How Low Can You Go? Broiler Breast Muscle Possesses a Mitochondrial Content of Just 2%

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Short Report

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Abstract

In 1977 the *pectoralis major* (breast) muscle mitochondrial content of domestic broiler chickens, *Gallus gallus domesticus*, was estimated at 4.1%. This very low value is unsurprising in light of the bird's unique muscle fibre composition, being exclusively composed of low aerobic capacity type IIB fibres. However, in the subsequent ~ 45 years broilers have been further modified by sustained intensive genetic selection on feed conversion efficiency and breast muscle mass.

Objective

With a view to understanding the metabolic implications of this historic selection – and also laying a foundation to better interpret current commercial performance - we quantitated the cellular bioenergetic structure of modern broilers. The breast muscle of 4 64 d old Ross308 broilers was subject to morphometric analysis based on transverse Transmission Electron Microscopy (TEM) images.

Results

This approach determined an average mitochondrial content, diameter and circularity ratio of 2.1%, 0.42 µm and 0.72, respectively. Broiler breast mitochondrial content has thus approximately halved in the last 45 years, and represents one of the lowest contents recorded for the muscle of any eukaryotic species. We have provided an updated benchmark for interpreting broiler muscle biochemistry and reinforce the view that domestic chickens are unique models of minimal aerobic capacity.

Introduction

Meat producing chickens (broilers) are one of the globes primary sources of animal protein, possessing a dramatically enlarged *pectoralis major* (breast) muscle that comprises a quarter of the birds' total body mass (1). Moreover, modern birds have a reputation for extremely high feed conversion efficiencies, with some producers reporting in elite performers a wet weight gain of 1 kg for every 1.38 kg of dry feed consumed. This makes domestic chickens compelling models of animal performance. However, interest in the species is not restricted to the exceptional commercial capabilities of industrial birds. The chick is also considered a leading comparative model of the vertebrate muscle development program, particularly with respect to cell lineage tracking and patterns of morphogenesis (2).

Along with other avian meat producing species (such as turkeys, pheasants and quail) chickens are members of the Phasianidae taxonomic clade, the largest branch of the Galliformes (3). The members of this group tend to be sedentary, resident ground-dwelling birds that use short, burst flights to escape predators (3). This behaviour explains the functionally unusual breast muscle metabolism of the ancestral birds such as the Red Junglefowl (*Gallus gallus*) progenitor of modern domestic chickens. This ancestry - taken together with the impacts of domestication and subsequent selection - accounts for a muscle structure in modern birds that is dominated by explosive, fast twitch contractile isoforms, large diameter type IIB fibres and a very low oxidative capacity (3).

Recent work across a population of 80 industrial Cobb broilers has indicated variation in breast muscle mitochondrial content (as estimated by a proxy, mtDNA copy number) is significantly associated with growth performance phenotypes. For example, birds with a relatively low breast mtDNA copy number tend to be heavier, more muscular and have higher abdominal fat (4). It has also been argued that selection for higher feed efficiency may reduce tissue mitochondrial content through considerations of economic design (5). After all, it would be energetically wasteful to pay for unnecessary spare physiological capacity. Given that intensively reared domestic birds do not face the demands (e.g. escaping the threat of predation) of wild animals there is considerable scope for economising on physiological function. However, the only *bona fide* record of mitochondrial content for broilers we could find was from the late 1970's (6, 7), where the breast muscle of 2 Hydro Compact broilers was estimated to be 4.1%.

This raises a fundamental question. What has happened to the metabolism of broiler breast muscle in the intervening 45 years given numerous subsequent generations of intensive genetic selection on both muscle mass and feed efficiency? To answer this, we phenotyped the breast muscle of modern Ross308 Aviagen broilers using both a) traditional morphometric estimation of mitochondrial content from transverse TEM images and b) mtDNA copy number estimations based on a duplex taqman qRT-PCR assay using total DNA as the template. We hypothesised that the more muscular, more feed efficient modern broilers would have a lower breast muscle mitochondrial content when compared to the historic 1970's birds. In so doing we provide an updated benchmark for modern broilers, and briefly interpret our findings in the twin contexts of comparative physiology and animal production.

