

EARLY VIEW

RESEARCH PAPER

Effect of Genotype and Feed Restriction on Productivity and Enteric Resilience Indicators in Commercial Chickens under Controlled Coccidial Challenge

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Abstract

Enteric stress impairs poultry productivity, but the magnitude and direction of the response depend on host genotype and nutritional status. This study evaluated the effects of genotype and feed restriction on productivity and enteric resilience indicators in commercial chickens using a controlled coccidial challenge. Ross 308 broilers, Hy-Line Brown pullets, and H&N Nick Brown layers were assigned to ad libitum or restricted feeding (10% quantitative reduction) and challenged or not challenged with a 10× dose of Paracox[®]. Body weight, live weight gain (LWG), egg production, feed conversion ratio (FCR), lesion score, and faecal oocyst output were measured. The coccidial challenge did not produce significant differences between treatment groups. Feed restriction, on the other hand, had clear genotype-dependent effects. It reduced body weight across all genotypes, lowered LWG in broilers, reduced total egg weight in layers, improved FCR in layers but worsened it in broilers. Broilers showed higher lesion scores and greater oocyst output than pullets and layers. The results indicate that genotype and nutritional plane explained more phenotypic variation than the applied challenge, supporting a genotype-dependent interpretation of productivity and enteric resilience in commercial chickens.

Introduction

Enteric disorders remain a major constraint in poultry production because they compromise intestinal integrity, impair nutrient utilization, and reduce productive efficiency. The biological consequences of enteric challenge are unlikely to depend on pathogen exposure alone; rather, they are shaped by the interaction between infectious pressure, nutritional status, and host genetic background. Variation in enteric resilience across poultry populations should therefore be understood as a function of the genotype-dependent capacity to maintain performance under intestinal stress (Klasing, 1998; Kau *et al.*, 2011; Belkaid and Hand, 2014). Coccidiosis, caused by protozoan parasites of the genus *Eimeria*, remains one of the most economically important enteric diseases in poultry worldwide

(Pellerdy, 1974; Conway and McKenzie, 2007; Quiroz-Castañeda and Dantán-González, 2015). Following ingestion of sporulated oocysts, invasive stages colonize the intestinal epithelium, damage mucosal tissues, and compromise nutrient absorption. The severity of the resulting disease depends on the infecting species, dose, age of the bird, and prior exposure history (Long *et al.*, 1976). Host susceptibility, lesion severity, and immune responsiveness may also differ according to genetic constitution (Caron *et al.*, 1997; Vervelde *et al.*, 1996; Smith *et al.*, 1994), and host genotype has been implicated in shaping gut-associated biological traits including intestinal microbial composition (Zhao *et al.*, 2013). Together, these observations support the view that resilience to enteric stress is a phenotype that

emerges from the interaction between challenge conditions and host genotype.

Commercial chickens are a useful system for examining genotype-dependent responses. Broiler and layer genotypes have been shaped by very different selection histories. Broiler lines have been selected for rapid growth and muscle accretion, whereas layer lines have been selected for sustained reproductive output and egg mass. These divergent genetic trajectories are likely to influence nutrient partitioning among growth, reproduction, maintenance, and host defence (Wright *et al.*, 2011; Buzala *et al.*, 2015). Thus, differences between broiler- and layer-type chickens are not just production-class labels but reflect distinct biological strategies for resource allocations.

Nutritional status is an important modifier of these genotype-dependent responses. When nutrient availability becomes limiting, trade-offs between productive output and physiological maintenance become more visible, and the direction of these trade-offs differs among genotypes with distinct metabolic priorities (Klasing, 1998). Feed restriction has been shown to influence growth, gastrointestinal development, and feed efficiency in poultry, with effects that vary according to genotype, degree of restriction, and production stage (De Jong *et al.*, 2002; Athar *et al.*, 2006; Bortoluzzi *et al.*, 2013; van der Klein *et al.*, 2017; Fondevila *et al.*, 2020; Trocino *et al.*, 2020). In laying hens, feed restriction can alter egg production and feed conversion dynamics (Olawuni *et al.*, 1992; Moreira *et al.*, 2012), whereas in broilers it has been associated with broader physiological and immune-related effects (Zulkifli *et al.*, 1993; Hangalapura *et al.*, 2005; Orso *et al.*, 2019). A quantitative reduction in feed supply should not be expected to produce a uniform biological response across genotypes with distinct selection backgrounds.

