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Histological and ultrastructural differentiation of cold-slaughtered and commercially available broiler chicken meat at early post-mortem intervals

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Abstract

Cold-slaughtered (CS) broiler chicken meat, derived from birds found dead prior to conventional processing, presents significant challenges for meat quality assurance and food safety verification due to the absence of validated confirmatory detection methods suitable for rapid regulatory application.

This study evaluated early post-mortem histological and ultrastructural alterations in breast muscle tissue collected at standardized intervals (0–2 h and 2–4 h post-mortem) to establish morphological criteria for differentiating CS meat from commercially available (CA) broiler chicken meat processed through conventional exsanguination.

Histological analysis was performed on five muscle samples of both slaughter groups and post-mortem intervals using haematoxylin and eosin (H&E) staining with standardized examination protocols. Transmission electron microscopy (TEM) was conducted on muscle samples from each treatment group at each time point to characterise subcellular ultrastructural changes. Structural integrity, myofibre organisation, sarcoplasmic alterations, mitochondrial morphology, and subcellular compartmentalization were systematically assessed according to predefined morphological criteria.

At 0–2 h post-mortem, CA breast muscle largely maintained fibre integrity with only mild degeneration, whereas CS samples already showed fibre separation, uneven eosinophilia, and focal myofibrillar damage. By 2–4 h, CS muscle exhibited considerable structural breakdown with extensive inter-fibre gaps, widespread vacuolation, and evident fragmentation, while CA tissue remained relatively better organised despite ongoing degeneration. Ultrastructurally, CS samples progressed from early mitochondrial vacuolation and cristae disruption to fibre separation and degenerative changes, whereas CA samples showed earlier myofibre hypercontraction accompanied by noticeable mitochondrial injury and glycogen debris, reflecting acute metabolic failure.

The combined histological and ultrastructural findings reveal distinct and reproducible early post-mortem degeneration trajectories between CS and CA broiler breast muscle.

Keywords: Cold-slaughtered meat; broiler chicken; histology; transmission electron microscopy; post-mortem degeneration; meat authentication

1. Introduction

Broiler chicken meat production serves as a critical global protein supply, with systems designed to meet escalating demand while maintaining commercial viability¹. The conversion of living muscle tissue into retail-ready meat initiates immediately following the cessation of blood circulation, triggering a complex cascade of interconnected biochemical and morphological modifications that profoundly influence the final product's nutritional composition, sensory attributes, microbiological status, and shelf stability².

Slaughter practices differ substantially across global production systems and cultural contexts. Commercially available slaughter approach, wherein severing of the carotid arteries, jugular veins, and esophagus occurs without prior unconsciousness-inducing procedures. Research comparing this method to alternative methods demonstrates that conscious-state slaughter

meets specific ritual requirements while maintaining meat safety standards¹. Scientific investigation reveals that commercially available slaughter procedures result in superior blood loss efficiency compared to mechanized methods. Higher bleeding percentages observed with unstunned procedures correlate with reduced residual hemoglobin concentrations in muscle tissue, which carries implications for meat preservation and microbiological stability³.

Post-mortem muscle composition demonstrates remarkable sensitivity to slaughter methodology. Studies comparing CA rabbits to other processing approaches revealed that absence of pre-slaughter stunning resulted in diminished residual blood content within muscle fibers while maintaining equivalent nutritional profiles³. Specifically, protein composition, fat content, cholesterol levels, and fatty acid distributions remained unaffected by slaughter approach, indicating that the primary quality differentiations relate to vascular integrity rather than fundamental tissue chemistry¹. Microbiological succession following slaughter shows stark contrasts between techniques. Extended bleeding duration during commercially available slaughter procedures permits greater removal of bacterial populations harbored within blood vessels and residual serum. Research on beef from CA cattle documented that extended bleeding intervals (15 minutes) significantly enhanced pathogen reduction compared to abbreviated bleeding periods⁴. Similarly, unstunned poultry processing yielded lower bacterial counts for both indicator organisms and pathogenic species during early post-mortem storage⁵.

Meat from properly bled carcasses exhibits decelerated oxidative deterioration during refrigerated storage, preserving sensory quality and extending retail viability³. The improved bleeding efficiency characteristic of CA contributes to this protective effect through reduction of iron-containing myoglobin and hemoglobin in tissue, which serve as oxidation catalysts⁶. Microscopic examination of muscle tissue reveals substantial architectural distinctions following different slaughter protocols. Properly exsanguinated carcasses maintain more organized myofibrillar arrangements compared to inadequately bled tissues, which exhibit accelerated autolytic enzyme activity due to continued cellular decomposition in residual blood. The presence of dissolved hemoglobin and associated ferrous ions within muscle tissue intensifies protease and lipase activity during post-mortem rigor development, hastening myofibril degradation and sarcomeric disorganization⁷.

