

Effects of infectious bursal disease (IBD) on shank length and diameter, body weight and mortality in broiler breeder at rearing period

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Abstract: Shank length is used as a tool for the determination of fertility, body weight, estimates of frame size, and the monitoring of growth and development of pullets. This study was carried out to investigate the effects of infectious bursal disease (IBD) on shank length, diameter, body weight, shank length and body weight growth curves, and mortality rate in males and females in the rearing period in broiler breeder flocks. In this study, 2 broiler breeder flocks (Ross 308 flocks) and 800 breeder chicks (both females and males) were selected for shank length and diameter assessments. All the management, nutrition, and geographical statuses were similar, except for one flock that was infected with IBD. Results of this study showed that IBD presence in the flock decreased shank length in male and female chickens compared with the control group. Shank length in male and female chickens at 20 weeks of age was 0.53 and 0.02 cm, respectively, which was less than that in the controls, but these differences were not statistically significant. Shank diameter in the diseased male and female chicks was also smaller than that in the controls, although this difference was not also significant. There were no significant differences in the body weight of both sexes at 20 weeks of age. Body weight to shank length ratio in the diseased males was higher than that in the controls, but in female chickens it was less than it was in the control group after 12 weeks. Mortality rate in diseased males was 2.26% more than that in the control group ($P < 0.05$) but mortality in females was not different from that in the control group. After observing clinical IBD in both male and female chicks, antibody titers against IBD virus were found to have increased compared to the control group ($P < 0.0001$). It could be concluded that male chickens are more sensitive to IBD than females.

Key words: Shank length, shank diameter, body weight, mortality, broiler breeder, infectious bursal disease

1. Introduction

Shank development and growth in chickens can be affected by many factors. Incubation conditions (i.e. hot temperatures during the late incubation stages), sex, warm climate (season) (1), house temperature, reduced thyroid size and increased T4 levels, age, body weight, strain, storage time, nutrition (2), feeding programs such as ad libitum and skip a day (2), and disease agents such as mycoplasma are among the most important factors that can influence shank development and length. Genetics and nutritional and environmental factors can also affect both shank strength and development (3).

Development of the bones of chickens starts in the early stage of embryo development and continues throughout the maturity period (4). The majority of bone development occurs in the first 12 to 14 weeks of life. Shank and leg bone development is highly correlated with body weight. It is thus important to ensure correct body weight profiles in combination with good uniformity in

the rearing period, especially in the period of weeks 1 to 12 (5). Good early skeletal development is influenced by good early growth. Normal skeletal development in the rearing period of chicken production is important in terms of obtaining high levels of fertility, as shank length is highly correlated with fertility (5). Shank length could affect the intersexual cloacal distance during mating (6) and thus affect fertility, but no significant differences in shank length were observed after 28 weeks of age. Selection of males with good shank length and thickness will result in having good body conformation during life and also will increase male fertility. Males with a good balance of shank length, keel length, and breast width had a high fertility rate. Shank length can affect chicken leg health, and longer shanks are considered as a source of leg problems in heavy chickens (7).

Linear body measurements such as shank length, drum stick length, and wing length can be used in predictive equations to predict body weight in broilers. Shank and

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keel length are also the most commonly used estimates of frame size in breeder management, and there are small differences in shank length among strains at the time of hatching (7). The frame size should be good enough to avoid excess fat collection when the birds gain weight later. The frame size can be judged by the shank length (8). Shank length is often considered as a parameter for monitoring growth and development of Leghorn pullets (9). Pullets with ideal weight but shorter shank length are more prone to prolapse and blow out, as these birds carry excess abdominal fat (1). Bone growth deviation from normal conditions will result in bone disorders that have the potential to present major economic losses to the poultry industry (4).

Infectious bursal disease virus (IBDV) specifically infects the B cells of chickens. IBDV has also been reported in ostriches (10), Baltic ducks, and herring gulls (11). The outcome of IBDV infections is dependent on the strain and amount of the infecting virus in the body, age and breed of the birds, route of infection, and level of IBDV-neutralizing antibodies. IBD generally occurs in young chickens of 3 to 6 weeks of age and is a highly contagious and acute viral disease that is characterized by the destruction of B cells in the bursa of Fabricius (12). Losses due to classical IBD can reach up to 50% morbidity and less than 3% mortality in broilers and up to 20% mortality in commercial Leghorn pullets (13). Very virulent strains of the virus can cause in approximately 30%–40% and 50%–70% mortality in broilers and commercial layers, respectively (14).

