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Physiological and Histological Consequences of Growth Stunting in Broiler Chickens

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Abstract | Growth stunting is one of the leading causes of low productivity in poultry and is negatively affecting animal wellbeing. This study aim to determine the effect of growth stunting on body weight at different age points, evaluating changes in blood and biochemical parameters and assessment of histological alterations in the liver of stunted chickens compared to health birds.. For this purpose, a total of 300 broiler chicken were collected with age of either 14 or 28 days over the period of 5 months (May-Oct, 2022). Birds belonging to either of two categories (n=150 normal-growth or n=150 exhibiting growth stunting) were sacrificed and examined. The intestinal lengths (total and small intestine segments) were measured and blood samples were collected to analyze red blood cell (RBC) count, white blood cell (WBC) count, hemoglobin concentration, and packed cell volume (PCV). Additionally, blood serum was separated to determine the concentration of total protein, cholesterol, and glucose which are key biochemical markers that provide insights into protein metabolism, lipid profile, and blood sugar regulation, respectively. Liver tissues samples from were subjected to histological examination. The results revealed a substantial decrease in body weight (160 gm) in stunted birds compared to the normal control group (479 and 1488 gm) at both 14 and 28 days of ag, respectively. Furthermore, we observed a significant decrease in cellular and biochemical blood parameters in stunted birds where RBC (1.65 and 1.83) showed a lower ($p<0.05$) count compared to health control (2.24 and 3.76) at both 14 and 28 days of age, respectively. This also applies to HB levels (7.62 and 9.44) in stunted birds compared to healthy birds (9.33 and12.65) at the ages of 14 and 28 days, respectively. Histological analysis of the liver from stunted chickens showed various changes, including areas of necrosis and fibrosis. Collectively, these findings highlight the detrimental effects of growth stunting, characterized by reduced weight gain, altered blood cell counts, and liver damage. This study provides valuable insights for optimizing broiler management practices to ensure optimal growth and health for enhanced productivity.

Keywords | Broiler, Stunted, Growth, Hematology, Histopathology

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INTRODUCTION

In many tropical countries including Iraq, the poultry sector faces a critical issue with growth stunting in broilers birds. Growth stunting in poultry a condition characterized by birds failing to achieve their expected weight or size for their age despite receiving adequate nutrition and care (Qamar *et al*., 2013). Several factors including elevated temperatures and other environmental challenges can influencing this syndrome. Several studies have identified that stunting can be attributed to genetics, dietary factors, and illnesses, however, the specific physiological and histological alterations linked to stunting are not fully understood (Health and Welfare, 2010). This lack of clarity hinders the formulation of precise strategies to upgrade animal well-being and production of broilers (Prameela Rani *et al*., 2011; Hoerr, 2010).

A comprehensive understanding of the physiological and histological changes associated with the condition remains elusive and this knowledge could facilitate development of interventions which can improve broiler health and productivity. This phenomenon manifests as reduced growth rates, compromised feed conversion efficiency, and a cascade of physiological and metabolic disturbances. The prevalence of stunting in broiler chickens and other poultry species necessitates a thorough understanding of the underlying etiological factors. These factors encompass genetic predisposition, environmental stressors, and infectious agents. Additionally, compromised welfare conditions, disease outbreaks, feed toxicosis, elevated ambient temperatures, inherent genetic makeup, malabsorption syndromes, and viral infections have all been implicated in the development of stunting (Hoerr, 2010) .

The precise mechanisms underlying growth stunting remain incompletely understood, however, it has been proposed that disruptions in essential physiological processes, such as nutrient absorption, metabolism, and hormonal regulation, are likely underline the syndrome (Shapiro *et al*., 1998). This phenomenon of stunted growth can exert a significant detrimental impact on both avian health and welfare, ultimately translating into substantial economic losses for poultry producers (Zavala, 2006). Consequently, the development of effective prevention and management strategies for stunting in poultry necessitates a multifaceted approach that encompasses considerations of genetics, nutrition, environmental and management practices, and comprehensive disease control measures (Li *et al*., 2020). Several studies conducted by Qamar *et al*. (2013) and Li *et al*. (2020) have highlighted the importance of comprehensive management practices that encompass improved breeding programs, optimized nutritional regimens, and stringent biosecurity measures to mitigate the risk of stunting. Additionally, it has been noticed that environmental factors such as housing conditions and temperature regulation play a crucial role in the overall health and growth performance of broilers (Prameela Rani *et al*., 2011).