Methods

Animal Resource

Birds were purchased from 9Dorf Farms, a pasture raised poultry farm at Lilydale, QLD, at 50 d and grown to 64 d at the Poultry Unit on the Gatton campus of The University of Queensland. Prior to purchase these birds received mixed feed and had access to grass, comparable to free range birds. After purchase they were fed an industry relevant broiler starter diet (see Table 1 for nutritional details) followed by Ridley's Barrastock grower diet *ad libitum*. All birds were weighed at 64 d and euthanized by cervical dislocation. Skinless and deboned breast was mechanically separated from the carcass, weighed, and *Pectoralis major* necropsy samples were consistently taken from the same portion of the pectoral muscle as previously described (4), placed into heavy duty aluminium foil and snap frozen in liquid nitrogen, and stored at -80° C.

Table 1
The nutritional composition of the broiler starter diet

Ingredient composition (% by weight)	
Wheat	39
Sorghum	22
Tallow	3
Canola Oil	-
Canola Meal Solvent	-
Soybean Meal	29
Limestone	1.5
Salt	0.2
Sodium Bicarbonate	0.2
DL-Methionine	0.2
L-Lysine HCL	0.2
Bovatec 20CC	0.1
Myco Curb Liquid	0.3
MDCP	1
Chick/Pullet Pmx	-
Faba Beans	3
Poultry Starter Pmx	0.2
Xylanase	0.1
Nutrient composition (per unit mass of feed)	
Moisture	10.5
Protein	22
Fat	4.7
Fibre	3.3
ME kcal	2950
Calcium Ca	1
Phosphorus P	0.6

Ingredient composition (% by weight)	
Available Phosphorus	0.5
Salt NaCl	0.2
Lysine	1.2
Synthetic Lysine	0.1
Methionine	0.5
Methionine + Cystine	0.9
Threonine	0.8
Leucine	1.8
Isoleucine	0.9
Tryptophan	0.3
Arginine	1.4

TEM estimation of pectoralis major mitochondrial content

Breast tissue samples (1mm × 1mm cubes) from 4 individual birds were fixed overnight (for 24h total) in 2.5% glutaraldehyde, washed with PBS then stored under PBS at 4° C until ready for mounting and sectioning. They were mounted such that transverse cross sections could be taken with respect to the orientation of the muscle fibres and the associated mitochondrial network. The sections were visualised using a Jeol JEM-1011 electron microscope operated at 80kV. Images were captured with an Olympus Morada camera using the Olympus AnalySIS software within the Centre of Microscopy and Microanalysis at The University of Queensland. Morphometric analysis (mitochondrial content, mitochondrial diameter and circularity ratio) was performed after importing the images into ImageJ software.

In brief, 15 images were assessed for each of the 4 individuals. Planimetric analysis was used to determine mitochondrial content by quantitating the total area covered by the tubules of the mitochondria with respect to the total area of the image under investigation. For the quantitation of mitochondrial diameter, the longest and shortest diameters of each of 10 transverse tubules of the mitochondrial network were estimated per image. For the estimation of circularity, the relationship between the shortest and longest diameter of each tubule under consideration was expressed as a ratio.

qRT-PCR estimation of pectoralis major mtDNA copy number

The mtDNA copy number is an established proxy for mitochondrial content. Total DNA contains the compositional information such that the number of copies of haploid mtDNA can be compared to the

number of copies of a single copy gene from the diploid nuclear genome. Total DNA was extracted using Qiagen DNeasy Blood and tissue kits following the manufacturers' instructions.