Acute clinical IBD in chickens is characterized by ruffled feathers, watery diarrhea, trembling, severe prostration, dehydration, hemorrhages in the leg and thigh muscles, increased mucus in the intestine, and enlargement of the bursa (12). Histopathological lesions can be observed in the bursa, spleen, thymus, harderian gland, and cecal tonsils. The first signs of the disease occur in the bursa, as it is the most severely affected organ (12). Skeletal disorders are mostly seen in the legs, muscular system, and nervous system. Skeletal problems are often associated with leg weakness or lameness in chickens that can result in poor growth, culled birds, and increased mortality from starvation and dehydration.

In this study, shank length and diameter were evaluated in both male and female breeder chicken flocks with clinical IBD.

2. Materials and methods

Two broiler breeder flocks (Ross 308) were reared in one region under similar nutrition, management, and vaccination programs. Male (3000) and female (5000) breeder chicks were monitored from day 1 in terms of bone growth, especially shank length and diameter, with the hope that this may result in developing a baseline for Ross 308 breeder flocks. A total of 5% of chicks were selected randomly and kept separate in the middle of the house, and broiler weight, shank length, and shank diameter were measured during weeks 4–20 of age. Mortality rate was recorded daily. The length of the right shank (tibia tarsus) measured from the hock to the footpad and diameter of shank after 28 days were measured using a Vernier caliper (1).

Feed was distributed in houses by an automatic feeding system with the speed of 20.8 m/min. It took 3.49 min for the feed to circulate throughout the house. Compositions of the rations are presented in Table 1. Breeder flocks were vaccinated against the IBD virus to provide maternal immunity to the offspring so that all broiler flocks were seropositive to the virus (15). Vaccine type and vaccination programs were similar between the 2 flocks. A live vaccine D78 Nobilis (Intervet) were administered through drinking water at 3 stages (days 15, 22, and 32 of age). Blood samples were collected from 20 chicks per pen at day 2 and weeks 4, 8, 19, 27, 37, and 52. This study was conducted as a completely randomized design with 2 treatments and 5 replicates (50 female and 30 male chicks in each replicate) per treatment. Data were analyzed by the GLM procedure of SAS. Mean values were compared using Duncan's test.

3. Results

Body weight of diseased males at 20 weeks of age was not lower than that of healthy males, but there was no significant difference between these 2 groups. Body weights in diseased and healthy female birds were not also significantly different (Table 2; Figure 1). When broilers

Table 1. Composition of starter and grower diets in broiler breeder flocks.

Nutrient	Starter 1	Starter 2	Grower 1	Grower 2
Age of flock (weeks)	1–3	4–6	7	8–16
AME (Kcal/kg)	2750	2750	2700	2635
Crude protein (%)	19	18	16.5	14.5
Calcium (%)	1	1	1	1
Available phosphorus (%)	0.45	0.45	0.4	0.35

Table 2. Mean shank length, shank diameter, body weight to shank length ratio, body weight, and mortality at ages 8–20 weeks in infected and uninfected flocks.

Sex	Treatment	Shank length (cm)	Shank diameter (cm)	Body weight/ Shank length (%)	Body weight (g)	Mortality (%)
Male	Infected	11.83	1.29	19.39	2317	3.31 ^a
	Uninfected	12.17	1.30	17.74	2284	1.05 ^b
	SEM	0.283	0.024	1.007	150.87	0.087
	P value	0.395	0.673	0.259	0.879	0.049
Female	Infected	9.92	1.13	15.04	1512	0.67
	Uninfected	10.05	1.17	14.85	1515	0.89
	SEM	0.201	0.017	0.921	118.90	0.007
	P value	0.651	0.109	0.885	0.986	1

^{a,b}: Means values within a column without a common superscript differ significantly ($P \leq 0.05$).

had IBD, shank length for both sexes (males and females) decreased compared with the control group, and this reduction in male birds was greater than it was in the females (Figure 2). Diameter of the shank also decreased in both infected sexes, but there were not significant differences.

Body weight to shank length ratio in the diseased males was higher than the healthy group. The ratio in females was similar to males until 12 weeks of age. After week 12, this ratio in female chickens was lower than the control. The mortality rate in the diseased males was 3.31% and was higher than in the control group ($P < 0.05$). However, there were not significant differences in the mortality rate between the diseased female chickens and the female controls (Table 2). Results of ELISA analysis showed that after clinical disease is observed, antibody titers in both sexes significantly increased compared to the control group ($P < 0.0001$) (Table 3).