Despite our limited understanding of growth stunting, current management strategies often fall short of effectively preventing and mitigating this condition in poultry production. This necessitates a multifaceted approach that delves deeper into the underlying physiological and histological changes associated with stunting. Primary aim of this study is to bridge the knowledge gap on the growth stunting in chicken by investigating physiological and histological changes in broiler chickens. Specifically, we aim to assess a range of blood parameters and liver histology to gain deeper insights into the physiological processes affected by stunting. Our findings contributed valuable information for poultry producers and researchers, ultimately aiding in the development of more effective management strategies to optimize broiler health, growth, and productivity.

MATERIALS AND METHODS

A total of 300 broiler chickens were systematically selected from four reputable breeding facilities in the region from May to October 2022. To minimize selection bias, a randomized sampling approach was applied and broilers were assigned to one of two groups (n=150/group): either in growth stunted or normal health group based on pre-established criteria. The normal growth group comprised birds exhibiting weights and body conformation within the standard growth parameters for their age (14 and 28 days). The stunted group included broilers displaying a minimum of 20% weight reduction compared to the expected weight for their age based on standard broiler growth charts.

Dietary Treatments

Birds were fed a standard commercial broiler diet formulated to meet their nutritional requirements throughout the experiment. The diets provided reflect standard diets used in the region. The dietary treatments included a Normal 14d, Stunted 14d, Normal 28d and Stunted 28d respectively as outlined in Table 1.

Ethical Considerations

The experimental procedures were approved by the Institutional Animal Care and Use Committee of the University of Basrah, College of Veterinary Medicine Research Ethics Committee approval number: (26-42-2022). All animal handling adhered to the guidelines set forth by the National Institutes of Health (NIH) Guide for the Care and Use of Laboratory Animals.

Euthanasia and Sample Collection

At 14 and 28 days of age, birds were euthanized using a humane method of CO_2 inhalation followed by cervical dis-

location according to the University of Basrah, College of Veterinary Medicine Research Ethics Committee guidelines. Following euthanasia, body weight was measured for each bird in both groups.

Table 1: Ingredients and nutrient analysis of diets used during the starter and finisher rearing periods.

Ingredient (%)	Starter period (1–19 days old)	Finisher period (20–28 days old)
Corn	44.29	47.95
Soybean meal	34.85	26.99
Wheat	10.00	10.0
Meat meal	5.00	5.00
Sunflower oil	2.17	6.25
DCP	0.78	0.54
CaCO3	0.86	1.05
Sodium bicarbonate	0.12	0.12
NaCl	0.25	0.25
DL-Methionine	0.07	0.22
Enzyme	1.00	1.00
Antioxidant	0.02	0.02
Vitamin mixture[1]	0.25	0.25
Mineral mixture[2]	0.25	0.25
Chemical analysis		
ME (kcal/kg)	2,900	3,200
Crude protein (%)	22.0	19.0
Calcium (%)	1.00	1.00
Available phosphorus (%)	0.48	0.42
Lysine (%)	1.28	1.06
Methionine (%)	0.40	0.48
Methionine + cysteine (%)	0.73	0.76
Threonine (%)	0.84	0.71

[1]vitamin A: 5 000 IU/g; vitamin D3: 500 IU/g; vitamin E: 3 mg/g; vitamin K3: 1.5 mg/g; vitamin B2: 1 mg/g; [2]calcium pantothenate: 4 mg/g; niacin: 15 mg/g; vitamin B6: 13 mg/g; Cu: 3 mg/g; Zn: 15 mg/g; Mn: 20 mg/g; Fe: 10 mg/g; K: 0.3 mg/g; ME: metabolizable energy. Diets were the same for all treatments.