The development and performance of the duplex taqman assay is described in another paper (4). In brief mtDNA copies were estimated by selective amplification of the mitochondrially encoded gene *ND2*. These values were compared to selective amplification of a single copy nuclear gene *MSTN*, with both reactions proceeding simultaneously in the same reaction vessel. We used the following formula to calculate mtDNA copies for every 2 nuclear genes based on the PCR cycles at which the fluorescence threshold was crossed.

$$mtDNAcopynumber = 2 * \left(2^{nDNACt - mtDNACt}\right)$$

Where nDNA Ct and mtDNA Ct are the PCR cycles at which the fluorescence threshold was crossed for the nuclear (*MSTN*) and mitochondrial (*ND2*) DNA segments, respectively.

Given chickens have a diploid nuclear genome this formula equates to a mtDNA copy number estimate expressed, in effect, on a 'per nucleus' basis. For most chicken tissues this approach is equivalent to expressing mtDNA copies on a 'per cell' basis. For the particular assessment of multinucleate muscle fibres it is best seen as representing a correction 'per unit volume of cytoplasm.'

We ran all assays on a Qiagen Rotorgene system using 20 µl total reaction volumes, Thermofisher's Fast Advanced mastermix and PCR cycling parameters (95°C denaturation for 20 seconds followed by 40 cycles of 95°C for 1 second and 60°C annealing for 20 seconds) recommended by Thermofisher. A given 20 µl reaction contained 10 µl 2x Fast Advanced mastermix, 8 µl ultrapure molecular grade water and 1 µl each of the mtDNA and nDNA primer / probe combinations. The fluorescence threshold was set manually at 0.05 and fixed across runs. No template controls were included for each run. Prior to quantitation all gDNA was standardised to a concentration of 1 ng / µl.

Statistical analysis

Summary statistics were calculated using Microsoft Excel and GraphPad Prism software version 9.

Results

TEM estimation of pectoralis major mitochondrial content

The summary data can be found in Table 2. A representative TEM image can be found in Fig. 1.

Table 2
The summary statistics for breast mitochondrial content, diameter and circularity ratio.

Individual	Mitochondrial content (%)[1]	Mitochondrial diameter (μm)[2]	Mitochondrial circularity (ratio)[2]
17	2.38 ± 0.19	0.44 ± 0.01	0.73 ± 0.02
27	2.22 ± 0.13	0.41 ± 0.01	0.73 ± 0.02
29	1.82 ± 0.10	0.44 ± 0.003	0.70 ± 0.003
32	1.79 ± 0.08	0.39 ± 0.01	0.70 ± 0.02
Average	2.05 ± 0.29	0.42 ± 0.02	0.72 ± 0.02

[1]Values represent the mean ± SD obtained from 15 images.

[2]Values represent the mean ± SD obtained from 10 transverse tubules in each of 15 images.

qRT-PCR estimation of pectoralis major mtDNA copy number

The mtDNA copy number values and associated bird phenotype data can be found in Table 3.

Table 3
The phenotype and mtDNA copy number data from the breast muscle of ten broilers.

Bird No.	Bird weight (g)	Breast muscle mass (g)	DNA yield (ng/μl)	DNA purity (A260/280)	mtDNA copy number per nucleus
1	2953	799	155.3	2.10	5023
2	3299	865	124.3	2.11	1463
3	3495	929	95.9	2.07	438
4	2930	740	86.8	2.08	1333
5	2832	871	97.8	2.08	917
6	3278	914	108.0	2.07	1237
7	3410	934	82.4	2.09	831
8	2720	778	121.2	2.10	1368
9	2850	960	105.7	2.12	1464
17	2871	792	120.9	2.12	734
Average ± SD	3064 ± 277	858 ± 77	109 ± 22	2.09 ± 0.02	1029 ± 394

Bird weight and breast muscle weight were positively correlated and approached significance at the 0.05 threshold (r = 0.57, *P* = 0.088). Breast muscle weight comprised an average of 28% of overall bird mass. The negative relationship between mtDNA copy number and bird weight was not significant here (r = -0.31, *P* = 0.39). For one individual (bird 17) we have both TEM estimates of mitochondrial content and qRT-PCR estimates of mtDNA copy number.