A critical but often overlooked concern in poultry processing involves the inadvertent entry of dead or moribund birds into the processing stream. This represents a substantial food safety hazard associated with pathogenic microorganism accumulation⁸. Animals subjected to extended pre-slaughter lairage under suboptimal conditions manifest elevated baseline intestinal bacterial counts prior to evisceration⁸. Dead or dying animals within holding pens present particularly acute hazards, as they serve as infection sources for surviving cohort members through faecal contact and aerosol pathways⁸.

While CA slaughter demonstrates superior bleeding efficiency and reduced pathogenic contamination¹, the cellular and ultrastructural mechanisms underlying these advantages remain inadequately characterized. Current literature documents that dead or moribund birds undergo rapid tissue autolysis and bacterial proliferation⁶, yet lacks systematic temporal characterization of these pathological changes

during early post-mortem periods.

2. Materials and Methods

2.1 Ethical Approval and Sample Source

This study did not involve any experimental procedures on live animals. Cold-slaughtered (CS) samples were obtained from broiler birds that were dead on arrival (DOA) at local retail poultry outlets, while commercially available (CA) samples were collected from broiler birds processed through routine commercial slaughter and exsanguination at licensed meat shops. All samples were collected post-mortem as part of routine commercial processing or market disposal, and no animals were specifically sacrificed for the purposes of this research. Therefore, formal institutional ethical approval was not required. Sample collection was conducted in accordance with standard hygiene and food safety practices applicable to commercial poultry handling.

2.2 Sample Collection and Tissue Handling

Breast muscle tissue (*Pectoralis major*) samples were collected from broiler chickens (commercial meat-type genetics, approximately 42 days of age, average slaughter weight 2.3–2.6 kg) at two standardized post-mortem intervals: 0–2 hours and 2–4 hours post-mortem. For each treatment group (CS and CA) at each post-mortem interval (0–2 h and 2–4 h), five independent breast muscle samples were collected for histological evaluation (n = 5 per group per interval). Transmission electron microscopy was performed on one representative sample per group per interval (n = 1), selected based on typical histological features observed within each group. The muscle samples approximately 1.5 cm × 1.5 cm × 1.0 cm in dimension were obtained from standardized anatomical locations within the muscle (ventral medial region, approximately 2 cm from the sternum and 3 cm from the anterior border). Sampling locations were marked on each carcass to ensure anatomical consistency across replicates. All tissue samples were obtained within 15 minutes of the designated post-mortem collection window to minimize temporal variability.

2.3 Histological Processing and Examination

Samples intended for histological analysis were immediately transferred to individual 50 ml sterile tubes containing 10% neutral buffered formalin (NBF) fixative, maintaining a tissue-to-fixative ratio of approximately 1:10 (v/v) to ensure adequate penetration. Muscle tissue samples were processed using standard routine histological protocols⁹.

2.4 Transmission Electron Microscopy (TEM) Protocol

TEM analysis was performed on one representative sample per group per time interval.

Samples for transmission electron microscopy were transferred to individual tubes containing 2.5% glutaraldehyde in 0.1 M sodium cacodylate buffer (pH 7.4) within 2 minutes of tissue collection. Standard protocol was followed for sample preparation for electron microscopy analysis^[10]

3. Results

3.1 Histological Findings

0–2 Hour Post-Mortem Interval

Commercially available (CA) breast muscle at 0–2 h post-mortem demonstrated largely preserved histological architecture characterized by regularly organized and densely

packed myofibers with distinct sarcolemmal membranes and clear demarcation between individual fibers. Sarcoplasmic staining was uniformly eosinophilic with consistent cytoplasmic clarity across examined fields. Only mild interstitial separation was observed in occasional regions between muscle fiber bundles. Occasional individual fibers displayed mild cytoplasmic vacuolation. Architecture integrity (largely preserved) (figure 1 A).