Gastrointestinal Tract Measurements

Gastrointestinal tract measurements were performed as described by Li *et al.* (2020). Briefly, the entire gastrointestinal tract was carefully removed, and the total intestinal length and the segmented lengths of the small intestine were measured using a measuring tape.

Blood Collection and Analysis

Five milliliters of blood was collected from the femoral vein of each bird at 14 and 28 days of age before euthanasia. Blood samples were divided into two aliquots. One aliquot was collected in EDTA-coated tubes for hematological analysis, including erythrocyte count, leukocyte counts using established protocol as outlined by (Natt and Herrick, 1952). Hemoglobin concentration, and packed cell volume were assessed using an automated cell counter (Sysmex® KX-530M) (Varley et al., 1980). The other aliquot was allowed to clot, and the serum was separated for subsequent biochemical analysis. Serum total protein, cholesterol, and glucose levels were determined using commercially available assay kits (Biolab technical) following the manufacturer's instructions.

Histological Examination of Liver Tissue

Liver samples were collected from each bird and fixed in 10% neutral buffered formalin for histological examination. Tissues were processed routinely, embedded in paraffin wax, sectioned at 5µm thickness, and stained with Hematoxylin and Eosin (H,E) for evaluation under a light microscope for any pathological changes associated with growth stunting.

Statistical Analysis

In a symphony of statistical inquiry, an ANOVA sonata was meticulously orchestrated within a randomized tableau. The enigmatic Duncan's Multiple Range Test emerged as the maestro, its nimble baton guiding us to discern the crescendo of significant treatment effects, resonating at a crystalline 5% level of confidence (JH and Dickey, 1980).

Table 2: Effect of stunting on body weight and some intestine parts.

Criteria/Organ	Period	Treatments	
		Normal	Stunted
BW (gm)	14 day	^{a}521 ± 3.0	^{b}160±2.0
	28 day	^{a}1488 ±11.0	^{b}479±4.0
Duodenum (cm)	14 day	^{a}8.7±0.2	^{b}2.6±0.11
	28 day	^{a}9.4±0.21	^{b}3.32±0.24
Jejunum (cm)	14 day	^{a}9.3±0.25	^{b}3.2±0.11
	28 day	^{a}9.6±0.27	^{b}3.32±0.24

In the row, the values $^{(a,b)}$ in different superscripts were significantly different.

RESULTS AND DISCUSSION

The data presented in Table 2 highlights the significant physiological impact of stunting on broilers, particularly evident in the body weight and intestinal measurements at 28-day-old in stunted broilers compared to their healthy counterparts. The marked reduction in body weight and intestinal part dimensions in stunted broilers underscores the severity of growth impediments caused by stunting. The body weight of stunted birds was significantly lower ($p<0.01$) compared to the control group at both 14 and 28 days of age Table 2. At 14 days, the average body weight of

stunted birds was 160 grams, while the control group carry an average of 521 grams. Similar weight differences were observed at 28 days.

Physiological Implications: Stunting syndrome, characterized by reduced growth rates and under-developed intestinal morphology, has been associated with various factors, including nutritional deficiencies, environmental stressors, and infectious agents. The observed decrease in body weight aligns with previous findings that stunted broilers exhibit compromised nutrient absorption and metabolism, leading to poor growth performance as reported before (de Oliveira *et al*., 2021;Qamar *et al*., 2013). It was observed that growth stunted birds consume more feed per unit of weight gain, leading to increased production costs and reduced profitability for producers (Lenhardt and Mozeš, 2003).