In the present study, three commercial chicken genotypes representing distinct production backgrounds—Ross 308 broilers, Hy-Line Brown pullets, and H&N Nick Brown layers—were evaluated under ad libitum or mildly restricted feeding, with or without a 10× Paracox challenge. The objective was to determine whether commercial genotypes differ in productivity and enteric resilience phenotypes under contrasting nutritional planes, and whether these responses support a genotype-dependent pattern of resilience to enteric stress.

Materials and Methods

Ethical approval

All experimental procedures were conducted in accordance with the UK Home Office Animals (Scientific Procedures) Act 1986 and approved by SRUC Animal Welfare and Ethical Review Body (approval no. AU AE 01-2018). The experiment was designed with consideration of reduction, replacement, and

refinement principles. Birds were monitored at least twice daily, and all euthanasia was carried out humanely.

Birds, housing and feeding

Three commercial chicken genotypes were used: Hy-Line Brown pullets (14 weeks of age), Ross 308 broilers (1 day of age), and H&N Nick Brown layers (22 weeks of age). For each genotype, 72 birds were obtained from commercial suppliers in the UK (JSR Services, Scotland; PD Hook, England), giving a total of 216 birds. Birds were housed in groups of six per pen. Diets were formulated according to the relevant commercial management guides (Aviagen, 2014; Hy-Line International, 2015). All diets were coccidiostat-free and obtained from a commercial feed supplier (Target Feeds, UK). All birds had ad libitum access to water throughout the experimental period.

Experimental design and treatments

The experiment followed a 2 × 2 factorial design with three replicate pens per treatment and six birds per pen within each genotype. Two experimental factors were applied: nutritional plane (ad libitum or 10% quantitative restriction relative to breed-specified daily intake) and coccidial challenge status. For the Hy-Line Brown pullets, the 10% restriction was calculated based on the standard guideline intake of 68–73 g/bird/day, resulting in an allowance of approximately 61–65 g/bird/day. Exact daily ad libitum feed consumption for this specific group was not continuously quantified. At day 28 of the study, challenged birds received 1 mL of Paracox® (MSD Animal Health) by oral gavage at 10× the recommended dose. Non-challenged controls received 1 mL of distilled water. The Paracox vaccine contains live sporulated oocysts of *Eimeria acervulina*, *E. brunetti*, *E. maxima*, *E. mitis*, *E. necatrix*, *E. praecox*, and *E. tenella*.

Measurements and sample collection

Individual body weight was recorded at day 0, day 28, and day 35. Live weight gain was calculated over the relevant intervals. Feed conversion ratio (FCR) was determined from feed intake and productive output. Faecal samples were collected on days 28 and 35 for oocyst counts. On day 28, two birds per pen were euthanized by electrical stunning followed by exsanguination; all remaining birds were euthanized at day 35. Intestinal lesions were scored from 0 (no lesions) to 4 (severe lesions) following Johnson and Reid (1970).

Statistical analysis

Data were analysed by ANOVA using GenStat version 17.1.0.14713 (VSN International, 2011). Statistical significance was declared at $P < 0.05$. Main effects of nutritional plane, challenge status, and genotype were evaluated together with two-factor

interactions (feeding × genotype, feeding × challenge, genotype × challenge). Power calculations indicated that the number of replicates used was sufficient to detect a treatment difference of 1.000 with 80% power at the 5% significance level.

Results

Overview of treatment effects

Across the measured traits, the 10 × Paracox challenge did not produce significant differences between challenged and non-challenged birds ($P > 0.05$). In contrast, feed restriction had clear effects on productivity-related traits, and these effects varied across commercial genotypes. Genotype-related differences were also evident for lesion score and faecal oocyst output, with broilers showing greater enteric responses than the pullet and layer groups ($P < 0.05$).

Effects of feeding regimen and genotype on body weight

Body weight was significantly affected by feeding regimen, whereas challenge status had no influence on this trait ($P > 0.05$). For pullets, body weight did not differ between feeding groups at day 0, but by day 28 ad libitum-fed birds were significantly heavier than restricted birds, and this difference persisted through day 35 ($P < 0.05$). The feeding effect was significant in layers only at day 28 ($P < 0.05$). Broilers on ad libitum feed were consistently heavier than restricted birds throughout the study ($P < 0.05$). Mean body weight values across genotypes and feeding treatments are presented in Table 1.