4. Discussion

In the present study, shank length of both sexes decreased in comparison with the control group. Reduction in shank length in male birds was greater than the reduction in the females (Figure 2). It suggested that males are more sensitive than females. The maximum shank length of 20-week-old infected males was 12.77 cm, which was 0.53 cm less than in the control group. Shank lengths in the control and diseased females were 106 mm and 106.8 mm, respectively. In this case, our findings confirmed the results of previous studies. Shank length at 18 weeks of age was 105.6 mm in the birds with normal weight (16). In free ranging local chickens in Tanzania, the mean shank length of females and males were 9.7 cm (7 to 12 cm) and 12.7 cm (8.5 to 15 cm), respectively (17). The shank length of the pullets at 11 weeks was 94 mm (9). Body weight had a significant effect on shank length; heavier chicks also tended to be longer and have longer shanks. In the

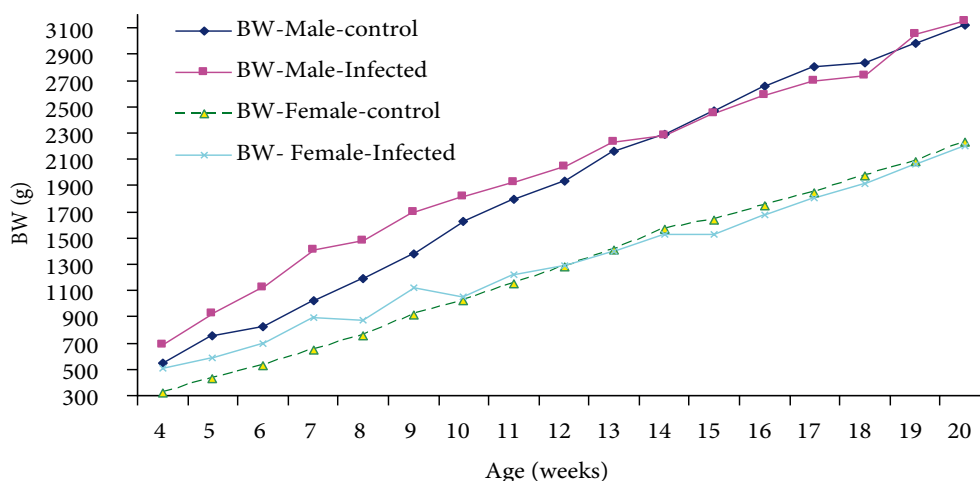


Figure 1. Effect of IBD on body weight. X= flock age (week), Y = body weight (g).

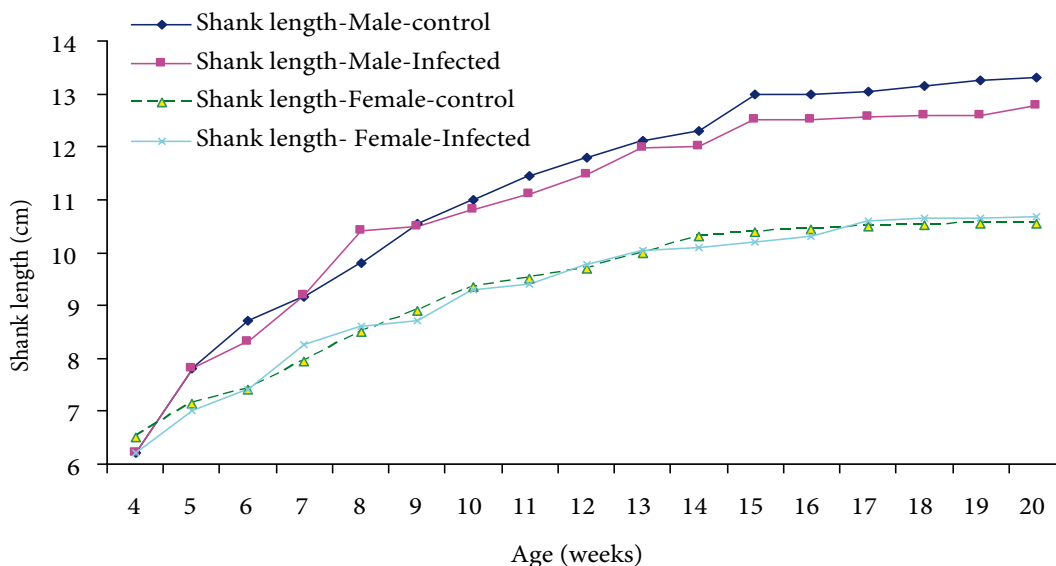


Figure 2. Effect of IBD on shank length. X = flock age (week), Y = shank length (cm).