Histopathological Insights: The histopathological examination of the duodenum and jejunum reveals structural alterations that may contribute to the malabsorption and maldigestion observed in stunted broilers. These changes could be indicative of underlying inflammatory processes or infections that disrupt normal gut function (Shapiro and Nir, 1995). The phenomenon of stunting in broilers is not only a concern for animal welfare but also poses significant economic challenges for the poultry industry. The findings of this study add to the growing body of evidence calling for improved management practices and further improve the animal wellbeing and productivity.

Our study investigated the physiological alterations associated with growth stunting in broiler chickens raised. The findings provide valuable insights into the detrimental effects of stunting on blood parameters. As expected, our results demonstrated a significant decrease ($p<0.05$) in red blood cell (RBC) count in stunted birds compared to healthy controls at both 14 and 28 days of age (Table 3). Additionally, RBC count were significantly lower ($p<0.05$) (1.65 and 1.83) in stunted birds compared to healthy controls (2.24 and 3.76) at both 14 and 28 days of age, respectively. White blood cell (WBC) counts were also markedly lower ($p<0.05$) in stunted birds at both tested time points. Based on the observed positive correlation with RBC count, hemoglobin and packed cell volume (PCV) are also likely lower in stunted birds.

These findings on blood cell parameters align with previous studies on growth stunting in poultry. For instance, Shapiro *et al*. (1998) have reported similar reductions in RBC and hemoglobin in stunted chickens. The observed decrease in RBC, hemoglobin, and PCV in our study suggests potential anemia in stunted birds compared to healthy birds. This anemia could be attributed to various factors including iron deficiency, impaired erythropoiesis (red blood cell production), or nutritional imbalances that require further investigation. Similarly, the decreased WBC count in stunted birds aligns with findings reported previously by Nili *et al*. (2007) who have suggesting a potential suppression of the immune system. However, further research is warranted to elucidate the specific mechanisms underlying these changes in immune function.

Table 3: Effect of stunting on some blood parameter.

Criteria	Period	Treatments	
		Normal	Stunted
RBC (ml /10^6)	14 day	^{a}2.24 ± 0.11	^{b}1.65±0.07
	28 day	^{a}3.76 ± 0.14	^{b}1.83±0.05
WBC (ml /10^3)	14 day	18.45±1.32	21.77±1.96
	28 day	19.71±1.51	23.55± 2.04
Hb (g/dl)	14 day	^{a}9.33±0.25	^{b}7.62±0.27
	28 day	^{a}12.65±0.56	^{b}9.44± 0.31
PCV (%)	14 day	^{a}27.55±0.88	^{b}24.43±0.72
	28 day	^{a}29.37±0.56	^{b}26.93 ± 0.33

In the row, the values $^{(a,b)}$ in different superscripts were significantly different.

Also, we noticed the observed alterations in blood parameters likely contribute to the detrimental effects of growth stunting on broiler health and performance (Table 3). Anemia can lead to reduced oxygen delivery to tissues, hindering growth and development. Suppression of the immune system can increase susceptibility to infectious diseases, further impacting bird health and productivity. These consequences of stunting can ultimately translate into economic losses for poultry producers. While quantifying the precise economic impact falls beyond the scope of this study, our findings highlight the importance of managing growth stunting to optimize broiler health and economic viability in the poultry industry.

Broilers with stunted growth also exhibited changes in their metabolic rate (Shapiro *et al*., 1997). The metabolic rate refers to the rate at which an organism uses energy and stunted birds have been shown to have lower metabolic rates compared to healthy birds. This decrease in metabolic rate may be a result of reduced thyroid hormone activity, which plays a critical role in regulating metabolic processes in the body (Arzour-Lakehal *et al*., 2013).

In addition to changes in metabolic rate, stunted broilers may also exhibit other metabolic disturbances, including alterations in carbohydrate, lipid, and protein metabolism. These disturbances can lead to changes in blood glucose and insulin levels, as well as alterations in liver and muscle metabolism. It has been demonstrated earlier that these changes can further impair growth and development in stunted birds (Qamar *et al*., 2015).