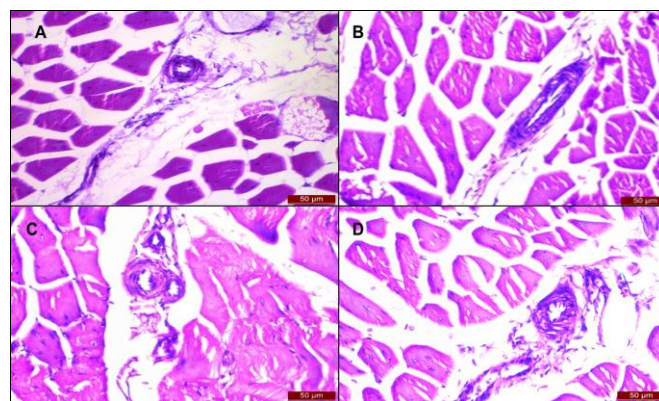
Cold-slaughtered (CS) breast muscle at the same 0–2 h interval demonstrated visibly different morphology. Mean interfiber spacing was significantly increased, indicating marked loss of normal tissue compaction. Multiple myofibers displayed irregular contours with regional waviness and asymmetric cross-sectional profiles. Fiber separation was evident in of examined fields, with some discrete myofibers showing focal detachment from adjacent fibers. Sarcoplasmic staining pattern was notably uneven, with regional patches of increased eosinophilia interspersed with areas of reduced staining intensity. Approximately 20–25% of observed myofibers exhibited mild to moderate cytoplasmic vacuolation. Overall assessment: architecture integrity moderately disrupted, indicating early post-mortem degeneration (figure 1 C).

2–4 Hour Post-Mortem Interval

CS breast muscle at 2–4 h post-mortem exhibited pronounced and advanced structural deterioration. Mean interfiber spacing expanded dramatically, indicating severe loss of normal tissue architecture. Extensive myofiber separation was evident in majority of examined fields, with many fibers appearing isolated rather than organized in bundles. Marked myofiber fragmentation was observed, characterized by irregular fiber breakage and discontinuity of sarcolemmal membranes in numerous regions. Sarcoplasmic staining exhibited pronounced patchy distribution with large areas of reduced eosinophilia alternating with zones of increased staining intensity. Widespread sarcoplasmic vacuolation (affecting approximately 60–70% of myofibers) was prominent, appearing as irregular clear or pale-staining spaces within myoplasm. Normal fiber architectural alignment was substantially disrupted or absent. Overall assessment: Architecture integrity severely disrupted, consistent with advanced post-mortem degeneration (figure 1 D).

CA breast muscle at 2–4 h post-mortem demonstrated comparatively preserved organization despite progressive post-mortem changes. Interfiber spacing remained relatively moderate, substantially lower than CS samples at equivalent time point. Most myofibers retained recognizable cross-sectional identity with preserved sarcolemmal continuity, though some degree of separation between fiber bundles was evident. Focal sarcoplasmic degeneration with limited vacuolation was present in restricted regions. Limited myofiber fragmentation was observed, with most fragmentation confined to peripheral muscle fibers. Overall assessment, architecture integrity score mildly to moderately disrupted, indicating relatively slow post-mortem degeneration trajectory (figure 1 B).

CS samples consistently demonstrated earlier onset (evident at 0–2 h) and greater severity of histological degeneration compared to CA samples at both time intervals. By 2–4 h post-mortem, the morphological distinctions between groups were striking, with CS muscle showing advanced structural collapse versus CA muscle maintaining relatively preserved architecture.



(A) CA breast, 0–2 h post-mortem showing largely preserved fiber architecture.

(B) CA breast, 2–4 h showing moderate interfiber separation with limited vacuolation.

(C) CS breast, 0–2 h demonstrating increased interfiber spacing and early cytoplasmic vacuolation.

(D) CS breast, 2–4 h exhibiting extensive myofiber separation, fragmentation, and widespread sarcoplasmic vacuolation.

Fig 1: Transverse (cross-sectional) histological features of breast muscle in commercially available (CA) and cold-slaughtered (CS) chicken. H&E staining. Scale bar (A–D) = 50 µm.