Discussion

Modern Ross308 broilers purchased in 2019 at 50 d age and raised on an industry relevant *ad libitum* diet to 64 d possess a breast muscle mitochondrial content of 2.05% (Table 2). This is half that of broilers phenotyped at 4.1% in the late 1970's (6, 7). This indicates that 45 years of additional genetic selection on muscle mass and feed efficiency has further reduced muscle aerobic capacity from what were already very low levels. It is possible some of the observed reduction is environmental in origin (e.g. diet, thermal regimen or activity levels). However, a partitioning analysis based on reciprocally feeding 1957 and 2001 broilers 1957 and 2001 diets concluded that numerous improvements in broiler performance are 85–90% attributable to genetic gain (8, 9). Moreover, given these birds were free-ranging prior to purchase at d 50 their muscle mitochondrial content would have, if anything, been stimulated by exercise compared to more intensively reared, more rapidly grown, heavier (~ 4 kg) industry birds. As such the low 2.05% estimate we have produced for these ~ 3 kg birds can be considered a conservative estimate. The true value for industry birds is likely even lower.

This combination of data from both TEM and qRT-PCR technologies taken from similar tissue samples now allows us to reassess the likely mitochondrial contents of other bird tissues for which only mtDNA copy number phenotypes (using the same qRT-PCR assay) exist (4). Building on our previous study on Cobb broilers (4) a revised breast muscle mitochondrial content of 2.05% (rather than the previously assumed 4%) suggests heart, drumstick and white fat have actual contents of 9.3%, 6.6% and 1.0%, respectively. These should be considered tentative predictions requiring independent validation by TEM, as the impact of tissue type on nuclear versus mitochondrial DNA extraction efficiency (and therefore the derived gDNA composition assayed by the molecular technology) remains unclear. Nevertheless, the predicted heart mitochondrial content value of 9.3% is dramatically lower than 10 differently sized mammals whose heart content has been typically measured to be between 24% (for larger mammals like dogs) – 37% (for smaller mammals like mice) (10).

The present finding of a relative reduction in breast muscle mitochondrial content in the modern more muscular and more feed efficient birds is entirely consistent with similar observations made in hyper-muscular, feed efficient breeds representing a number of mammalian production species. These include *MSTN* mutant cattle (11, 12), *MSTN* mutant sheep (13), Callipyge sheep (13–15), Large White pigs (16) and Yorkshire pigs (17). In those muscular, feed efficient breeds a muscle fibre composition shift favouring the low mitochondrial content Type IIB fibres has been repeatedly observed.

Indeed, an argument has been made that the gain in production efficiency is partially caused by the diminished tissue mitochondrial content (5) based on application of an economic design theory called symmorphosis (18). The logic runs as follows: it is inefficient to first construct (the cost of mitochondrial biogenesis) and then have to maintain (the cost of running the inner membrane proton gradient, *inter alia*) unnecessary bioenergetic capacity. The highly controlled environments we have created for intensively reared production animals, largely free of predation and other threats, open up the possibility of diminishing many aspects of physiological capacity in a quest for ever increasing gains in efficiency.

Nevertheless, the science relating aspects of the mitochondria (such as gene/protein expression) to feed efficiency is equivocal. For example, transcriptome analysis of the breast muscle of elite Cobb broilers differing in feed efficiency by 1.4 fold found that the most efficient birds expressed less myoglobin and less slow twitch contractile subunits (in line with expectation) but displayed higher expression of mRNA encoding mitochondrial proteins (contrary to expectation) (3). These basic findings were supported by an independent proteomics analysis performed on the same tissue samples suggesting they have been characterised correctly (19). One explanation is that abundance of mitochondrial proteins may not always have a simple relationship to mitochondrial content. After all, (20) found that more efficient Hereford x Angus steers had rumen tissue with *lower* mtDNA copy numbers but *higher* expression of mRNA encoding mitochondrial proteins. Data from multiple levels of biological organisation is probably desirable when interpreting mitochondrial function.