Findings reported in this study demonstrated that growth stunting in broiler chickens is associated with significant alterations in blood cell parameters, potentially leading to anemia and immune system suppression. These findings contribute to our understanding of the physiological consequences of stunting and warrant further investigations into the underlying mechanisms and potential mitigation strategies.

Furthermore, an analysis of blood serum parameters revealed the detrimental effects of stunting on total protein, glucose, and cholesterol concentrations (Table 4). Notably, a significant decrease in total protein concentration was observed in the stunted group at 14 days of age compared to the control group. This decline persisted with age, as evidenced by continued lower protein levels at 28 days. The decline in total protein levels suggests impaired synthesis or increased catabolism, which could be attributed to malnutrition or liver dysfunction. Protein is vital for growth and immune function, therefore, this deficiency can exacerbate the stunting condition (Nili *et al*., 2007).

Table 4: Effect of stunting on some blood biochemical parameter.

Criteria	period	Treatments	
		Normal	Stunted
Total protein g/100ml	14 day	^{a}4.6 ± 0.32	^{b}3.71 ± 0.11
	28 day	^{a}6.4 ± 0.54	^{b}3.85 ± 0.27
Glucose mg/100 ml	14 day	^{a}190 ± 9.8	^{b}166.56 ± 7.5
	28 day	^{a}204 ± 8.7	^{b}176 ± 6.3
Cholesterol mg/100 ml	14 day	144 ± 7.48	132 ± 4.8
	28 day	^{a}166 ± 5.6	^{b}143 ± 4.2

In the row, the values $^{(a,b)}$ in different superscripts were significantly different.

Additionally, the concentration of blood serum glucose displayed a significant decrease in stunted birds at both 14 and 28 days. It is plausible to speculate that the decreased glucose concentration reflects possible alterations in carbohydrate metabolism or increased utilization due to stress responses. Glucose is a primary energy source, and its scarcity can lead to energy deficits, affecting overall growth (Shapiro *et al*., 1997). A pronounced reduction in serum cholesterol concentration was observed in the stunted group at both age points. Lower cholesterol levels might attribute to reduced lipid absorption or synthesis. Cholesterol is essential for cell membrane integrity and hormone production, and its reduction could further compromise the health of stunted broilers (Nili *et al*., 2007). In alignment with previous findings (Qamar *et al*., 2013), these results suggest that stunting in broilers triggers significant physiological alterations, potentially hindering growth, development, and feed conversion efficiency. Elucidating the underlying mechanisms of stunting and developing effective preventive and management strategies are crucial for promoting bird health and welfare, while concurrently maximizing producer profitability. Several factors have been implicated in the development of stunting in broiler chickens, with immunosuppression being a prominent contributor (Hoerr, 2010). It is postulated that stunted birds exhibit a compromised immune system, rendering them more susceptible to infections and diseases.

This immunosuppression is likely a consequence of various factors, including malnutrition, chronic stress, and inadequate management practices (Bhattacharya and Bhowmik, 2014).

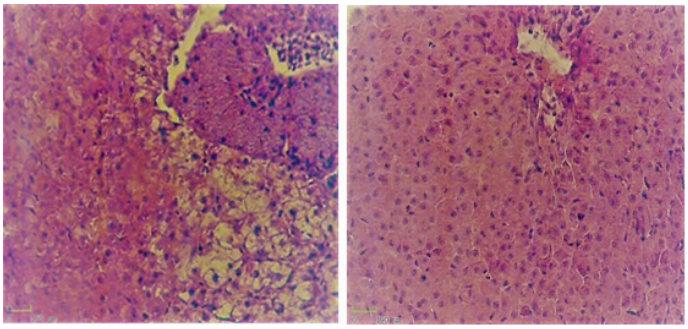

Figure 1: The effect of stunning on broiler liver in 28 day old.