3.2 Ultrastructural Findings (TEM)

0–2 Hour Post-Mortem Interval

CS breast muscle at 0–2 h post-mortem demonstrated largely preserved sarcomeric organization when examined ultrastructurally. Z-bands remained intact and regularly spaced, A-bands and I-bands were clearly defined, and H-zones appeared normal. Myofibrillar organization was regular with myofibrils maintaining normal spatial relationships to adjacent myofibrils. Only mild regional myofiber contraction was observed in isolated areas. The predominant ultrastructural alteration involved mitochondrial morphology: mitochondria displayed evident vacuolation with partial to complete disruption of cristae architecture. Cristae appeared broken or disorganized rather than maintaining the normal organized pattern. Mitochondrial matrix appeared rarefied (less dense), consistent with early energy depletion. Outer mitochondrial membranes remained largely intact though some regions showed membrane undulation. Sarcoplasm displayed normal ribosomal density and glycogen granule distribution. Subsarcolemmal regions appeared normal with good adhesion of myofibrils to sarcolemma. Overall ultrastructural assessment: Predominantly organelle-level damage with preserved myofibrillar organization (figure 2 C). CA breast muscle at 0–2 h exhibited markedly different ultrastructural presentation. Myofibers displayed pronounced hypercontraction, with Z-bands compressed and I-bands nearly absent, indicating severe myofiber shortening. Sarcomeric organization was disrupted by contracture effects, though basic sarcomeric integrity remained recognizable. Myofibrillar separation was evident in multiple regions, with individual myofibrils appearing displaced from normal spatial relationships. Mitochondria displayed prominent vacuolation with extensive cristae disruption comparable to CS samples but with additional severe mitochondrial membrane damage. Some mitochondria showed partial membrane rupture with matrix contents dissipating into surrounding sarcoplasm. Mitochondrial matrix density was markedly reduced. Additionally, glycogen granules appeared depleted (reduced density and number compared to normal) (figure 2 A). Overall ultrastructural assessment, Extensive contracture-driven damage with early organelle-level degeneration.

2–4 Hour Post-Mortem Interval

CS breast muscle at 2–4 h post-mortem demonstrated progressive ultrastructural degeneration from the earlier interval. Increased myofibrillar separation was evident, with myofibrils showing irregular spacing and some degree of architectural disruption. However, sarcomeric organization remained partially recognizable in many regions rather than completely disorganized. Z-bands showed structural deterioration with some Z-band disruption and misalignment but maintenance of basic band pattern in many sarcomeres. Mitochondrial damage had progressed substantially: extensive vacuolation was present, cristae were nearly completely disorganized, and matrix density was markedly reduced. Some mitochondria displayed partial membrane disruption. Critically, features consistent with subsarcolemmal fluid accumulation were prominent, appearing as clear spaces between myofibrils and the sarcolemmal membrane, suggesting increased tissue permeability and fluid leakage. Sarcoplasm showed retained ribosomes and some glycogen granules despite overall progressive deterioration. Overall ultrastructural assessment: Progressive ischemic-pattern degeneration with increasing tissue fluid accumulation and continuing mitochondrial damage (figure 2 D).

CA breast muscle at 2–4 h exhibited extensive ultrastructural collapse. Severe myofiber hypercontraction was predominant, with complete loss of normal I-band structure and severe Z-band compression. Sarcomeric organization was largely obliterated by contracture effects, appearing as dense packed material rather than recognizable sarcomeric units. Myofibrillar separation was extensive with myofibrils severely displaced from normal relationships. Mitochondrial pathology was catastrophic: widespread vacuolation involved essentially all visible mitochondria, cristae were completely disorganized and fragmented, mitochondrial membranes showed partial to complete disruption with visible matrix leakage, and matrix density was severely reduced. Glycogen debris was prominent and accumulated in the sarcoplasm, appearing as electron-dense granular material. Subsarcolemmal regions showed extensive fluid accumulation and separation from myofibrils. Overall ultrastructural assessment: Severe contracture-driven collapse with catastrophic organelle-level degeneration consistent with acute metabolic shock (figure 2 B).

CS muscle demonstrated a relatively gradual and progressive degeneration pattern dominated initially by organelle-level mitochondrial changes, with myofibrillar disruption becoming more evident in the later time interval. CA muscle displayed more rapid and severe overall ultrastructural collapse, with marked hypercontraction prominent from the earliest time interval and progressively worsening, accompanied by extensive organelle. The distinct degeneration trajectories reflected fundamentally different physiological injury patterns.

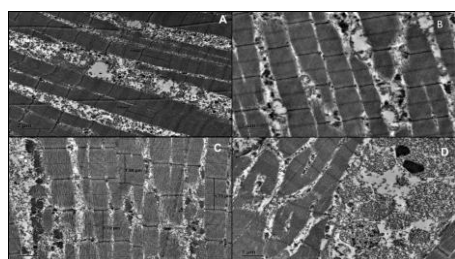


Fig 2: Ultrastructural alterations in breast muscle (longitudinal section) observed by transmission electron microscopy. Scale bar (A = 2 μ m, B–D = 1 μ m)

(A) CA breast at 0–2 h post-mortem showing marked myofiber hypercontraction with Z-band compression and mitochondrial vacuolation.

(B) CA breast at 2–4 h exhibiting severe sarcomeric collapse, extensive myofibrillar separation, and widespread mitochondrial disruption with subsarcolemmal fluid accumulation.

(C) CS breast at 0–2 h demonstrating largely preserved sarcomeric organization with early mitochondrial vacuolation and cristae disruption.