Effects of feed restriction on LWG, egg production, and FCR across genotypes

Feed restriction significantly reduced broiler LWG between days 28 and 35 ($P < 0.05$), while challenge status had no effect ($P > 0.05$). Total egg weight in layers was lower under restriction ($P < 0.05$) and was not influenced by challenge ($P > 0.05$). The effect of feeding on FCR differed between genotypes. Restricted layers had better FCR than ad libitum layers (2.03 vs. 2.80), but the opposite was true for broilers, where restriction worsened FCR (1.71 vs. 1.35; $P < 0.05$). Challenge status did not affect FCR in either genotype ($P > 0.05$). Summary data are presented in Table 2.

Lesion score and oocyst output across genotypes

Neither lesion scores nor oocyst output differed significantly between challenged and non-challenged birds, nor were they affected by the feeding regimen ($P > 0.05$). No intestinal lesions were detected in pullet or layer groups regardless of whether they were fed ad libitum or restricted diets. Broilers, however, showed lesions primarily in the duodenum, followed by the jejunum and ileum. Faecal oocyst counts were also

highest in broilers ($P < 0.05$), confirming a stronger detectable enteric response in this genotype. Detailed values are presented in Table 3.

Discussion

The principal finding of this study was that the 10× Paracox challenge failed to generate meaningful separation between challenged and non-challenged groups for any of the measured traits. This was unexpected. It suggests that the biological intensity of the challenge was too low to induce a clear response under the conditions tested. The present results therefore do not provide strong evidence of differential genotype-challenge interactions; rather, they point to a challenge framework that was too mild to reveal such differences.

Nevertheless, genotype-related differences in lesion scores and oocyst output remain informative. No lesions were observed in pullet or layer groups, whereas broilers showed visible lesions, particularly in the duodenum, together with substantially higher oocyst shedding. This is in line with reports that younger birds are more susceptible to *Eimeria* infection, while older birds may show reduced lesion development due to age-related or prior-acquired immunity (Pellerdy, 1974; Levine, 1985; Bachaya *et al.*, 2012). Age is a confound here. The broilers in our experiment were considerably younger than pullets and layers, and this likely contributed to the stronger enteric response. Still, within the framework of this study, the result demonstrates that commercially distinct chicken genotypes did not respond uniformly under identical enteric challenge conditions.

The productivity data provide stronger support for a genotype-dependent interpretation. Feed restriction reduced body weight across all three genotypes, but the expression of this effect differed by genotype and productive state. A 10% reduction in feed supply produced measurable decreases in body weight and LWG in broilers, which is consistent with earlier findings on the growth sensitivity of modern broiler genotypes to restricted nutrient intake (Athar *et al.*, 2006; Bortoluzzi *et al.*, 2013). The layer response told a different story. Feed restriction reduced total egg weight yet improved FCR—an opposing pattern to the FCR worsening seen in broilers. This divergence strongly suggests that the biological consequences of nutrient limitation are shaped by the productive priorities encoded within each genotype rather than by feeding level alone.

Such divergent responses can be interpreted in the context of the contrasting selection histories underlying commercial broiler and layer genotypes. Broilers have been optimised for rapid tissue deposition, where even a small reduction in nutrient supply can disrupt growth trajectories and worsen feed efficiency. Layers, in contrast, have been selected for sustained reproductive output, and may display a different pattern of nutrient partitioning under restriction—maintaining egg output with reduced overall feed intake, thus yielding a numerically better FCR. The opposing FCR responses observed between these genotypes provide direct evidence that

commercial genotype is a major determinant of how birds partition resources under nutritional stress.

This study also has applied relevance for low-input and resource-constrained poultry systems, where birds may routinely encounter suboptimal nutrition alongside endemic enteric pathogens. Under such conditions, genotype-associated differences in the capacity to sustain production may have practical significance. While no direct molecular mechanism was investigated here, the data clearly show that commercially distinct chicken genotypes diverge in measurable productivity and enteric response indicators under mild feed restriction, supporting the view that host genetic background should be considered when evaluating resilience to nutritional and enteric stressors.