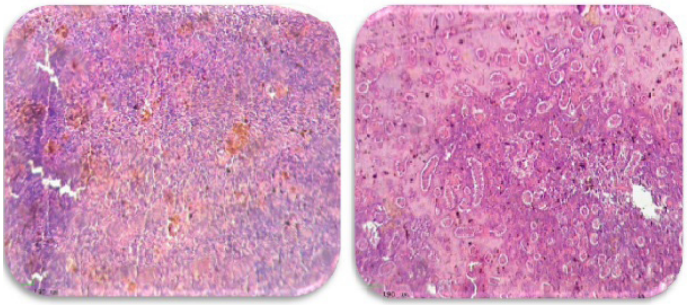

Figure 2: The effect of stunning on broiler liver in 28 day old.

Histological examination of liver tissue from 28-day-old stunted broilers (Figure 1) revealed irregularities in hepatocyte morphology surrounding the central vein. Additionally, necrotic changes were observed in cells located near the portal vein. Microscopic examination of liver tissues from birds exhibiting stunted growth revealed sporadic centers of cellular demise within the hepatic lobules. These necrotic areas were discernible due to their pinkish hue and the breakdown of the customary cellular architecture, coupled with the detection of pyknotic nuclei—shrunken and intensely stained nuclei within the hepatocytes (Figure 2). Cells neighboring these areas often displayed cytoplasmic vacuolation, indicative of intracellular voids, hinting at cellular distress. Additionally, evidence of fibrosis was present, characterized by the deposition of dense connective tissue. These findings suggest a compromised hepatic architecture, potentially hindering the ability of liver to perform essen-

tial metabolic and physiological functions. The observed lack of brittleness does not negate the presence of significant cellular damage. These findings are consistent with previous reports suggesting potential hepatic damage associated with growth stunting according to (Li *et al*., 2020; Carter *et al*., 1983).

CONCLUSIONS AND RECOMMENDATIONS

The present investigation delved into the physiological disruptions linked to growth retardation in broiler chickens reared in the Basra Governorate of Iraq. The data revealed that growth impediment correlated with marked decrements in erythrocyte and leukocyte counts, hemoglobin levels, and hematocrit values. Microscopic analysis of hepatic tissues from the affected avians showed cellular death and connective tissue proliferation. These pathological lesions point towards possible anemic conditions and immunological compromise, potentially detrimental to the avians' vitality, development, and disease resistance. It is imperative to conduct further research to decode the specific causal relationships affecting these hematological metrics and to assess ameliorative measures, including nutritional adjustments or precise supplementation. Future research endeavors should also encompass a wider geographic scope and extended study periods to yield a more holistic comprehension of the environmental determinants that contribute to stunting in broiler farming.

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NOVELTY STATEMENT

This study contributed valuable information to poultry producers and researchers, ultimately allowing them to develop more effective management strategies to optimize broiler health, growth, and productivity.

AUTHORS' CONTRIBUTIONS

Qutaiba Jasim Gheni, Tarek Ibrahim. Majeed, and Alfred Solaqa. Karomy: Development of the Methodology, Collection of samples, and Laboratory procedures.
Qutaiba Jasim Gheni, Tarek Ibrahim. Majeed, Wessam Monther Mohammed Saleh, and Alfred Solaqa Karomy: Preparing and writing the initial draft, review and editing the manuscript and analyze the data.

Ethical Approval

This study has been approved by the General Animal Use and Care Committee, College of Veterinary Medicine, University of Basrah, Basra State, Iraq.

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Conflict of Interest

The authors declare that there are no competing interests in the current study.

REFERENCES

Arzour-Lakehal N, Siliart B, Benlatrèche C (2013). Relationship between plasma free thyroxine levels and some biochemical parameters in two strains of broiler chickens. Global Vet., 10(3): 243-249.

Bhattacharya T, Bhowmik M (2014). In vivo immunosuppression in broiler chickens witb spontaneous infectious stunting syndrome. Indian J. Anim. Sci., 71(7).