Table 3. Mean of IBD titer in male and female broiler breeder during rearing period.

Treat	SEX	IBD titer				
		2 days	4 weeks	8 weeks	19 weeks	27 weeks
1(infected)		10,540	640 ^b	15,120 ^a	13,410 ^a	14,210 ^a
2(uninfected)	Male	10,540	2650 ^a	2800 ^b	3500 ^b	12,400 ^b
SEM		0.39	6.61	4.37	9.40	23.34
P value		1	0.0001	0.0001	0.0001	0.0001
1(infected)		10,210	618 ^b	10,270 ^a	11,730 ^a	12,740 ^a
2(uninfected)	Female	10,210	7490 ^a	7650 ^b	6450 ^b	11,500 ^b
SEM		0.13	4.67	11.98	6.53	6.61
P value		0.280	0.0001	0.0001	0.0001	0.0001

^{a,b}: Means values within a column without a common superscript differ significantly ($P \leq 0.05$).

current study, shank length in the control and IBD-affected females at 11 weeks of age was 96 and 94 mm, respectively. Heritability for shank length in the male lines was also higher than in female lines (18).

According to our study, the maximum shank length in the diseased female chickens was 10.68 cm, which was 0.02 cm less than in the control group. As shown in Figure 2, the difference in the shank length between the infected females and males was 2.09, and this difference in the control group was 2.7 cm. We observed that, at 20 weeks of age, shank length in males was 24.2% longer than in females. At 25 weeks of age, the shank length in males was 27% longer than in females at 21 weeks of age (19). Physiological development and growth in broiler breeders was very rapid

in the rearing period from 28 to 70 days (4–10 weeks). From 1 to 8 weeks of age, standard weights should be achieved, and at week 8, the males should reach 75% of their skeletal size (20). In a UK trial quoted by Pattison (21), 0.8% of broilers between 15 days old and slaughter were culled for lameness. Femoral head necrosis was the most common cause of lameness (38%), followed by infected hocks (13%), twisted legs (11%), and tibial dyschondroplasia (7%). It is estimated that femoral head necrosis can cause mortality within a range of 0.5% to 3% (21). However, Rahman et al. (22) observed hemorrhages on thigh and breast (pectoral) muscles, inflammation, edema, hyperemic lesions, occasional atrophy and frequent hemorrhages in the bursa of Fabricius, and, in some cases, swollen kidneys.

In the current study, the diameter of the shank decreased in both sexes in the IBD-infected flock. Maximum diameter of the shank at 20 weeks in the infected male group was 1.42 cm, compared to the control group, which was 0.02 cm less. Maximum shank diameter of diseased female chickens was 1.22 cm, compared to the control group, which showed a smaller shank diameter by 0.03 cm. Shank diameter and length were significantly correlated and ranged from 0.36 cm to 0.53 cm across all stocks (23).

The present study showed that body weight of diseased males decreased after 14 weeks. This reduction occurred after week 12 in females (Figure 1). There was no reduction in the body weight of infected males at the age of 20 weeks compared to the control group. The same pattern was also observed in diseased and healthy females at 20 weeks. Also, changes were obvious in body weights of diseased males compared to the controls (healthy). Birds with IBD were dehydrated, had darkened discoloration of the pectoral muscles, and had a reduction in number and size of microvilli 48 h after inoculation (12). Heritability values for body weight in the male lines at 20, 30, and 40 weeks of age were 0.892, 0.677, and 0.559, respectively. These values for female lines were 0.700, 0.562, and 0.429 (18).

The male growth profile is the single most important factor that correlates with flock fertility. The period of 42–91 days (6–13 weeks) is crucial in the development of males. During this period, rapid development of legs occurs (i.e. muscles, ligaments, and bones). Any deviation from the target growth profile may cause subsequent problems with livability and the performance of adult males. A good start in rearing males is crucial for body weight uniformity, as well as good organ and skeletal development, which are correlated with subsequent male fertility. It is important that the males achieve body weight targets according to the standards developed for that particular line. In males, sexual organs begin to develop around 70 days (10 weeks). Stress or interruption in growth over this period will affect growth of the testes and reduce adult fertility. In fact, we could decrease negative effects of IBD on body weight by good flock management.

The body weight to shank length ratio in the diseased males was higher than that in the control group. This ratio in females was similar to that in males until 12 weeks of age. After 12 weeks, this ratio in female chickens was less than in the control group, but the differences were very minor. This is another indication that males are more sensitive than female chickens.

