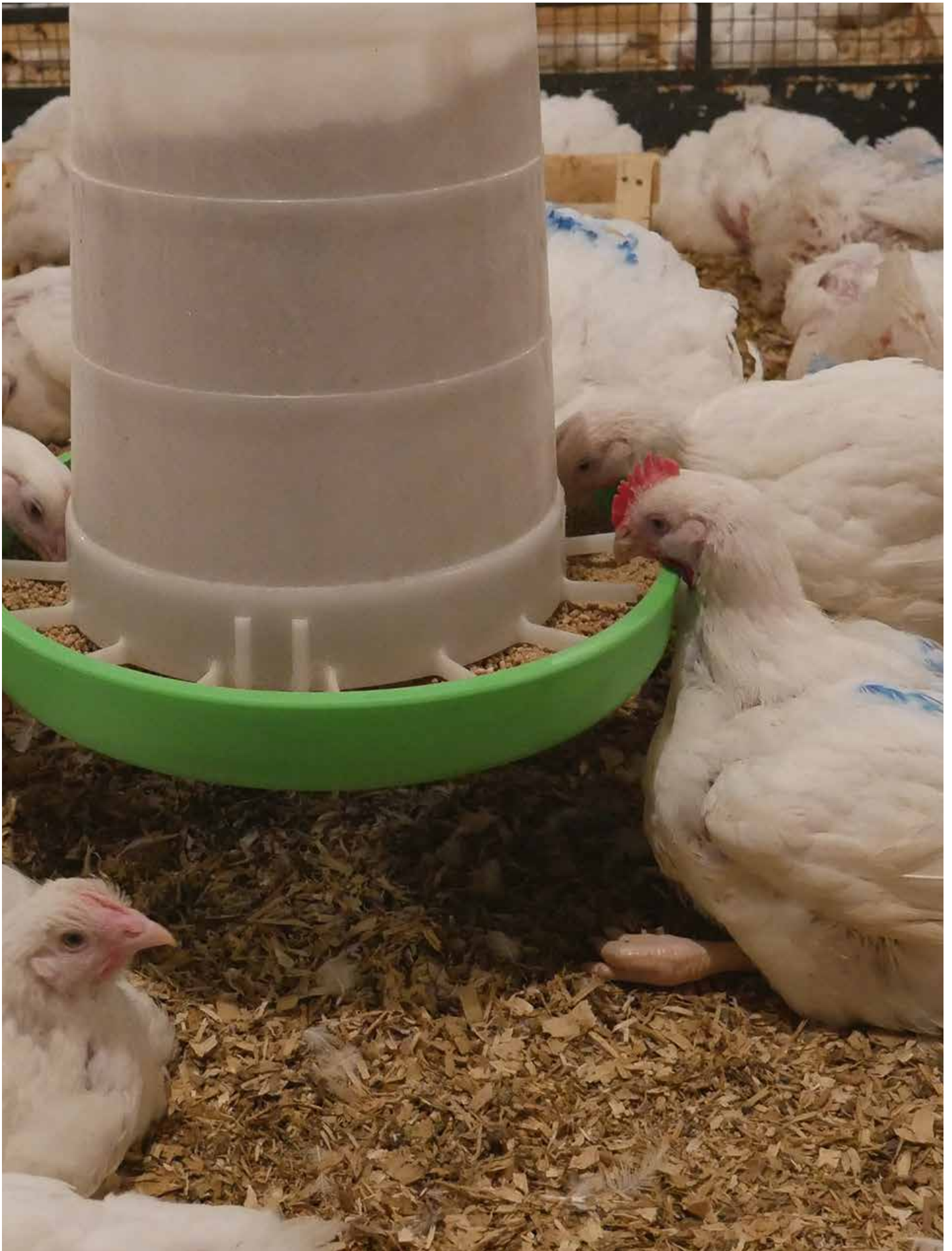
Illustration of campylobacter bacteria in the human gut

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