Where does a skeletal muscle mitochondrial content of just 2.05% sit in the broader context of life on earth? In fact, it is among the lowest measurements we could find for any muscle in any species. Intriguingly, it is at lower end of the estimations of 2.0 to 3.9% made for limb muscles of two wild cheetah, *Acinonyx jubatus*, the ultimate sprint adapted mammal (21). It is a staggering 17 fold lower than the 35.5% documented in the breast muscle of the highly athletic hummingbird, *Selaphorus rufus* (22). Indeed, the only lower estimates we could identify come from two large ruminants: the *semitendinosus* muscle in Zebu cattle, *Bos indicus* (1.1–1.3%), and the *longissimus dorsi* muscle in Eland, *Taurotragus oryx* (1.4%) (23). Given tissue mitochondrial content scales negatively with body mass e.g. (5) such that smaller animals have higher contents, the extremely low breast muscle content observed here in ~ 3 kg birds is even more striking that when taken at face value. Moreover, given the breast muscle in these birds is 28% of total body mass, the implications for the birds' overall bioenergetics are far greater than for the ruminant examples given.

One conclusion is certain. For a given muscle in every individual of every species, there must be a lower limit beyond which muscle mitochondrial content can no longer be further diminished. Beyond this metabolic 'point of no return,' sustainable aerobic ATP supply will be unable to satisfy basic cellular ATP demand and tissue damage will inevitably ensue. Even for low bioenergetic demand sedentary commercial broilers reared in highly controlled settings there are indications we may already be approaching that particular tipping point.

Concerning muscle pathologies have recently emerged, particularly noticeable in rapidly grown larger birds which we know are the very individuals possessing the lowest mitochondrial contents (4). These include ‘wooden breast’ (24) and ‘white striping,’ (25). These are conditions characterised by lipid infiltration, fibrosis and mitochondrial degeneration (26) in breast tissue - signs of a muscle system gone awry. Furthermore, evidence for hypoxia and lactic acidosis in afflicted birds (24, 25) indicates aerobic supply of ATP is not adequate to meet bioenergetic needs and that unsustainable use of anaerobic pathways of ATP generation have been resorted to. Other authors have indicated that a borderline inadequate capillary density in breast muscle is responsible (25), to which we would add the likely impact of a borderline inadequate mitochondrial content.

Taking this suite of comparative and production data together reinforces the notion that the modern broiler represents a remarkable and very extreme model of skeletal muscle function. We suggest that the broiler industry needs to be cognisant not to push the birds bioenergetic system too hard (i.e. too low) in the relentless quest for increasing feed efficiencies.

Limitations

This study is limited by the relatively small number of birds (n = 4 for TEM, n = 10 for qRT-PCR). The diet and behaviour of the birds prior to purchase was not quantitated in a controlled environment.

Declarations

Ethics approval and consent to participate

All experimental procedures, including animal handing and husbandries, were approved

by the Animal Ethics Committee of Research Ethics Unit of The University of

Queensland, AEC Approval Number 2018/AE000575. All methods were performed in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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No funding was sought for this research project.

Authors contributions

NJH conceived the study, managed the laboratory component of the project, contributed to the analysis of the data and wrote the first draft. EAS managed the live bird component of the project and organised tissue sample collection. WB provided conceptual insight into avian mitochondrial physiology. All authors read and approved the final manuscript.