Shank length and body weight are positively correlated. The relationship between body weight and shank length can be calculated by using the following formula (24): shank length (mm) = $\mu W(\text{kg})^{\beta}$. Both body length and shank length correlate better with the body weight at 14 days of age than with the hatch weight (25). There was

a highly positive genetic and phenotypic correlation between body weight and shank length at 20, 30, and 40 weeks of age (18).

Shank length is often considered to be a parameter for monitoring growth and development of Leghorn pullets, because shank length is so closely correlated with body weight. Shank length has almost reached its maximum value at week 14, and there is a potential for the body weight–shank relationship to become imbalanced due to subsequent compensatory growth of pullets that are underweight at this early age (1). Field experience suggests that pullets of ideal weight but shorter shank length are more prone to prolapse and blow out, as these birds carry excess abdominal fat. Environmental temperature also seems to influence shank development independent of body weight. No published data were found on the effects of skeletal disorders on weight gain, although they may have an impact.

In the present study, mortality rate in male chickens with IBD was 3.31%, which was 2.26% higher than in the control group ($P < 0.05$). However, there were no differences in the mortality rates between diseased and control female chickens. All other studies showed that IBD increased the mortality rate and showed results higher than our results. Rajaonarison et al. (26) reported that in IBD-affected birds 3 to 5 weeks old, the mortality rate ranged from 5.70% to 27.4%. However, Farooq et al. (27) reported that losses in broilers at the age above 32 days (17.66%) were higher than in broilers of 19–23 days of age (12.42%). The most affected birds were 4 weeks (28 days) old (28). Rate of IBD infection at 4, 3, and 5 weeks of age was 38%, 28%, and 28%, respectively (28). Also, data from 50 broiler farms in Pakistan showed that average mortality due to IBD was 15.31% and that mortality in winter (19.84%) was higher than in spring (9%) (27). Pattison (21) reported that the annual loss from mortality due to femoral head necrosis in broilers in the UK was £3.78 million. This estimate was based on 0.5% mortality from femoral head necrosis in 600 million broilers with a meat value of £1.26 per bird.

Mean ELISA titer of unvaccinated birds (194) is higher than in vaccinated ones (161 and 57.82) (29). ELISA titers data in Table 3 showed that after observation of the clinical signs of IBD, the antibody titer in both sexes increased more than in the control group, and this increase was highly significant ($P < 0.0001$), indicating that this additional antibody titer increased in the diseased group.

It can be concluded, based on the results of this trial, that male chickens are more sensitive to IBD than females, and, thus, the mortality rate in male chickens was increased. Also, IBD presence in the flock decreased shank length and shank diameter in males and females, but these differences were not significant.