Several limitations should be noted. The challenge model was not strong enough to generate clear treatment separation, which limits what we can say about direct genotype-challenge interactions. The three genotypes also differed in age and physiological state, so genotype effects are confounded with these variables to some extent. We tried to account for this by choosing genotypes that represent commercially relevant production categories, where age and physiological status are inherent to the biological context. It is also possible that feed intake in the ad libitum groups did not fully match expected commercial values, which would narrow the effective contrast between feeding treatments. Future work should use a stronger challenge model while controlling more tightly for age and physiological stage. Including direct measures of intestinal integrity, immune activity, or gut microbial composition would add mechanistic depth that the current phenotypic dataset cannot provide.

Conclusions

Genotype and nutritional plane were more informative determinants of phenotypic variation than the applied 10× Paracox challenge under the conditions tested. Feed restriction produced consistent effects on body weight, LWG, egg production, and FCR, but these responses were not uniform across commercial genotypes. Broilers showed greater reductions in growth-related traits and higher lesion scores and oocyst output, whereas layers exhibited reduced egg production alongside improved FCR under restriction. The results support a genotype-dependent interpretation of productivity and enteric resilience phenotypes in commercial chickens and underline the importance of considering host genetic background when evaluating nutritional and enteric stress responses in poultry.

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Table 1. Mean body weight (g) of three commercial chicken genotypes under ad libitum and restricted feeding regimens

Genotype	Day	Ad libitum (g)	Restricted (g)	P (Feed)	P (Challenge)
Hy-Line Brown (pullet)	0	1279	1295	> 0.05	> 0.05
	28	1651	1438	< 0.05	> 0.05
	35	1756	1475	< 0.05	> 0.05
H&N Nick Brown (layer)	0	1792	1758	> 0.05	> 0.05
	28	1832	1725	< 0.05	> 0.05
	35	1754	1731	> 0.05	> 0.05
Ross 308 (broiler)	0	40.33	39.50	< 0.05	> 0.05
	28	1635	1323	< 0.05	> 0.05
	35	2390	1986	< 0.05	> 0.05

Note: Pullet = Hy-Line Brown (14 wk.); Layer = H&N Nick Brown (22 wk.); Broiler = Ross 308 (1 d). Restricted = 10% quantitative reduction. Significance for feeding effect: $P < 0.05$ for all genotypes except layers at day 0 and day 35 ($P > 0.05$). Challenge had no significant effect ($P > 0.05$).

Table 2. Production parameters across commercial chicken genotypes by feeding regimen and challenge status (day 28–35)

Trait	Genotype	Feeding regimen		Challenge status		P (Feed)	P (Challenge)
		Ad lib.	Restricted	Challenged	Non-chall.		
LWG (g), d28–35	Broiler	755	662	716	702	< 0.05	> 0.05
Total egg wt. (g), d28–35	Layer	370	351	359	362	< 0.05	> 0.05
FCR	Layer	2.80	2.03	2.28	2.56	< 0.05	> 0.05
FCR	Broiler	1.35	1.71	1.56	1.50	< 0.05	> 0.05

Note: LWG = live weight gain; FCR = feed conversion ratio. Ad lib. = ad libitum feeding. Chall. = challenged with 10× Paracox®. Non-chall. = gavaged with distilled water.

Table 3. Lesion scores and faecal oocyst output (\log_{10}) by genotype, feeding regimen, and challenge status

Trait (Broiler only)	Feeding regimen		Challenge status		P (Feed)	P (Chall.)
	Ad lib.	Restricted	Challenged	Non-chall.		
Duodenal lesion score	2.43	2.33	2.56	2.21	> 0.05	> 0.05
Jejunum lesion score	2.21	1.92	2.17	1.96	> 0.05	> 0.05
Ileal lesion score	1.06	0.92	0.97	1.00	> 0.05	> 0.05
			Challenged	Non-chall.		
Oocyst count (\log_{10}) – Broiler	–	–	4.32 ^a	3.94 ^a	–	> 0.05
Oocyst count (\log_{10}) – Layer	–	–	2.23 ^b	2.37 ^b	–	> 0.05
Oocyst count (\log_{10}) – Pullet	–	–	2.24 ^b	2.40 ^b	–	> 0.05

Note: Lesion scores reported only for broilers; no lesions were detected in pullet or layer groups. Lesion scoring: 0 (no lesions) to 4 (severe), following Johnson and Reid (1970). Oocyst counts are post-challenge (day 35). a, b, means within the same column with different superscript letters differ significantly among genotypes ($P < 0.05$).