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Figures

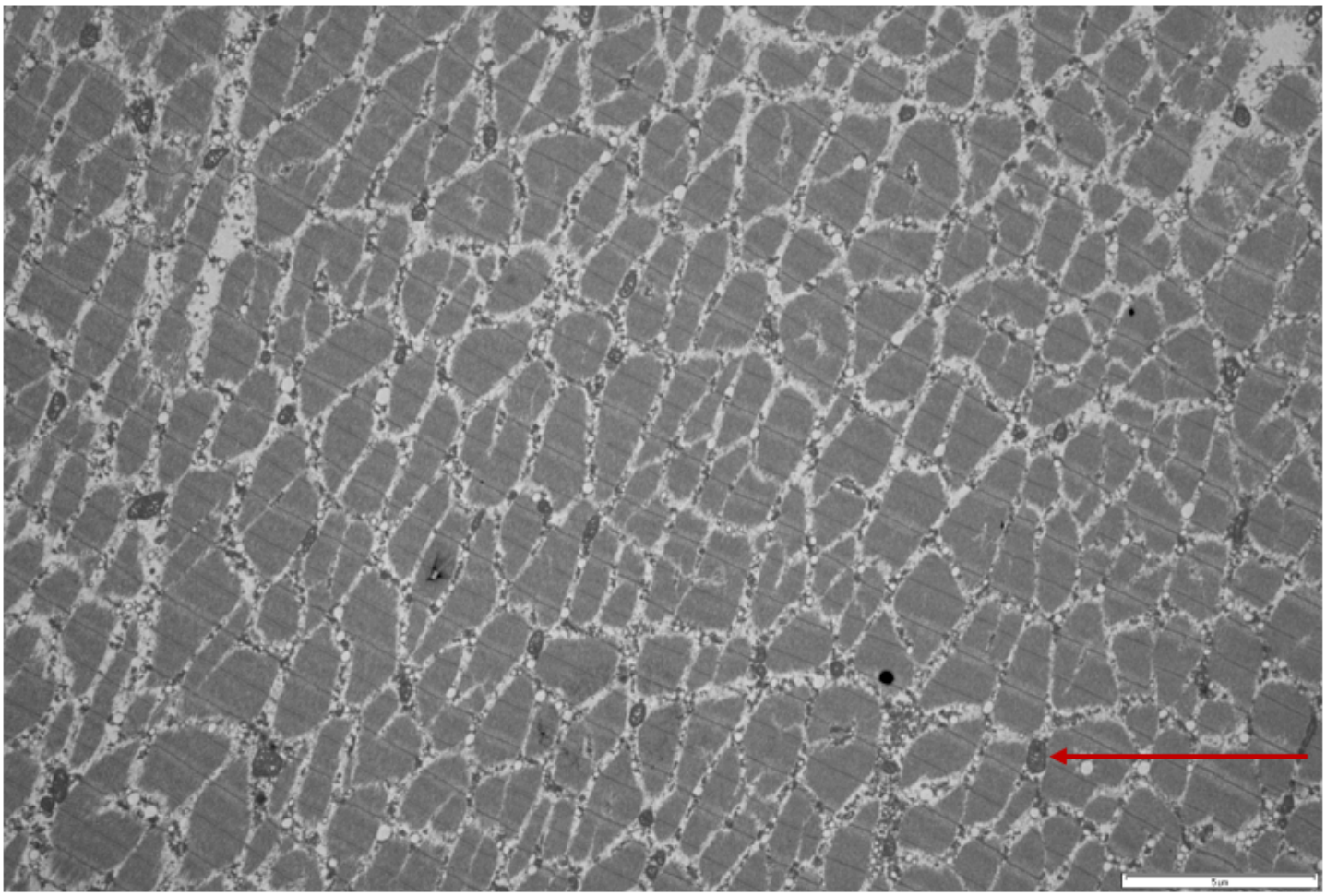

Figure 1

Representative TEM micrograph taken at 5000x magnification of a transverse section through the mitochondrial network of broiler breast muscle. The mitochondrial network is both of small diameter and sparse. An example mitochondrial tubule is identified by a red arrow.