References

1. Leeson S, Caston JL. Does environment temperature influence body weight, shank length in Leghorn pullets? *J Appl Poult Res* 1993; 2: 245–248.
2. Romero-Sanchez HP, Plumstead W, Leksrisompong N, Brake J. Feeding broiler breeder males. 2. Effect of cumulative rearing nutrition on body weight, shank length, comb height and fertility. *Poult Sci* 2007; 86: 175–181.
3. Whitehead CC. Causes and prevention of bone fracture. In: *Proceedings of the 19th Australian Poultry Science Symposium*. New South Wales, Australia: 2007. pp. 1–4.
4. Pines M. Poultry bone disorders. In: *Proceedings of the 19th Australian Poultry Science Symposium*. New South Wales, Australia: 2007. pp. 1–12.
5. Dudgeon JS. *Breeder Male Management. Important Management Points to Ensure High Levels of Fertility and Hatchability*. Newbridge, Scotland, UK: Aviagen, Ltd, 2010.
6. McGary S, Estevez I, Bakst MR. Potential relationships between physical traits and male broiler breeder fertility. *Poult Sci* 2003; 82: 328–337.
7. Gao Y, Du ZQ, Feng CG, Deng XM, Li N, Da Y, Hu XX. Identification of quantitative trait loci for shank length and growth at different development stages in chicken. *Anim Genet* 2010; 41: 101–104.
8. Robinson FE, Zuidhof MJ, Renema RA. Reproductive efficiency and metabolism of female broiler breeders as affected by genotype, feed allocation, and age at photo stimulation. 1. Pullet Growth and Development. *Poult Sci* 2007; 86: 2256–2266.
9. Poulvet. *Poultry layer farm management-growing (6 to 11 weeks)*. Hyderabad, Andhra Pradesh, India. 2010. <http://www.poulvet.com/directory/profile.php?addrid=15070>.
10. Gouch RE, Drury SE, Cox WJ, Johnson CT, Courtenay AE. Isolation and identification of birnavirus from ostriches (*Struthio camelus*). *Vet Res* 1998; 142: 115–116.
11. Hollmen T, Franson JC, Docherty DE, Kilpi M, Hario M, Creekmore LH, Peterson MR. Infectious bursal disease virus antibodies in eider ducks and herring gulls. *Condor* 2000; 102: 688–691.
12. Ashraf S. *Studies on infectious bursal disease virus*. PhD thesis, Ohio State University, Ohio, USA, 2005.
13. Moody A, Sellers S, Bumstead N. Measuring infectious bursal disease virus RNA in blood by multiplex real-time quantitative RT-PCR. *J Virol Met* 2000; 85: 55–64.
14. Tanimura N, Tsukamoto K, Nakamura K, Narita M, Maeda M. Association between pathogenicity of infectious bursal disease virus and viral antigen distribution detected by immunohistochemistry. *Avi Dis* 1995; 39: 9–20.
15. Lukert PD, Saif YM. Infectious bursal disease. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, editors. *Diseases of Poultry*. 11th ed. Ames, IA, USA: Iowa State Press; 2003. pp. 161–179.
16. Renema RA, Robinson FE, Zuidhof MJ. Manipulating reproductive potential :growth profile and photostimulation age effects in broiler breeders. In: *Proceedings of the Broiler Breeder and Hatchery Conference*. North Carolina, USA: North Carolina University; 2004. pp. 51–62.
17. Msoffe PLM, Mtambo MMA, Minga UM, Gwakisa PS, Mdegela RH, Olsen JE. Productivity and natural disease resistance potential of free-ranging local chicken ecotypes in Tanzania. *Live Res Rural Dev* 2002; 14: 1–12.
18. Kabir M Oni OO, Akpa GN, Adeyinka IA. Heritability estimates and the interrelationships of body weight and shank length in Rhode Island Red and white chickens. *Pakistan J Biol Sci* 2006; 9: 2892–2896.
19. Buss EG. Genetics of growth and meat production in turkeys. In: Crawford RD, editor. *Poultry Breeding and Genetics*. 3rd ed. Amsterdam: Elsevier; 2003. pp. 649–653.
20. Casanovas P. Managing males to maximise hatchability. *World Poult* 2008; 24: 26–28.
21. Pattison M. Impacts of bone problems on the poultry meat industry. In: Whitehead CC, editor. *Bone, Biology and Skeletal Disorders in Poultry*. Abingdon, UK: Carfax Publishing; 1992. pp. 329–338.
22. Rahman MS, Islam MS, Rahman MT, Parvez NH, Rahman MM. Analysis of prevalence of infectious Bursal disease in broiler flocks in Dinajpur. *Int J Sus Crop Prod* 2010; 5: 15–18.
23. Yang AE, Dunnington A, Siegel PB. Developmental stability in stocks of white leghorn chickens. *Poult Sci* 1997; 76: 1632–1636.
24. Tierce JF, Nordskog AW. Performance of layer type chickens as related to body conformation and composition. 1. A static analysis of shank length and body weight at 20 weeks of age. *Poult Sci* 1985; 64: 605–609.
25. Wolanski NJ, Renema RA, Robinson FE, Carney VL, Fancher BI. Relationships among egg characteristics, chick measurements, and early growth traits in ten broiler breeder strains. *Poult Sci* 2007; 86: 1784–1792.
26. Rajaonarison JJ, Rakotonindrina SM, Rakotondramary EK, Razafimanjary S. Gumboro disease (infectious bursitis) in Madagascar. *Rev Elev Med Vet Pays Trop* 1994; 47: 15–17.
27. Farooq MF, Durrani R, Imran N, Durrani Z, Chand N. Prevalence and economic losses due to infectious bursal disease in broilers in Mirpur and Kotli districts of Kashmir. *Int J Poult Sci* 2003; 2: 267–270.
28. Khan RW, Khan FA, Farid K, Khan I, Tariq M. Prevalence of infectious bursal disease in broiler in district Peshawar. *ARPN J Agri Biol Sci* 2009; 4: 1–5.
29. Islam MT, Samad MA. Outbreaks of infectious bursal disease in vaccinated and unvaccinated commercial cockerel farms in Bangladesh. *Bangladesh J Vet Med* 2003; 1: 21–24.