(D) CS breast at 2–4 h showing progressive myofibrillar separation, Z-band disruption, and advanced mitochondrial degeneration with subsarcolemmal spaces.

4. Discussion

The present investigation revealed markedly distinct progressions of post-mortem histological changes between commercially available (CA) and cold-slaughtered (CS) broiler breast muscle during the early post-mortem period, indicating divergent underlying physiological injury mechanisms.

The preservation of relatively compact fiber architecture in CA muscle at 0–2 h post-mortem aligns with established biochemical understanding that cytoskeletal anchoring proteins such as desmin, vinculin, talin-2, and integrin β 1 maintain structural stability during the initial post-mortem hours, thereby preserving fiber cohesion and sarcoplasmic organization [11]. Furthermore, myofibrillar degradation progresses gradually through calpain-mediated proteolytic pathways rather than occurring immediately after death, explaining the absence of marked degeneration in CA muscle at this stage [12].

In contrast, CS muscle exhibited early disruption of fiber compaction, including widened interfiber spacing, uneven eosinophilic staining, fiber waviness, and fragmentation at 0–2 h post-mortem. These findings indicate premature initiation of degenerative processes in CS muscle. Similar early structural alterations, including fiber kinking and loss of tissue compaction, have been reported in post-mortem fowl muscle [13]. Such rapid deterioration is likely due to uncontrolled physiological stress prior to death, which accelerates post-mortem tissue breakdown [14].

By 2–4 h post-mortem, degeneration in CS muscle became pronounced, characterized by extensive fiber separation, patchy eosinophilic staining, sarcoplasmic vacuolation, and myofibrillar fragmentation. These changes reflect advanced post-mortem degradation and are consistent with calpain-mediated proteolysis of cytoskeletal proteins [12]. Patchy staining and sarcoplasmic alterations likely indicate protein denaturation and reduced water-holding capacity [15]. Additionally, severe anoxic conditions associated with sudden death lead to increased tissue damage compared to controlled slaughter, resulting in edema-like changes and widened connective tissue spaces due to ischemia-induced fluid accumulation [16, 17].

In contrast, CA muscle at 2–4 h post-mortem showed only mild structural alterations with limited interfiber separation and preserved fiber alignment. This delayed degeneration supports previous findings that structural protein breakdown occurs progressively over time [11].

Ultrastructural analysis further demonstrated distinct degeneration patterns. In CS muscle, early mitochondrial alterations, including vacuolation and cristae disruption, preceded major myofibrillar damage, indicating progressive energy depletion [12].

Conversely, CA muscle exhibited early hypercontraction and mitochondrial damage, suggesting acute metabolic stress associated with exsanguination. Similar contracture-related damage has been observed in experimental shock conditions, characterized by rapid structural collapse and mitochondrial injury [18]

By 2–4 h, CS muscle showed progressive mitochondrial damage and fluid accumulation due to increased membrane permeability and oxidative stress [19, 20]. Meanwhile, CA muscle exhibited severe hypercontraction, mitochondrial disruption, and glycogen depletion, reflecting acute metabolic failure [21].

Overall, CS muscle followed a gradual ischemia-driven degeneration pathway, whereas CA muscle exhibited rapid contracture-driven degeneration. These findings highlight distinct physiological mechanisms underlying post-mortem changes in the two groups.

The integration of histological and ultrastructural observations provides objective morphological criteria for differentiating CS and CA meat during early post-mortem intervals. However, limitations include small sample size, evaluation of a single muscle type, and restricted sampling conditions. Further studies incorporating larger sample sizes, multiple processing environments, and additional biochemical markers are required to validate these findings and enhance their applicability for regulatory purposes.

5. Conclusion

The present study demonstrates that cold-slaughtered (CS) and commercially available (CA) broiler breast muscle exhibit distinct and reproducible post-mortem degeneration patterns during early post-mortem intervals.

CS muscle showed earlier onset and greater severity of histological and ultrastructural alterations, characterized by fiber separation, vacuolation, mitochondrial damage, and progressive ischemic degeneration. In contrast, CA muscle exhibited comparatively delayed structural changes, with early hypercontraction and metabolic stress-related alterations.

These findings indicate that CS muscle follows a gradual ischemia-driven degeneration pathway, whereas CA muscle undergoes rapid contracture-associated degeneration due to acute metabolic disturbance.

The combined use of histological and ultrastructural techniques provides reliable morphological criteria for differentiating cold-slaughtered and commercially available poultry meat. However, further validation under diverse field conditions and integration with biochemical markers are necessary before routine application in meat quality authentication and regulatory monitoring.

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Conflict of Interest

Not available

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