Carter JK, Ow CL, Smith R (1983). Rous-associated virus type 7 induces a syndrome in chickens characterized by stunting and obesity. Infect. Immun., 39(1): 410-422. https://doi.org/10.1128/iai.39.1.410-422.1983

de Oliveira LB, Stanton JB, Zhang J, Brown C, Butt SL, Dimitrov K Afonso CL, Volkening JD, Lara LJ, de Oliveira CSF (2021). Runting and stunting syndrome in broiler chickens: Histopathology and association with a novel picornavirus. Vet. Pathol., 58(1): 123-135. https://doi.org/10.1177/0300985820969971

Health EPoA, Welfare (2010). Scientific opinion on the influence of genetic parameters on the welfare and the resistance to stress of commercial broilers. EFSA J., 8(7): 1666. https://doi.org/10.2903/j.efsa.2010.1666

Hoerr FJ (2010). Clinical aspects of immunosuppression in poultry. Avian Dis., 54(1): 2-15. https://doi.org/10.1637/8909-043009-Review.1

JH SRT, Dickey D (1980). Principles and procedures of statistics. A biometrical approach. McGraw Hill Book Company, New York, 1980)[Google Scholar].

Lenhardt Ľ, Mozeš Š (2003). Morphological and functional changes of the small intestine in growth-stunted broilers. Acta Vet. Brno, 72(3): 353-358. https://doi.org/10.2754/avb200372030353

Li H, Hu B, Luo Q, Hu S, Luo Y, Zhao B, Gan Y, Li Y, Shi M, Nie Q (2020). Runting and stunting syndrome is associated with mitochondrial dysfunction in sex-linked dwarf chicken. Front. Genet., 10: 1337. https://doi.org/10.3389/fgene.2019.01337

Natt M, Herrick C (1952). A new blood diluents for counting the erythrocytes of the chicken, poult. Sci., 31: 735-738. https://doi.org/10.3382/ps.0310735

Nili H, Jahantigh M, Nazifi S (2007). Clinical observation,

pathology, and serum biochemical changes in infectious stunting syndrome of broiler chickens. Comp. Clin. Pathol., 16: 161-166. https://doi.org/10.1007/s00580-007-0681-3

Prameela Rani M, NissarAhmad N, EswaraPrasad P, SriLatha C (2011). Haematological and Biochemical changes of stunting syndrome in Broiler chicken. Vet. World, 4(3): 124. https://doi.org/10.5455/vetworld.2011.124-125

Qamar M, Aslam H, Liaqat A (2015). Haematological studies on stunting syndrome in broilers. Res. J. Vet. Pract., 3(1): 19-24. https://doi.org/10.14737/journal.rjvp/2015/3.1.19.24

Qamar MF, Aslam H, Jahan N (2013). Histopathological studies on stunting syndrome in broilers, Lahore, Pakistan. Vet. Med. Int., (1), 212830. https://doi.org/10.1155/2013/212830

Shapiro F, Mahagna M, Nir I (1997). Stunting syndrome in broilers: effect of glucose or maltose supplementation on digestive organs, intestinal disaccharidases, and some blood metabolites. Poult. Sci., 76(2): 369-380. https://doi.org/10.1093/ps/76.2.369

Shapiro F, Nir I (1995). Stunting syndrome in broilers: physical, physiological, and behavioral aspects. Poult. Sci., 74(1): 33-44. https://doi.org/10.3382/ps.0740033

Shapiro F, Nir I, Heller D (1998). Stunting syndrome in broilers: effect of stunting syndrome inoculum obtained from stunting syndrome affected broilers, on broilers, leghorns and turkey poults. Poult. Sci., 77(2): 230-236. https://doi.org/10.1093/ps/77.2.230

Varley H, Gowenlock, A. H., & Bell, M. (1980). Practical Clinical Biochemistry. William Heinemann Medical Books. https://books.google.iq/books?id=2BPyjgEACAAJ

Zavala G (2006). Runting stunting syndrome (RSS) in broilers: In vivo studies. http://www.poultry-health.com/fora/inthelth/ zavala_wpdc_06.pdf Accessed 3/31/08.